THE PATIENT

Biological, Psychological, and Social Dimensions of Medical Practice

SECOND EDITION

Hoyle Leigh, M.D.
Morton F. Reiser, M.D.

THE PATIENT

Biological, Psychological, and Social Dimensions of Medical Practice

SECOND EDITION

THE PATIENT

Biological, Psychological, and Social Dimensions of Medical Practice

SECOND EDITION

Hoyle Leigh, M.D., F.A.P.A.
Professor of Psychiatry
Yale University School of Medicine and
Director, Psychiatric Consultation–Liaison and Outpatient Services
Yale–New Haven Hospital
New Haven, Connecticut

and

Morton F. Reiser, M.D., F.A.P.A.
Charles B. G. Murphy Professor of Psychiatry and
Chairman, Department of Psychiatry
Yale University School of Medicine
New Haven, Connecticut

© 1985 Hoyle Leigh and Morton F. Reiser
Softcover reprint of the hardcover 1st edition 1985

FOR VINNIE, LYNN, AND ALEXANDER

Foreword

The old-fashioned doctor, whose departure from the modern medical scene is so greatly lamented, was amply aware of each patient's personality, family, work, and way of life. Today, we often blame a doctor's absence of that awareness on moral or ethical deficiency either in medical education or in the character of people who become physicians.

An alternative explanation, however, is that doctors are just as moral, ethical, and concerned as ever before, but that a vast amount of additional new information has won the competition for attention. The data available to the old-fashioned doctor were a patient's history, physical examination, and "personal profile," together with a limited number of generally ineffectual therapeutic agents. A doctor today deals with an enormous array of additional new information, which comes from X-rays, biopsies, cytology, electrographic tracings, and the phantasmagoria of contemporary laboratory tests, and the doctor must also be aware of a list of therapeutic possibilities that are both far more effective and far more extensive than ever before.

The old-fashioned doctor could concentrate on the patient's personal profile, therefore, because it was one of the few sources of available information and because it has a major therapeutic role. Unable to rely on powerful drugs and other treatments, the old-fashioned doctor made himself into an agent of treatment, using his skillful knowledge of people, human reactions, and communication as the basis for the "iatrotherapy." What the doctor learned about a patient's personal status was a guide to the way the iatrotherapy was planned and delivered.

Today's doctor must contend not only with all the additional data and treatments of modern technology, but also with the scientific advances that provide pathophysiological explanations for the data, phar-

macological and other bases for the treatment, and a coordinated rationale for the practice. Struggling to keep abreast of all the new data, treatments, and advances, and often having a specialized focus restricted to only some of them, the doctor may not find time or make time for the personal features that were once so preeminent and predominant in medical practice.

And yet, despite all the technological changes, people have not changed. They still have personalities, families, work, and a way of life. These "psychological and social dimensions" influence the course of illness and also act as important determinants of gratification, distress, hope, anxiety, and the diverse joys and sorrows of human existence. When seeking medical aid today, people want all the benefits of modern medical technology, but they still want (and still need) attention to their attributes as people.

As technology brought its powerful new revelations and interventions for ailments of the soma, consideration of the patient's psyche became increasingly detached from concomitant medical consideration. Instead, a patient's personal problems and psychic discomforts became relegated to a separate group of specialists who had their own ideas about psychodynamic patterns in pathophysiology, about treatments that usually emphasized talking, and about scientific explanations for the observed phenomena and therapeutic tactics.

With psychiatry developing in one set of directions and "organic medicine" in another, patients became increasingly partitioned among modern specialists. This trend seems to have crested about a decade ago, however, and remedial action has already begun. In "somatic medicine," a new emphasis or renascence has occurred for the generalist, whose job is sometimes named with a bewildering array of titles (e.g., *general practitioner, family practitioner, general internist, ambulatory pediatrician, primary care physician*), but whose medical role clearly includes a return of attention to the patient as a person. In psychiatry, the new developments have been a powerful set of "psychotropic" drugs, which are often more promptly effectual than years of talking, and a more open approach to pathophysiology, which has liberalized the constraints of a dominant emphasis on older psychodynamic formulations.

As this progress was occurring in the individual domains of both soma and psyche, a reunion of the two seemed logical as well as necessary. The conjunction has now begun to happen with the development of liaison services, in which psychiatrists work directly with somaticists in the care of patients in medical, surgical, and other nonpsychiatric sections of medical institutions. This book emanates from two authors who have been both prominent and successful in developing this type of liaison.

From their professional familiarity with conventional psychiatry as well as their extensive experience in medical liaison work, Drs. Leigh and Reiser have integrated the biological aspects of the soma with the psychological and social attributes of the psyche. The authors have identified a series of important clinical features that seldom receive enough medical attention and discussion: the *iatrotropic stimulus* and other reasons patients seek medical help, the expectations and behavior patterns of sick people, and the communication mechanisms that can help or hinder the relationship between doctors and patients. Separate chapters deal with clinical phenomena that commonly occur in ordinary medical practice: problems in anxiety, defense mechanisms, depression, pain, and sleep.

An outstanding part of the book is a "Patient Evaluation Grid," which demonstrates how personal-profile information can be organized chronologically in a manner analogous to the family history, past history, and present illness of the routine medical record. This new grid also coordinates the personal profile as an "environmental" concomitant of the "biological dimensions" that are usually the main constituent of medical records.

A particularly attractive feature of the book is the "vignettes" used to introduce each chapter. These brief case reports, which represent problems that are constantly encountered in medical practice, have been carefully chosen to illustrate the themes of each chapter and to provide direct clinical relevance for the discussions. In the last part of the book, the authors make use of vignettes, previous discussions, and Patient Evaluation Grid to demonstrate and help solve a series of professional and pharmaceutical challenges in the management of patients.

The book should be especially worthwhile for medical students and for house staff who want to transcend modern technocratic wisdom by developing their own talents as skillful iatrotherapists. The comments and suggestions will also appeal to both psychiatric and medical practitioners whose main clinical concerns are with helping patients rather than maintaining professional paradigms. The perceptions cited in the text were formerly available only to the small group of physicans and students who encountered the authors during the activities of a psychiatric department and liaison service. With this book, the authors have made their useful and important ideas accessible to everyone else.

Alvan R. Feinstein, M.D., F.A.C.P.
Professor of Medicine and Epidemiology
Yale University School of Medicine
New Haven, Connecticut

Preface to the Second Edition

The first edition of *The Patient* was published at a time when it was not entirely clear what to include or how to present a behavioral science course so that it would be optimally suited for the needs of medical students. *The Patient* had two goals: It represented our effort to impart, first, what we regarded as essential core data for medical behavioral science and, second, a way of integrating that information with data from other biomedical sciences. It seemed clear then, as it does now, that an integrated biomedical approach is an essential ingredient of patient care. Our experience in using *The Patient* as the basic textbook for the behavioral science course at Yale has been quite successful, and judging from its widespread acceptance elsewhere, from trends in the National Boards, and from requests for a revised second edition, there seems to be quite general agreement that it provides both coverage of essential core material and a useful way of approaching patients.

Medical science advances at an exciting pace, and behavioral science is no exception. Accordingly, revisions, some of them quite extensive, have been made in order to incorporate the latests advances. Two new chapters have been added—one on *psychosis* and another on *organic brain syndromes*. Both of these chapters cover clinical phenomena with which all physicians, not only psychiatrists, must be familiar. The chapter on organic brain syndromes devotes a major section to discussion of the *elderly patient*, to direct special attention to a subject that is becoming increasingly important in the practice of medicine.

In writing the first edition, we felt that the book should not aim to serve as a conventional textbook introduction to clinical psychiatry. It still is not that. But as it has developed, we feel it has grown into a text that can serve to facilitate the transition from the preclinical to the clinical

years. It should furnish the student the opportunity to learn what it is necessary to know about clinical psychiatry as it is encountered in general medicine. All important syndromes in psychiatry relevant to the general physician are discussed in this second edition. We were encouraged in developing this aspect of the book by many of our students, who told us that rereading *The Patient* in their clinical years during their medical and psychiatric rotations had greatly facilitated their understanding of patients and their ability to formulate treatment plans.

This second edition of *The Patient* continues the thrust of the first edition in emphasizing basic principles in patient evaluation and management. The Patient Evaluation Grid (PEG) that was presented in the first edition has been revised and further systematized, and we present a new companion form, the PEG-Management Form, which provides three-dimensional diagnoses and management plans for the patient.

We hope that this edition will be a useful companion to students of medicine throughout their years as they are learning to understand and treat patients.

In preparing this second edition of *The Patient*, we gratefully acknowledge the editorial assistance of Ms. Janice Stern of Plenum.

<div style="text-align: right">

Hoyle Leigh, M.D.
Morton F. Reiser, M.D.

</div>

New Haven, Connecticut

Preface to the First Edition

The primary subject matter of medicine is the patient. Diagnosis and treatment of disease are an integral part of the patient's care, but the latter includes, in addition, an understanding of the behavioral aspects of the patient and of the surrounding environment, including the family and the health-care personnel. Until recently, behavioral aspects of patient care have been relegated by many physicians to the "art of medicine" rather than the "science of medicine," and much of the information relevant to the care of a patient as a person came from researchers in nonmedical disciplines, often through the study of normal healthy persons rather than patients. This led to conceptual and practical gaps between behavioral science and the practice of medicine.

In our work with medical students as lecturers and seminar leaders in a basic behavioral science course, as teachers and preceptors of medical student clinical clerks in both psychiatry and medicine, and as faculty members intimately involved in curriculum development at the Yale University School of Medicine, we became acutely aware that many students were bemoaning the lack of a good practical and relevant textbook that would bridge the gap between the behavioral sciences and medicine. Initially, our plan was to present information relevant to medicine gleaned primarily from behavioral sciences under the tentative title "Behavioral Foundations of Medical Practice." In the course of actual writing, however, we came to realize that straight presentations of behavioral science knowledge, however relevant, would be of limited value to the student of medicine. We have found in our liaison consultation practice and teaching that the clinician's best use of behavioral science data is achieved when it can be integrated with the biological clinical data of medicine. We also realized that integration of behavioral

xiii

and biological foundations of medicine was not taught anywhere in the medical curriculum. Although many experienced physicians have achieved such an integration on their own many others never even attempt it. Without basic behavioral science information, this integration is haphazard even when there is motivation for it. Thus, we decided to attempt a practical demonstration of integration between behavioral science knowledge and the biological underpinnings of medicine and to focus on patient care. The practice of medicine involves, for each physician, the challenge of achieving such integration. The care of patients provides an opportunity for linking the spheres of medical biology and the behavioral sciences.

Some basic principles concerning patient care will be the same for some time to come, even though facts and data bases may change. One of the basic principles we refer to is the principle referable to good patient care—that comprehensiveness and thoroughness are essential. Simple as it may seem, this involves the simultaneous addressing of three dimensions of information—that of the disease, the patient as a person, and the surrounding environmental system.

This book is intended to challenge medical students in the preclinical years and those who are preparing for their first clinical clerkships. The challenge is to think about the patients as entities in their own right and to integrate the basic knowledge that they are in the process of gaining into an approach to the care of patients. This book, if successful, should also challenge serious students at all levels of experience, including house staff and attending physicians in nonpsychiatric clinical specialties as well as psychiatrists who work and teach in the field of consultation liaison psychiatry.

It is *not* a conventional textbook of behavioral science.
It is *not* a conventional textbook of medicine.
It is *not* an introduction to clinical psychiatry.

This book *is* intended to be a practical book. Many people have expounded on the need for the biopsychosocial perspective in medicine, but few have shown how this perspective could actually be used in practice. The Patient Evaluation Grid (PEG) (Chapter 11) is a concrete and practical method through which the reader may plan the management of his or her patient in the biological, personal,* and environmental

---

* We use the term "personal" here to denote all the attributes of the whole person as a unit. This, of course, includes the psychological dimension, but also includes behavioral characteristics such as habits.

dimensions. In integrating behavioral sciences with medical practice, we used as an organizing principle the general systems approach.*

References are given in order that the reader may read further about the topic being discussed. Thus, our references do not necessarily refer to the original source; review papers and original papers are referred to without distinction.

Much impetus for writing this book came from our students and colleagues. In the course of our work, we learned more from our students than from anyone else. Many versions of various chapters of this book have been read by them in various contexts, and their incisive and thoughtful comments and suggestions have served as stimuli to improve the work. Student interest (and encouragement) served as powerful support when the progress of the book seemed to lag. Our first thanks, then, are due our students, past and present.

Many friends and colleagues have read portions of this book and given us valuable advice. It is impossible to name all of them, but we owe special thanks to the following: Malcolm Bowers, Jr., M.D., Robert Byck, M.D., William Collins, M.D., Marshall Edelson, M.D., Ph.D., Alvan R. Feinstein, M.D., Stephen Fleck, M.D., Earl Giller, M.D., Ph.D., Richard Goldberg, M.D., Vincenta Leigh, M.S.N., Eugene Redmond, M.D., Lynne Whisnant Reiser, M.D., Richard Ross, M.D., and Gary Schwartz, Ph.D.

We are also indebted to Ms. Hilary Evans of Plenum Publishing Corporation for editorial assistance and to Mss. Erica Fritz, Judy Guy, Jacqueline Collimore, and Marcia Cooper for typing innumerable drafts and for other secretarial work.

<div align="right">

Hoyle Leigh, M.D.
Morton F. Reiser, M.D.

</div>

New Haven, Connecticut

---

* For a comprehensive and authoritative exposition on general systems theory of living things, we recommend James G. Miller's *Living Systems* (New York, McGraw-Hill, 1978). For a concise introduction, we recommend Miller's chapter, General systems theory, in *Comprehensive Textbook of Psychiatry, II*, Vol. 1, edited by A. M. Freedman, H. I. Kaplan, and B. J. Sadock (Baltimore, Williams & Wilkins, 1975), pp. 75–78.

Contents

PART II.   ON BEING A PATIENT: PSYCHOPHYSIOLOGICAL CONSIDERATIONS

PART III.   ON ASSESSING A PATIENT:
A CLINICAL SYSTEMS APPROACH

List of Figures and Tables

PART I
ON BECOMING A PATIENT
Psychosocial Considerations

Illness and Help-Seeking Behavior

In the past, the term *patient* designated a person who was suffering or enduring pain, related to the word *patience* (*Webster's Third New International Dictionary*). Modern usage of the term *patient* denotes a person who is seeking or is being given medical care. Although an illness usually leads a person to seek medical help, not all who are ill become patients nor are all patients necessarily ill.

1. A man, age 39, has been experiencing occasional sharp pain in his chest for the last two months. Being a pressured junior executive, he has put off seeing a doctor, attributing the pain to indigestion associated with pressure at work. In fact, Alka Seltzer seemed to help it somewhat. Last night, during an argument with his wife, he had another bout of chest pain that took several hours to subside even after he took Alka-Seltzer. He called his doctor this morning for an urgent appointment.

2. Three months ago, a 30-year-old woman developed severe headaches and fainting spells following a "head cold." She told her close friends, who then arranged a prayer meeting to chase away the "evil spirits" that they felt were causing her problems. Today, after the prayer meeting she felt much better.

3. A 45-year-old woman decided to do something about her varicose veins, which she had had for 20 years. She told her doctor today that she wanted to have the operation as soon as possible, even tomorrow.

4. A coal-mine worker came to see the company physician, complaining of chest pain and difficulty in breathing. Careful medical workup was negative

3

*for any physical disease. The physician felt that psychological factors might
be involved in his coming for medical evaluation when he learned that a co-
worker of the patient had recently been found to have "black lung" disease
and had been given a sizable compensation.*

All four patient vignettes presented above involve help-seeking behav-
ior, but the patients were quite dissimilar in how they perceived their
symptoms and when and how they went about getting help.

How is help-seeking behavior to be conceptualized, and how are
the wide variations in the form it may take to be understood? Medical
sociologists have studied these questions and developed concepts, def-
initions, and data that are useful to the physician in his efforts to un-
derstand the circumstances that bring the patient for help and in facil-
itating development of a productive relationship with the patient from
the very start.

Concepts and Definitions: Help-Seeking Behavior and Illness Behavior

Help-seeking behavior arises from the decision of an individual to
do something about a symptom or distress. In *medical* help-seeking be-
havior, a patient contacts a doctor. This initiates a process whereby a
small health-care system forms around the patient. Calling the doctor
to make an appointment, visiting the doctor in his office, or going to
the emergency room of a hospital are examples (vignettes 1, 3 and 4
above). *Nonmedical* help-seeking behaviors include going to a clergyman
or to a variety of nonprofessional people such as "rootworkers" and
faith healers, or asking a relative or friend for advice (vignette 2). Of
course, nonmedical help-seeking behaviors often lead to medical con-
tacts through advice and referral, and many patients engage in medical
and nonmedical help-seeking behaviors simultaneously, for example,
mobilizing family support and visiting a doctor. Quasi-medical help-
seeking behaviors, such as self-medication or discussing symptoms with
a druggist, while nonmedical by definition since they do not involve the
immediate formation of doctor–patient contact, may also lead to it even-
tually. (In vignette 1, buying Alka-Seltzer, a quasi-medical help-seeking
behavior, was an intermediate step toward medical help-seeking be-
havior.)

Help-seeking behavior is closely related to its antecedent, *illness
behavior*. Illness behavior, described by Mechanic (1962), consists of the
ways in which given symptoms may be perceived, evaluated and acted
(or not acted) on. Illness behavior does not always lead to help-seeking

Table 1. Factors That Affect Help-Seeking Behavior

Symptoms and signs
Commonality, familiarity, predictability, threat
Demography
Socioeconomic class, religion, ethnicity
Stress
Previous experience
Personal, others

behavior. Help-seeking behavior stimulated by a symptom can be expected to be influenced by the severity and quality of the symptom. It would seem that the more acute, severe, distressful, frightening, and persistent symptoms are, the more likely they are to lead to a quest for medical help, but these common-sense expectations are overly simple. Help-seeking behaviors as actually observed in clinical practice are often far more complex and hard to understand. Even in the presence of severe persistent and frightening symptoms like fainting spells, an individual often does not seek medical help (vignette 2). A patient's response to symptoms is modified considerably by a variety of factors that influence the ultimate decision whether or not to seek help, and, if help is sought, when and what kind (Table 1).

Factors That Influence Help-Seeking Behavior

Factors That Influence How Symptoms and Illness May Be Perceived

Four dimensions of an illness (or a symptom) are important in influencing how the illness is perceived: (1) the frequency with which it occurs in a given population (*commonality*), (2) the familiarity of the symptoms to the average member of the community (*familiarity*), (3) the predictability of the outcome of the illness, and (4) the amount of threat and loss likely to result from the illness (Mechanic, 1962). For example, since the common cold is familiar, easily recognized, and self-limited, and carries minimal risk of major loss, a person with a cold is unlikely to seek medical help. Coughing up blood, in contrast, greatly increases the probability of contact with a physician, since this symptom is uncommon, unfamiliar, unpredictable, and threatening.

Naturally, the patient's personality is also important in determining help-seeking behavior. Personality factors are taken up in Chapter 17.

Demographic Factors

Socioeconomic Class. Whether or not symptoms lead to help-seeking behavior may be influenced by the patient's socioeconomic class. For example, upper-class persons tend to report themselves ill more often than lower-class persons (Koos, 1954). This class difference can be related in large part to realistic economic considerations—the upper classes being more easily able to afford medical bills and lose a day's work to see a doctor—as well as to the higher educational level and greater awareness of methods of getting help. The very poor on public assistance can take the time to engage in help-seeking behavior more readily than the working poor, since they do not have to lose hourly pay to make use of the medical facility in the community, but there is some evidence that these social-class differences in medical-care utilization are diminishing (Ross, 1962).

Orientations toward illness differ according to social class (Mechanic, 1968). In general, lower-class populations are more fatalistic about contracting disease and thus less likely to be oriented toward preventive medicine (Deasy, 1956; Rosenstock, 1969) and toward consultation with medical professionals when ill (Koos, 1954; Redlich *et al.*, 1955; Brightman *et al.*, 1958).

There is some evidence to suggest that similar disease processes may present differently in patients in different socioeconomic classes. Upper-class persons with coronary disease often present themselves to the medical facility with angina pectoris, while lower-class persons are more likely to present themselves with acute myocardial infarction or sudden death (Shekelle and Ostfeld, 1969). It is not clear why this is so, although upper-class persons might be readier to come to a medical facility with a milder distress.

Diagnoses of neuroses and personality disorders have been made with higher frequency in upper-class persons, while diagnoses of psychoses, "psychosomatic" reactions, or hysterical reactions have been more commonly made in lower-class populations (Hollingshead and Redlich, 1958). This difference may indicate both an actual prevalence difference and a tendency on the part of doctors to diagnose more benign conditions in the upper-class persons because of their own biases. Greater prevalence of schizophrenia (psychosis) in lower-class populations may be explained in one of two ways: either (1) lower-class environment and genetic pool contribute to the development of the disorder ("origin hypothesis") or (2) schizophrenic individuals wind up in lower classes because of the disability caused by the illness ("drift hypothesis").

Religion and Ethnic Origin. Religion and ethnic origin influence illness behavior through different cultural attitudes and expectations. Some cultural groups (e.g., Spanish-speaking people in the Southwest) are more likely than others (English-speaking people in the same area) to seek medical care and support. They rely on folk medicine when ill (Saunders, 1954), while English-speaking people in the same area tend to seek modern medical treatment. In a questionnaire study in a large university, Jews and Episcopalians reported that they would have a higher inclination to use medical facilities for various hypothetical symptoms than did Christian Scientists and Catholics (Mechanic, 1962). Religious influence on illness behavior is independent of social class.

Zborowski (1952, 1969) studied attitudes and responses to pain of patients belonging to different ethnic groups. His subjects consisted of "Old American," Irish, Jewish, and Italian male patients. The Old American and Irish patients tended to minimize pain, to delay seeking consultation with a physician, and to respond to pain stoically, while the Jewish and Italian patients tended to exaggerate pain and react to it more emotionally. The Jewish and Old American patients were more concerned about pain as a "warning signal," that is, its implications as a symptom; the Irish were more concerned about its crippling indications; and the Italians were more concerned about the immediate consequences of the painful experience and its instant relief. Ethnic differences were also related to help-seeking behavior and attitude toward doctors. The Old Americans tended to consult druggists, osteopaths, chiropractors, and other nonphysicians before finally consulting the doctor, but had implicit trust in the professional competence of the doctor on the basis of his being an M.D., once he was consulted. The Jewish patients, on the other hand, tended to consult the doctor quite readily and also to check into the doctor's qualifications and to "doctor shop." The attitude and warmth of the doctor rather than his professional skills were seen to be of overriding importance to the Italian patients.

Although these findings represent a cross-sectional picture of illness behavior in the recent past and their validity at present is uncertain, they nevertheless demonstrate the importance of socioethnic milieu with respect to the development of psychological sets concerning symptoms, illness, and the medical profession.

Role of Stress

Psychosocial stress plays a major role in transforming illness behavior into help-seeking behavior (Mechanic, 1962).

The type of stress is particularly important. By and large, *interpersonal stresses* such as difficulties with loved ones are more likely to lead to medical help-seeking behavior than are noninterpersonal stresses such as financial hardship. This may be because communication, interaction, and nurturance usually expected from a doctor are especially (and unconsciously) sought after by persons in interpersonal difficulty. People are often able to initiate a personal interaction with the physician more easily than with someone with whom the contact is less structured and the role less defined. Interaction with the physician can usually be initiated with little difficulty and does not usually require complex and subtle cues and responses necessary in social relationships. In other words, social skills on the part of the patient are not as necessary in interaction with physicians as in other social situations.

Interpersonal difficulties are also frequent precipitating events of anxiety, depression, and grief, and these are probably accompanied by physiological states that may precipitate an illness or exacerbate a symptom (see Chapters 4, 6, and 13).

Finally, it should be remembered that somatic symptoms themselves are emotional stressors, and the stress they generate may finally push the illness behavior toward help-seeking behavior.

Role of Previous Experience ("Priming Factors")

Previous personal experiences as a patient with illness and health care may strongly influence subsequent help-seeking behavior. Positive memories of a hospital experience, such as uneventful recovery from an appendectomy, make it easier to initiate contact with a doctor the next time. On the other hand, exposure to negative experiences, such as complications arising from medical treatment, prolonged recovery, and exposure to impersonal and brusque behavior on the part of health care personnel may discourage or delay future contacts with the setting. Even when a patient with negative past experiences does contact the physician, he is likely to do so with negative expectations, and this may result in a self-fulfilling prophecy.

Exposure to the *experiences of relatives or friends* with medical systems in the community may also have its effects, shaping the expectations that the person might bring to the system when he becomes ill. Similarly, such exposure may influence attitudes toward a particular illness or set of symptoms.

> A woman was convinced that she had cancer of the breast when she developed pains in her joints. This conviction persisted in the face of her doctor's repeated reassurances that she had arthritis. As it turns out, a friend had suffered bone pain from spread of breast cancer to the bone. The conviction was strong enough to make her delay return visits to the doctor for fear that she would have to undergo mutilating breast surgery as had her friend.

In these ways, direct or indirect personal exposure to illness and health-care systems affects help-seeking attitudes and behavior. Additionally, it may "prime" a patient for psychological complications such as depression based on unfounded or fantasized expectations. Similarly, educational programs, reading medical books or magazine articles, watching television programs about medical matters, and the like, may also set the stage for unfounded fears, although it is our impression that their impact is somewhat weaker than the personal exposures discussed above.

Community attitudes toward health care provide an explicit and implicit background against which an individual contemplates the possibility of seeking care. We have observed that when a hospital that originally had been used for chronic and terminally ill patients was converted to a modern, acute-care, general hospital, many patients admitted to that hospital as long as ten years later expressed uneasiness because of the lingering reputation of the hospital as "a place to die." Community and peer-group myths and superstitions also influence the type of help that is sought, for example, physician or hospital emergency room.

Taxonomy of Medical Help-Seeking Behavior

McWhinney (1972) proposed a taxonomy of medical help-seeking behavior that he calls *patient behavior*, using the doctor–patient contact as the reference point. This classificatory scheme is useful in assessing clinical situations. It consists of five mutually exclusive categories as immediate precipitating causes of the doctor–patient contact.

Limit of Tolerance. The patient initiates contact with the doctor because the discomfort, pain, or disability has become intolerable. A subcategory is limit of tolerance for unhappiness.

Limit of Anxiety. The patient initiates contact with the doctor because of anxiety concerning the implications of the symptom. The symptom

itself may be quite tolerable, but the implications are not. An example is bloody urine.

Problems of Living Presenting as a Symptom (Heterothetic* Presentation). The patient initiates contact with a physician ostensibly because of symptoms that are minor (exceed the limit of neither tolerance nor anxiety) but in the context of disturbing emotional difficulties that constitute the underlying but unrecognized motive for the help-seeking action. An example is a woman who, at the time of painful interpersonal conflict with her boss, comes to the doctor complaining of back pain that she has had occasionally for many years.

Here, the underlying reason for the doctor–patient contact is an *attempt to establish a supportive interpersonal relationship* and find *relief from the emotional distress*. Many patients who present with vague and long-standing symptoms (20 years in vignette 3 above) and many patients who experience unexplained delay in recovering from illness will often on further inquiry be found to have some major emotional problems.

Conversely, some patients who present with *emotional problems* to mental health personnel or nonmedical counselors may turn out to be suffering from *serious medical disorders*, and it is important that the *medical condition* of the patient be checked before assuming that the symptoms are indeed psychological in origin.

The next two categories are doctor–patient contacts that are not help-seeking in the usual medical sense.

Administrative. These doctor–patient contacts are for administrative reasons only, whether or not the patient is ill, for example, insurance examination.

No Illness. This includes all visits for preventive purposes, as well as routine physical examinations, for example, antenatal care.

In this scheme of classification, all patients who present with symptoms should be classified as belonging in one of the first three categories. If the visit cannot be categorized as due to either limit of anxiety or limit of tolerance, the physician should consider the possibility that he is dealing with *problems of living presenting as a symptom*. This type of presentation is often motivated by an unrecognized wish to hide the true nature of suffering. It is therefore necessary for the physician to inquire about personal problems and psychosocial aspects of the patient's life since the patient will not himself put them forward.

---

* Heterothetic means "putting forward other things."
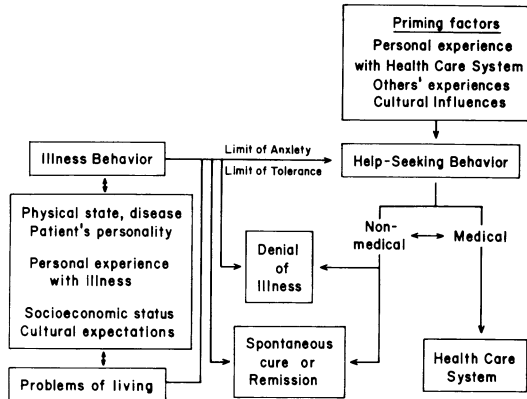
Figure 1. Help-seeking behavior.

Patient behavior is often a result of complex interpersonal interactions between the patient and his immediate environment (Figure 1). There are instances when the patient comes to the doctor not by choice but because he is forced by others. This is especially true in the case of mental disorders. For example, a schizophrenic patient may be brought to a doctor only because others cannot tolerate his bizarre behavior. Although McWhinney's classification system of patient behavior is intended primarily for individual patients, we can use the same scheme to classify patient behavior instigated by others, for example, limit of tolerance—family, limit of anxiety—employer, or problems of living—family.

> *Example 1.* The patient comes to the doctor because his wife is concerned about his weight loss and change in behavior (social withdrawal), although the patient himself feels that he is just tired because of overwork. He comes only because his wife insisted. Here, the behavioral classification would be limit of anxiety—wife; the patient's clinical diagnosis may be depressive syndrome (see Chapter 6).

> *Example 2.* The patient, a 7-year-old boy, is brought in by the parents because of frequent fights at school and minor accidents. Further inquiry reveals that these problems started a year ago when the father lost his job after an industrial accident. This, in turn, had led to financial difficulties and frequent fights between the parents. Behavioral classification: problems of living on the part of parents. Clinical diagnosis (child): transient situational reaction of childhood related to problems of living.

Reference to such a behavioral classification in parallel with medical diagnosis will help the physician identify the reasons for initiating the medical contact and alert him to the implicit as well as explicit expectations of both patient and family.

Summary

What one does in the presence of symptoms (illness behavior) is determined not only by the nature of the symptom(s) but also by demographic factors, presence or absence of stress, and, most important, previous experience with or exposure to similar symptoms or the health-care system or both (priming experiences).

Help-seeking behavior occurs when a person does something to get help for his symptom or distress. This may be medical (seeing a doctor) or nonmedical (talking with a friend, attending a prayer meeting). In medical help-seeking behavior, the immediate reason for the patient–doctor contact may be the patient's (or the family's) having reached the limit of tolerance for the symptoms or for the associated anxiety. On the other hand, the patient (family) may be emotionally distressed and more in need of *interpersonal support* than elimination of a benign symptom; this would be an example of problems of living presenting as a physical symptom.

Implications

For the Patient

The presence of a symptom may not by itself constitute a sufficient condition for seeking medical help. The interpretation of the symptom by the patient in the light of his own unique background and personal experience, together with the prevailing community expectations, determines the action. Many patients engage in nonmedical help-seeking behavior before consulting a physician. Contact with the physician represents, for many patients, a last resort—having reached the limit of anxiety or tolerance concerning the symptom. It may also be an indication of problems of living about which the patient is unaware.

For the Physician

Patients are different. What they do in the hospital or consulting room is determined by a number of factors that are not directly involved in

the disease process and its associated symptoms. *Demographic data* concerning the patient are important in enabling the physician to understand the patient's behavior in the context of the normative attitudes and expectations of his cultural and social groups. Information concerning the patient's *previous medical experiences* and his exposure to similar symptoms and to the health-care system (doctors, hospitals) may also be essential for understanding the patient and for effective communication with him. It is often necessary to clarify misunderstandings and misconceptions carried over by the patient from priming experiences before the patient can attend to the words and recommendations of the doctor. Recognition of problems of living presenting as a symptom will enable the physician to avoid unnecessary laboratory tests and medical procedures and to provide more effective support, such as simple listening and follow-up or referral to a psychiatrist. Remember that problems of living may not be apparent unless the patient is queried about his life situation. For example, it turned out that the woman in vignette 3 was undergoing menopause and had recently separated from her husband. It was these difficulties that had motivated her seeking help in the form of a request for surgery on her varicose veins. The operation, which was unnecessary, was postponed by her doctor, and she was referred for counseling and followed at regular intervals by the physician. In six months, she had resolved the difficulties with her husband, and her (initially urgent) request for operation was dropped.

For the Community and the Health-Care System

The underutilization of health-care services by lower socioeconomic classes calls for community efforts to increase their accessibility and acceptability, especially the preventive and educational facilities. Since cultural and ethnic factors play a role in help-seeking behavior, hospitals should, if possible, have members of various cultural and ethnic groups available to assist in communication between the health-care personnel and patients in need of their services. Nonmedical personnel (e.g., clergy, teachers) should be educated to be alert to the possible presence of medical disorders and to facilitate medical help-seeking behavior when it is indicated.

Recommended Reading

Hollingshead AP, Redlich FC: *Social Class and Mental Illness.* New York, John Wiley & Sons, 1958. A classic study on the relationship between social class and the prevalence of mental disorders in the psychiatric patient population in the New Haven area in 1950.

McWhinney IR: Beyond diagnosis. *N Engl J Med* **287**:384–387, 1972. The author proposes a taxonomy of medical help-seeking behavior that includes "heterothetic" presentation, or problems of living presenting as a symptom. In addition, he proposes a taxonomy of social factors in illness and medical help-seeking behavior, such as loss, conflict, and change.

Mechanic D: The concept of illness behavior. *J Chronic Dis* **15**:189–194, 1962. A relatively brief but comprehensive exposition of the concept of illness behavior. The author developed the concept of the basis of his findings concerning how students in a large university behaved differently according to demographic and psychosocial variables in the presence of a symptom.

Mechanic D: Social psychologic factors affecting the presentation of bodily complaints. *N Engl J Med* **286**:1132–1139, 1972. A brief but comprehensive review of cultural and social factors in illness behavior. There is also a brief discussion on the "medical student's disease."

Srole L, Langner T, Michael ST, *et al.: Mental Health in the Metropolis: The Midtown Manhattan Study*. New York, Harper Torch Books, 1975. Originally published in 1962, this study, somewhat similar in scope to the New Haven study, also utilized home-interview techniques. An important study showing a high prevalence of emotional distress (80%) in the general population. Demographic data are shown to be associated with prevalence and type of mental disorders. Low socioeconomic class and increasing age are related to higher prevalence of mental illness.

Zborowski M: *People in Pain*. San Francisco, Jossey-Bass, 1969. An easily readable, sometimes dramatic presentation of the ethnic differences in how people behave in the presence of pain. The author studied "Old Americans," Italian, Jewish, and Irish Americans who were being treated for pain in a Veterans Administration Hospital in New York City. Somewhat stereotyped and oversimplified.

References

Brightman I, Notkin H, Brumfield W, *et al.*: Knowledge and utilization of health resources by public assistance recipients. *Am J Public Health* **48**:188–199, 319–327, 1958.

Deasy L: Socioeconomic status and participation in the poliomyelitis vaccine trial. *Am Social Rev* **21**:185–191, 1956.

Hollingshead AP, Redlich FC: *Social Class and Mental Illness*. New York, John Wiley & Sons, 1958.

Koos E: *The Health of Regionville: What the People Thought and Did about It*. New York, Columbia University Press, 1954.

McWhinney IR: Beyond diagnosis. *N Engl J Med* **287**:384–387, 1972.

Mechanic D: The concept of illness behavior. *J Chronic Dis* **15**:189–194, 1962.

Mechanic D: *Medical Sociology: A Selective View*. New York, The Free Press, 1968.

Redlich F, Hollingshead A, Bellis E: Social class differences in attitudes toward psychiatry. *Am J Orthopsychiatry* **25**:60–70, 1955.

Rosenstock I: Prevention of illness and maintenance of health, in Kosa J, Antonovsky A, Zola IK (eds): *Poverty and Health*. Cambridge, Harvard University Press, 1969, pp. 168–190.

Ross J: Social class and medical care. *J Health Hum Behav* **2**:35–40, 1962.

Saunders L: *Cultural Differences and Medical Care.* New York, Russell Sage Foundation, 1954.
Shekelle R, Ostfeld AM: Social status and incidence of coronary heart disease. *J Chronic Dis* **22:**381–394, 1969.
Zborowski M: Cultural components in response to pain. *J Soc Issues* **8:**16–30, 1952.
Zborowski M: *People in Pain.* San Francisco, Jossey-Bass, 1969.

CHAPTER 2

The Sick Role

1. An epidemic of influenza swept through town. Absenteeism was highest among students, followed by white-collar workers, and lowest among the blue-collar workers at the steel mill, especially those with least seniority.

2. [A newspaper excerpt] Policemen in the city today called in sick in droves as negotiations between the city and the police union broke down.

3. A 35-year-old secretary, who had never missed a day's work, became noticeably pale and seemed to have trouble walking but would not miss work to see a doctor. Since she lived alone, there was no one to be alarmed when she started vomiting in the evening or when excruciating pains kept her awake at night. She would, however, invariably show up at work with a smile on her white parched lips. Then, one day she did not come to work. She had died of massive stomach bleeding in the early hours of the morning.

4. [The late Senator Hubert Humphrey, on returning to the Senate after discovery of inoperable cancer] "I must believe that I am going to win this battle. . . . I have to do it, nobody else can. The doctors can't do it for me, although they can help."

5. A 25-year-old man was admitted to the hospital when he came to the emergency room vomiting blood. All tests were negative, and he was discovered stealing a test tube of blood from a technician's cart, whereupon he immediately walked out of the hospital. Two days later, he was admitted to another hospital in an adjacent city after coming to that hospital's emergency room vomiting blood.

6. [In a bedroom] "No, not tonight. I have a terrible headache."

7. Having been found incompetent to stand trial, the defendant was committed to the mental hospital.

17

Sick-Role Expectations

Society assigns specific expectations to the ill person by virtue of his being ill. These expectations comprise what is known as the "sick role."

Talcott Parsons, a sociologist, formulated the sick role as consisting of four aspects. The first is an *exemption from normal social role responsibilities* (Parsons, 1951). The degree of such exemption is, of course, dependent on the nature and severity of the illness. This exemption, according to Parsons, is a right of the sick. In order to prevent abuse of this right (malingering), society also requires that this right be legitimized by others. The physician is most often the key person in this legitimizing process, both as a direct legitimizing agent and as a "court of appeal." The sick role may be assumed by some persons without having seen a doctor or having been recognized as ill (vignettes 2 and 6). In these instances, the legitimacy of the exemption from social responsibilities may be seen as tenuous; further, should such persons eventually seek medical help (after having assumed the sick role for prolonged periods without medical legitimization), the physician may be reluctant to grant legitimization. Thus, patients suspected of being "crocks" or "hysterics" tend not to be taken seriously by doctors even when their complaints might otherwise stimulate further investigation.

The second aspect of the sick role is *recognition "that the sick person cannot be expected by 'pulling himself together' to get well* by an act of decision or will. . . . He is in a condition that requires being 'taken care of.' His 'condition' must be changed, not merely his 'attitude.' Of course the process of recovery may be spontaneous, but while the illness lasts he can't 'help it.' This element in the definition of being ill is obviously crucial as a bridge to the acceptance of 'help'" (Parsons, 1951).

The third expectation is that *"the state of being ill* is . . . *undesirable . . . and the patient should want to 'get well.'"* According to Parsons, the first two elements of the sick role, the exemption from normal role responsibilities and legitimization of dependency on others, are contingent on the acceptance of the idea that the assumption of the sick role is an undesirable and unfortunate necessity that should be relinquished as soon as possible (vignette 4). Although the sick person cannot be expected to "pull himself together" to get well, he may be seen as responsible for his condition if he does not show motivation to get well, to get out of the sick role.

The fourth aspect of the sick role is the obligation—in proportion to the severity of the condition, of course—*to seek technically competent help,* usually from a physician, and to cooperate with him in the process of

trying to get well. It is here, of course, that the role of the sick person as patient becomes articulated with that of the physician in a complementary role structure.

Parsons attached great importance to motivational factors concerning the sick role, realizing that the privileges and exemptions of the sick role would give rise to attempts by some to assume the role for "secondary gain." The balance between the pull toward the sick role and the push toward relinquishing it is of utmost importance.

The sick role is considered to be a "contingent" role that anyone may assume regardless of his status in other respects.

In essence, then, the sick role consists of two rights and two obligations: the rights of exemption from normal responsibilities and the recognition that the ill person is not to blame for his illness, and the obligations to desire to get well and to seek competent medical help.

The sick role is seen as being "universalistic": generalized objective criteria determine whether one is or is not sick, and with what kind of sickness (Parsons, 1951).

When the physician recognizes a person as being ill, that person becomes a *patient*, legitimately assuming the sick role, exempted from normal responsibilities such as going to work, and expected to want to get well and to cooperate with the physician and other health-care personnel.

Sick-Role Performance by Patients

The sick-role expectations are obviously shared by the doctors and other health-care personnel. The patients who fail to conform to the sick role are considered to be "bad" patients. When a patient fails to conform, the legitimacy of the illness itself is often questioned—that is, the patient may be suspected of malingering. An uncooperative patient may be denied continuation of hospitalization by premature discharge because the physicians become angry with him for not living up to the fourth expectation. A convalescing patient who repeatedly complains of minor symptoms is seen as not fulfilling the third expectation to regard the sick role as an undesirable one that should be relinquished as soon as possible. This may even tempt the physician to withdraw legitimization of the sick role from such a patient. Some patients, on the other hand, may foster approved prolongation of the sick role by being "good" patients, that is, by extreme cooperativeness (for example, even volunteering to participate in experimental procedures).

The way a person actually behaves when he becomes sick may be conceptualized as *sick-role performance*. Sick-role preformance is influ-

enced by socioeconomic and ethnic–cultural factors (vignette 1). Persons
of deprived economic status might be reluctant to assume the sick role
because of loss of income and increased financial hardship. For persons
involved in industrial accidents, assumption of the sick role (for a le-
gitimate injury) may mean prolonged exemption from work and secure
income as long as the sickness continues. Students in training for careers
may be reluctant to assume the sick role due to possible disruption of
studies and delay in career achievement.

Personal experiences are also important in sick-role performance. Ex-
posure to persons who have derived benefit from being sick may pre-
dispose one to seek out sick-role exemptions and secondary gains when-
ever the opportunity presents itself. For example, a friend of a patient
who received a large compensation after an industrial accident may come
to the doctor with exaggerated lingering symptoms of a minor injury.
Patients who seem to want to prolong the sick role have often been
exposed to and envied the prerogatives of persons who had been se-
riously ill for long periods of time.

Society's view of a particular illness also affects whether a person is
more or less likely to be motivated to assume the sick role for the illness.
Patients suffering from socially stigmatized illnesses such as venereal
disease or mental illness may be reluctant to assume the sick role. Stig-
matized illnesses (such as venereal disease and alcoholism) are not
treated with neutral attitudes (as Parsons formulated), and they often
carry an assumption of responsibility for the illness on the patient's part.
Heterothetic presentation of symptoms (McWhinney; see Chapter 1)
may be seen as an attempt on the part of the patient with emotional
difficulties to assume some elements of the sick role.

The concept of the sick role as formulated by Parsons does not apply
to all illnesses. Specifically, it is *inadequate* to deal with (1) minor illnesses,
for which no exemption from normal social roles is granted and no
requirement to contact the physician is made; (2) incurable illnesses,
which require adjustment rather than a motivation to recover; and (3)
legitimate "ill" roles that do not require continuing attention by a phy-
sician or exemption from normal responsibilities or motivation toward
recovery, such as in the handicapped (Freidson, 1962). More recently,
physicians are becoming more and more aware that many illnesses occur
due to neglect on the part of potential patients of measures that would
clearly be preventive. Such neglect may be active, such as smoking de-
spite respiratory symptoms, or passive, such as not consulting a phy-
sician after discovering a lump in the breast. In this sense, society's sick-
role expectations concerning responsibility for illness may need revision.

Role-conflict problems are relatively frequent. For example, an elderly person may become very sick with a viral illness that usually runs a mild course. He may feel guilty if he assumes the sick role, and yet he may be chided by others if he does not.

We often see patients with incurable illnesses such as advanced cancer attempting to recover from the illness completely through the use of irrational methods of treatment (such as Laetrile)—and spending enormous amounts of money and energy in the attempt. This would be an example of conflict between the third and fourth expectations.

In psychiatric illnesses, there are divergent role expectations depending on the physician's orientation to the illness, with varying degrees of modification from Parson's sick role. A biologically oriented psychiatrist using mainly a medical model of psychiatric illness may legitimize the full assumption of the sick role for the mentally ill patient. On the other hand, psychiatrists with a more sociopsychological orientation may be reluctant to grant their patients exemptions from social role responsibilities and expect them to work at getting well through their own efforts assisted by the psychotherapist. Then, there are some who still believe that mental illness is a condition for which the patients are totally responsible, that they could "pull themselves together" if they really wanted to. This attitude contributes to the continuing stigmatization of mental illness and, consequently, the reluctance of the patients to seek help.

The sick role, like any other newly assumed role, requires a period of adjustment. In newly hospitalized patients, conflicts often arise between the patient and the staff because of the inability or difficulty of some patients to accept the sick role, which leads to the patient being perceived as uncooperative rather than inexperienced in the role.

The patient's personality has a major bearing on how he perceives and accepts the sick role (see Chapter 17).

Summary

Society's expectations of a person who becomes ill constitute the "sick role." Parsons describes four components of the sick role: (1) exemption from normal role responsibilities; (2) recognition that the sick person cannot be expected to get well by an act of will or decision on his part; (3) definition of the state of being ill as an undesirable state; and thus (4) the obligation to seek technically competent help from a physician.

As generalized social expectations, they are generally shared by both physicians and patients. The "sick-role performance," that is, how a patient actually behaves when he becomes sick, is determined by many factors besides the "sick-role expectations." These factors include the realities of economics, personal experiences with illness, the context of being ill, and the personality of the patient, to list a few. The sick-role performance of specific patients may be at variance with the sick-role expectations of society or of physicians, resulting in misunderstandings or conflicts between patients and health-care personnel.

Implications

For the Patient

The generalized sick-role expectations form the basis or "ground rules" for persons when they become ill. Thus, patients "know" that they can and should stay home from work in the presence of high fever and, if it continues, that they should go to the doctor; also, that they should take the medications prescribed by the doctor to get well. The sick-role performance of individual patients, however, is influenced by a number of individualized or class-dependent factors, including unique personal experience with illness or ill persons. Thus, some patients behave in ways at variance with the way they should behave according to society's expectations. For example, some patients may not seek help in the presence of serious symptoms (as in vignette 3); some may "assume" parts of the sick role as a means to a goal (e.g., the policemen in vignette 2), as an excuse to be exempted from normal role expectations (e.g., that of a spouse in vignette 6), or as a legitimate reason for such an exemption (vignette 7).

For the Physician

The physician should first *understand what his expectations are of the sick persons he is treating*, since they may influence his attitudes or generate prejudices concerning a particular patient. Then, the physician should attempt to understand the determinants of the patient's sick-role performance—the influence of socioeconomic class, personal experiences, contexts of the assumption of the sick role, and other factors. For example, did the patient delay seeking help (assumption of sick role) because of economic reasons? Is the patient seeking certain aspects of

the sick role (e.g., exemption from normal responsibilities) because of psychological needs (being unable to cope with a severe emotional stress)? *Many problem patients can be understood if the problem is analyzed in terms of the sick-role expectations of the physician and the sick-role perform-ance of the patient.* For example, in vignette 5, we see a patient who is "addicted" to the sick-role performance, assuming it by deception. Part of society's sick-role expectation is that it is an "undesirable state" and should not be voluntarily assumed in the absence of illness. Physicians, in general, tend to be angry at such patients. However, once the phy-sician recognizes that his anger is directed at the sick-role performance of the patient (not playing by the rules) and also that this performance gives him a sense of being "cheated" by the patient, he can recognize that the person's need to be hospitalized is itself an illness requiring treatment. With this perspective, the physician can then endeavor to understand the personal determinants of the sick-role performance of the patient, including the meaning of hospitalization.

For the Community and the Health-Care System

The generalized sick-role expectations as described by Parsons may be modified in specific communities and health-care systems. For ex-ample, in certain communities, the expectation that an ill person should seek professional help may not apply (e.g., a community of Christian Scientists). In certain hospitals for chronic or terminal diseases, the ex-pectation of hoping to get well may not be appropriate. A community may have latent prejudice against certain minority members who do not share the sick-role expectations of the larger community. It may also show prejudice against persons who cannot fulfill the community's sick-role expectations when they become sick. For example, patients with chronic or terminal diseases or some geriatric patients often are not treated with the same degree of compassion and caring that more acutely ill persons receive in the community.

Generalized sick-role expectations of the community change as we become more aware that certain diseases might have been prevented but for the patient's inaction—for example, failing to stop smoking— or might have been precipitated by patients' actions—for example, be-coming malnourished because of a dietary fad.

In understanding "problem" patients in a given health-care system, it is important to understand what the sick-role expectations of that system are and how they interact with the patient's sick-role perform-ance. Many problems may turn out to be a conflict between the expec-

tations of the health-care system as to how patients should behave and the patients' expectations (determined by their own unique experiences) as to how they should behave and be treated.

Recommended Reading

Parsons T: *The Social System.* New York, The Free Press, 1951, Chapt X (Social structure and dynamic process: The case of modern medical practice), pp 428–479. This chapter by Parsons is a classic that lucidly formulates the concept of the sick role and the social expectations of the doctor's role; it also contains a thoughtful functional analysis of the institution of medicine. Very highly recommended.

Twaddle AC: The concepts of sick role and illness behavior, in Lipowski ZJ (ed): *Advances in Psychosomatic Medicine.* Vol 8: *Psychosocial Aspects of Physical Illness.* Basel, Karger, 1972, pp 162–179. This is a succinct summary of the concepts and controversies concerning the sick role and illness behavior. With extensive references.

References

Freidson E: The sociology of medicine. *Curr Sociol* **x/xi**:3, 1962.
Parsons T: *The Social System.* New York, The Free Press, 1951.

CHAPTER 3

Expectations in the Consulting Room

1. A 32-year-old woman came to her doctor for low back pain of one month's duration. Physical examination was within normal limits except for a small hard lump in her left breast. The physician suspected a malignant breast tumor and referred her to a surgeon, who admitted her to the hospital for breast biopsy. On admission, the patient was noted to be anxious and reticent. When the admitting intern asked why she came to the hospital, she said, "I don't know. I've had this back pain for a month or so—after straining it lifting a heavy suitcase. I guess you want to do surgery to straighten my back?" When the intern asked about the lump in her breast, she said, "Yes, my doctor told me about that. It doesn't hurt or anything, and I told him not to bother. He said it should be checked just to be sure, though. But my question is what are you going to do about my back pain?" The chart revealed the following note by the intern: "This 32-year-old white female was admitted to the hospital with the chief complaint of a lump in the breast."

2. A conversation at the bedside of a patient in a community hospital. Intern: "This is the 58-year-old gentleman with possible mitotic lesions of the liver." Attending: "Hello, Mr. X. How are you this morning?" Patient: "I am fine . . . er, much better, I think, Doc. I think I am getting a little less yellow now . . . maybe the X-rays yesterday helped." Attending: "Fine, I think things are moving along O.K., Mr. X." Intern to Attending: "Mr. X had chest X-rays yesterday for possible mets—negative." Attending to Intern: "Ought to repeat the LFT, lytes, and bilirubin and get a GI consult."

3. A 38-year-old woman visited her family doctor complaining of severe migraine headaches. She had had migraine for more than ten years, but both

the frequency and intensity had increased during the past three months. The intensity had increased to the point where she had nearly collapsed on several occasions. She made three visits to the doctor's office in three weeks, urgently asking him to "do something" about the headaches. Careful physical examination was within normal limits, and the headaches did not respond to the usual antimigraine regimen despite good results in the past. Finally, her doctor decided to ask her specific questions about any changes in her life during the past several months. Initially, she denied any major changes, but when questioned about personal habits, including sexual activity, she revealed that she had been involved in an extramarital affair for four months and that she felt intensely guilty and worried. Having shared her concerns about the affair, she was assured that the physician would help her to deal with her personal problems. The migraine headaches decreased in frequency and intensity.

A patient brings to the consultation with the physician both overt and covert expectations of the doctor. Conversely, the physician has expectations of the patient consonant with societal expectations about the *sick role* and automatically expects the patient to behave according to them (see Chapter 2). Especially important is the expectation that the patient will *cooperate* with the doctor to get well. Often there is an associated implicit idea that patients should and will be *candid* with physicians and that they will *volunteer* pertinent information. Because of this implicit expectation, some *doctors may fail to inquire* into personal situations and matters that may be relevant to help-seeking behavior. In vignette 3, if the doctor had *not* specifically asked about the patient's sexual life, a successful outcome would not have been likely.

Some of the patient's expectations in the consulting room manifest themselves only when they are not met, that is, when the patient is frustrated. An important conscious attitude shared by patient and doctor alike is that of *hope*; the patient's expectant hope that the doctor can help him and the doctor's hopeful expectation that he can do something for the patient (Freidson, 1970). *Relief from distress* usually holds top priority for the immediate help that the patient wishes and seeks to gain by consulting the physician. Implicit in this hope, of course, is the expectation that the doctor will be competent and that he will be interested.

In this chapter, we will discuss some of the expectations brought to the consulting room and examine how divergence or incongruity of such expectations between patient and doctor can interfere with optimal medical care. In the course of the discussion, expectations of the physician, that is, the "doctor role," will also be examined.

Relief from Distress

As noted above, the immediate expectation of the patient is that the doctor will relieve his distress. This may, in fact, occur to a considerable degree simply by the experience of visiting the physician, even before the symptom or underlying disease has been changed. Freidson states that because of the mutual hope that the doctor can and will do something to help the patient, both patient and doctor are motivated to believe that something effective has occurred during the doctor–patient contact (Freidson, 1970). Consequently, according to Freidson, the patient may feel better and the physician may consider his ministrations to have been responsible (whether or not this was actually the case). But if the patient does not, in follow-up contacts, perceive that specific procedures to alleviate or remove the symptom are being instituted or planned, the distress may again increase.

The physician's expectations generally focus on a related but somewhat differently timed process: relieving the patient's distress *by treating the underlying disease*. This means that the doctor's inclination is first to identify and then to treat the underlying disease, with relief following naturally. Here, then, is the potential for a covert difference in priorities between the patient (relief from distress) and the doctor (treatment of underlying disease). Such a situation is particularly well illustrated when the physician diagnoses a serious medical condition unrelated to the immediate (coincidental benign) distress that brought the patient to him and then understandably subordinates the treatment of the immediately distressing, but minor, condition to the treatment of the serious one (vignette 1).

Depending on the nature of the symptom and the level of sophistication, most patients, of course, also expect treatment of the underlying cause. This may hold first priority for some patients, particularly those coming because of "limit of anxiety," but it most often takes second place to more immediate relief of discomfort. Covert differences in expectations can, of course, lead to difficulties in the doctor–patient relationship and interfere with the treatment process. Clearly, problems of this nature can be prevented or corrected by effective and open *communication*, and it is the doctor's responsibility to be alert to this phenomenon and to take the initiative in dealing with it.

Physicians expect their patients to cooperate with recommended diagnostic and treatment regimens, and patients do, indeed, expect to render such cooperation and compliance with the doctor's instructions.

This cooperation, however, is *conditional* on their understanding that such cooperation will eventually result in relief from distress.

Physicians expect (and require) that patients will provide them with information concerning past history, history of present illness, and other aspects of their illness to aid in the diagnosis and assessment of the illness, but patients do not know the relevant details and kinds of information that are needed. Yet some patients have an *implicit expectation* that the doctor will *divine* the source of their distress and do not expect to give detailed specific descriptions and history. Furthermore, body sensations are difficult to describe in words, even when the sensations are not threatening. Such communication problems can only be ameliorated by the physician's skill and patience in taking a medical history. This is an art that the good physician works an entire lifetime to perfect. Patients expect the physician to *communicate* diagnostic findings and therapeutic plans as well as to manifest an *interest* in providing relief. Communicating about diagnosis and treatment may be difficult and often requires considerable thought and planning. It can be misleading to assume that it is sufficient to convey only as much as the patient appears to be interested in and capable of understanding. Patients' interest in information, and their capacity for understanding, tend to be underestimated by physicians (Pratt *et al.*, 1957), and the amount of information received from the physician seems to influence the patient's readiness to comply with the physician's orders—the less information furnished by the doctor relative to the amount provided by the patient, the less compliance the patient is likely to show (Davis, 1968).

The physician may overestimate the strength of the patient's blind trust or the extent of his understanding. Overestimating the patient's trust leads to the unwarranted assumption that the patient will understand that every procedure and advice the doctor gives is aimed at relief from distress in the long run. The physician may then fail to make connections for the patient and express the intent to eventually relieve his distress. Overestimating the patient's level of understanding and sophistication leads to an assumption that the explanations are unnecessary, again resulting in neglect of full communication.

Many studies indicate that there is a higher level of dissatisfaction on the part of patients about the amount of information physicians provide than about any other aspect of medical care (Cartwright, 1964; Duff and Hollingshead, 1968; Waitzkin and Stoeckle, 1972). In addition to the factors discussed above, Waitzkin and Stoeckle (1972) postulate that the feeling of power arising from keeping the patient uncertain and uninformed may (unwittingly) motivate some physicians to withhold information.

The problems of frustrated expectations generally arise from problems in communication. In essence, both patient and doctor have the same and/or mutually syntonic central expectations, but the physician's intentions are often *implicit* and need to be made *explicit*. Treating the underlying illness first will lead to relief of symptoms; there is no inherent conflict, and, in fact, one follows the other. The patient, however, does not understand how an immediately distressful and seemingly irrelevant procedure like a breast biopsy (vignette 1) might eventually help relieve the felt distress of low back pain *unless* the physician explains *explicitly* that (1) the lump in the breast may be unrelated to the back pain but needs to be evaluated before it becomes a problem; (2) although unlikely, the back pain may be related to the lump in the breast (metastatic cancer), in which case, both need to be treated; and (3) in any case, the doctor *will* treat the back pain with medications. Physicians do attend exclusively to relief of symptoms when it is necessary, that is, when prompt treatment of the underlying cause is not possible.

Inaccurate estimation of patients' desire to receive information is particularly problematic in dealing with patients of *lower socioeconomic status*. Cartwright (1964) reports that professional white-collar workers obtained most of their medical information by asking their physicians and nurses direct questions, white blue-collar workers received such information through a passive process not involving active asking and consequently tended to receive less information than the upper classes. He attributes this diffidence of the lower class concerning medical personnel to four factors: (1) their sense that doctors do not expect them to ask questions, (2) a problem of language that results from their unfamiliarity with the technical terms doctors use, (3) the awe with which they regard physicians, and (4) their social distance from physicians' higher social class. This reluctance to engage in active information-seeking behavior is often misinterpreted by the physician as a lack of interest. But it should be emphasized that *there is no general class difference in patients' desire for as much medical information as possible* presented in nontechnical language. "Good explanation" of illness is considered to be one of the most important qualities of a "good doctor" by a majority of hospitalized patients (Skipper and Leonard, 1965).

Differences in language skills and pattern of *linguistic use* may also contribute to the phenomenon of the lower-class patients receiving inadequate information from physicians. There are two basic linguistic codes differentially used by the middle and working classes (Bernstein, 1964). The *elaborated code* refers to the mode of speech in which the speaker selects from a wide range of syntactic alternatives, and it renders itself more easily to descriptions and reasoning based on the content of

the speech. The *restricted code,* on the other hand, has a reduced range of alternatives and syntactic options, and the vocabulary tends to be drawn from a narrow range. The restricted code tends to discourage verbal elaboration and discussion. The elaborated code also often involves "expression of intent," while nonverbal signals are usually used for this purpose in the restricted code. The elaborated code uses higher levels of abstractions than the restricted code. The elaborated code, then, is the linguistic style used by the middle class, while the lower class tends to use the restricted code.

Physicians, using an elaborated code, are likely to expect patients to express intent verbally, while lower-class patients, using a restricted code, are not accustomed to making such verbal requests. Thus, their expectations, expressed nonverbally, are likely to be frustrated.

The physician often concludes incorrectly on the basis of the restricted-code language used by lower-class patients, with its usage of a lower level of abstraction and a lack of verbal expression of intention, that the patient lacks the *competence* to understand his explanations and that the patient also lacks the desire to know about his disease processes and plans for its treatment (Waitzkin and Stoeckle, 1972).

Actually, this difference in language use between the classes is largely one of *performance* rather than of competence. Chomsky (1965) uses the term "performance" to refer to language use in concrete, specific situations, while "competence" is used to refer to the person's actual knowledge of language. Although a lower-class person is more accustomed to the restricted code in speaking, influenced by early experiences and current practice of his social station, he is nevertheless usually able to understand the elaborated code of the upper classes.

Communication of Information

Reference was made above to patients' desire to know about illness in nontechnical language. Even if a patient belongs to the middle class, with its elaborated code, he still has a class difference from the physician—doctors belong to a special closed class whose members habitually speak in a language comprehended only by its own members. Technical language or jargon serves several useful *functions.* One, among others, is setting the context of meaning (in this case, medical) and being specific. For example, carcinoma of the cervix refers to a particular form of cancer arising from a specific area of the female genital organ, and it is clear that this term is used in a medical context.

When communicating with nonmedical persons (patient or family or both), however, the doctor may encounter problems in attempting

to translate medical jargon into nontechnical language. One problem with such translations is the loss of *specificity*. In the example given above, translation of "carcinoma of the cervix" to "cancer of the womb" clearly loses the specificity concerning the type and the exact site of the lesion. This can give rise to confusion in the mind of the lay person, who may have heard the same term applied to such diverse conditions as endometrial carcinoma, fibroadenoma (considering that "tumor" is often used synonymously with cancer), ovarian tumor (some lay people may confuse the womb and ovaries), and carcinoma of the vagina. Of course, there are marked differences in the course, treatment, and prognoses of these conditions.

Translation to nontechnical language is nonetheless desirable, since the use of jargon results in both noncommunication and misinterpretation. It is important to recognize that in translating medical jargon to lay terms, specificity is very often lost and that the person is likely to have his *own fantasies and ideas* about what the doctor is telling him. For information to be communicated accurately, therefore, it is essential that the physician attempt to be as specific as possible and that he *ask* the lay person what he *understood* from the explanation provided. This will provide an opportunity for prompt clarification of possible misunderstandings and misinterpretations.

Physicians sometimes use medical jargon deliberately when they are communicating with each other in the presence of the patient. This can be very risky. *If the patient is not provided with sufficient information, he will attempt to construct his own meaning out of whatever he heard*. Just imagine for yourself the fantastic notions the patient in vignette 2 might have developed from the wholly unfamiliar terms he heard the doctors using.

Another problem with translation of medical terms into lay terms is that the loss of medical context may result in the concomitant loss of "affective neutrality" (see below). This is obvious when one considers the nontechnical terms that denote such body organs as the uterus, vagina, and esophagus. With the popularization of medical terminology in nonmedical populations, however, this particular problem has largely abated. Nonetheless, some terms may still run into difficulties in translation (e.g., "mortality rate," "prognosis"). Medical context should be kept in translations as much as possible.

Effect of Priming Factors on Expectations

Previous experiences with illness, physicians, and hospitals also determine the patient's expectations in the consulting room. This is es-

pecially so if the present illness to be treated is in any way related to the previous experience—for example, same symptoms, same body organ, or same physician.

The physician's expectations are also influenced by his own previous experiences with the particular type of disease or symptoms and even with the personality type of the patient.

The Physician's Covert Expectations

In addition to the shared expectations of the doctor and patient, some physicians have additional covert expectations of patients. For example, some expect that the patient *should be suffering* to see a doctor and that the suffering must be *physical*. In such a case, if a patient visits the physician with a minor symptom from which little suffering is evident, he may be viewed with suspicion or even derision because of this expectation. When the expectation is that the suffering must be physical, there is a tendency not to consider the possibility that the presenting symptoms might be heterothetic, that is, problems of living presenting as a symptom. Such expectations on the physician's part, then, may result in *inadequate or delayed diagnosis* through the lack of vigilance and concern.

Society's Expectations of the Physician—The "Doctor Role"

Society has certain expectations of the conduct of physicians just as it does of ill persons. These will be referred to as the "doctor role" to complement the "sick role" of the patient.

Parsons (1951) elucidated the role of the physician together with the sick role in his book *The Social System*. According to Parsons, there are five essential aspects to the role of the physician. They are discussed below.

Technical Competence

The physician is expected to facilitate the patient's recovery from illness to the best of his ability. To meet this responsibility, he is expected to acquire and practice high technical competence in "medical science" and the techniques based on it.

Parsons points out that this can be a cause of frustration for the physician because of the inherent uncertainties in medicine and because scientific advances do not necessarily result in an increase in the physician's ability to facilitate recovery from illness.

In the context of "doing everything possible," the physician is *exempted from certain social prohibitions*. These include the need to invade the patient's *privacy* in handling and examining his body (physicians are allowed to look and feel and otherwise explore another person's body in ways barred even to a spouse or lover), to acquire confidential and personal information from the patient, and to subject the patient's body to discomfort and injury (such as surgical procedures).

Universalism

Parsons characterized the doctor role as being "universalistic," as opposed to "particularistic," in two senses. First, this role is *open to anyone* who meets the performance criteria. This tends to reduce nepotism and to facilite interdisciplinary communication and thus the furtherance of medical science. Second, the universalism of the role *protects the physician* from "assimilation to the nexus of personal relationships in which the patient is placed." This particular aspect also implies that the treatment the physician renders is universal; that is, he renders his professional services to *any patient*, not just to friends and relatives. This is clearly related to the functional specificity and affective neutrality described below.

Functional Specificity

"Specificity of competence" and "specificity of the scope of concern" are considered under this rubric. The former refers to the expectation that the physician will practice only the techniques and areas of medicine in which he is competent, and it also involves his right not to treat patients requiring skills he does not possess.

Through expectations concerning the specificity of the scope, the physician is expected to avail himself of the privileges, such as the exemption from the prohibitions concerning invasion of privacy, *only for the purpose of medical care*. This expectation tends to allay anxieties on the patient's part about being exploited by the physician.

Society supports the maintenance of the functional specificity of the physician by "segregation of the context of professional practice from

other contexts." Thus, information gained in the context of medical prac-
tice is expected to be privileged and confidential, and situations sugges-
tive of sexual or aggressive encounters in other contexts are perceived
differently in the medical context, for example, having patients of the
opposite sex undress in the same room or cutting a person's skin with
a knife.

"The importance of functional specificity is to define the limits of
the 'privileges' in the 'dangerous' area which the physician might
claim," in situations where potential illegitimate involvements might
develop.

Affective neutrality is considered to be the expected attitude of the
physicians within these limits.

Affective Neutrality

This refers to the expectation that physicians will maintain *objectivity*
in regard to their patients and will not become "emotionally involved."
Included in this are the expectations that the doctor will treat his patients
equally, whether he likes them personally or not; that he will not become
emotionally aroused in the course of his professional activity (such as
erotic arousal); and that he will not reciprocate some patients' pull to
become more "intimate" with them, such as becoming personal friends.
Parsons sees a similarity between this and the affective neutrality es-
sential in psychotherapy situations and infers that a functional signifi-
cance of this aspect in medical practice might be that *there is a certain
amount of "unconscious psychotherapy" in all medical practice*.

Affective neutrality does not mean that the physician should ex-
press no concern about his patient, but rather that this attitude is ex-
pected to be one of *professional* concern.

Collectivity Orientation

This refers to the service orientation of the physician to *subordinate
his own personal gain to the welfare of the patient*. The collectivity orientation
is considered to be the foundation of the "trust" that the patient is
expected to have in his physician. Parsons states that this orientation is
found in all cases of institutionalized authority. In the doctor–patient
relationship, this authority is legitimized in a reciprocal relationship—
the doctor has the "obligation faithfully to accept" the implications of
the fact that he is the patient's doctor.

The significance of this orientation is in allowing the development of a trusting relationship between the doctor and the patient by reducing the threat of exploitation on the doctor's part.

In summary, then, society accords the physician certain *privileges* in helping the patient, accompanied by the expectation that he has high *technical competence*, that he will treat his patients scientifically, using *objective criteria* for diagnosis and treatment rather than personal feelings, that he will treat patients *only in the areas in which he has professional competence* and not spread his practice and areas of competence too thin, that he will maintain *objectivity* concerning patients by not becoming personally involved with them, and that he will always put the *welfare of the patient* before his own welfare in the practice of medicine.

Summary

Physicians and patients have the mutual expectation that the doctor will *help* the patient. The patient's priority is usually *relief* from suffering or distress. The physician's priority is generally the treatment of underlying *disease*, which is then expected to result in relief from distress. Neglect of the patient's main concern of relief from distress can result in a strained doctor–patient relationship.

In assuming the sick role, the patient expects to offer cooperation and compliance to the doctor, in return for some assurance that attempts will be made to relieve the distress. Effective doctor–patient *communication* and *information exchange* are necessary for optimal cooperation and compliance of the patient and effective treatment. Factors that can impede effective communication include differences in *social class* and *language style* (elaborated code vs. restricted code) and problems related to *medical jargon*.

Previous experiences with illness, doctors, and hospitals determine the patient's unique expectations in the consulting room. The physician's past experiences also influence his own expectations about a particular class of patients or illnesses. Many physicians have the covert expectation that distress should stem from physical causes, and patients with distress due to psychosocial events might, unfortunately, be viewed with negative bias.

Doctors are expected, in general, to fulfill the doctor-role expectations: (1) technical competence; (2) universalism, (3) functional specificity; (4) affective neutrality; and (5) collectivity orientation.

Implications

For the Patient

When a person consults a doctor, he has certain expectations about what the doctor will do for him, how he should behave in becoming a patient, and how the doctor will behave as a professional. Some of the expectations are generally shared by most patients, while others are uniquely personal, based on "priming experiences." Some general expectations include *relief from distress, treatment of the underlying cause of the symptom producing the distress, cooperation,* and *compliance* with the physician who is *competent* and *interested* in helping the patient, and *communication* of information from the physician. The patient may feel that the doctor is uninterested in relieving his immediate distress or incompetent if he does not understand that the doctor's concern over the underlying disease is a necessary step in the ultimate relief from his distress. This danger arises especially if there is a problem in communication between the doctor and the patient.

For the Physician

The physician should be aware that the *first priority for the patient* in seeking medical help is relief from distress. The physician should effectively *communicate* to the patient his interest in relieving the distress and his *overall plans* in evaluating and managing the patient. He should recognize that the social class and language differences (related both to the social class and to medical jargon) can interfere with effective communication with the patient. After imparting information to a patient, the physician should specifically *ask the patient to explain, in his own words,* what he understands the information to mean. In general, patients are interested in *receiving more information than they actually request.* The physician should *ask specific questions* about the patient's experiences with illness, doctors, and health-care systems to understand the implication of individual "priming factors" for expectations. He should also understand the effect of his own past experiences on his expectations about patients and this particular patient. The physician should also be aware of the *doctor role,* which defines what is generally expected of physicians by the patients and by society.

Above all, the physician should recognize that the *expectations* and *hopes* of both the doctor and the patient are essentially the same, and

he should form a *collaborative alliance* with the patient in planning evaluation and management.

For the Community and the Health-Care System

The health-care system should recognize that the "problem" patient is often a person whose adjustment to the sick role (expectations of health-care personnel) is difficult. Recognition of its own expectations of the patient and understanding the patient's personality traits that may make it difficult for him to meet these expectations can resolve an impasse (see Chapter 17). Medical education should emphasize relief of distress as well as treatment of underlying disease, to facilitate collaboration between physicians and patients. Hospitals and medical schools should devise methods of more effective *communication* with patients concerning medical matters such as diagnostic and treatment procedures. Health-policy planning should include considerations of the impact on the doctor role, and the sick role and of the expectations on both sides and how they might change as a response to policy decisions (e.g., advertising by doctors). Attempts should be made to prevent an increase in the frustration levels of patients concerning their expectations that the physician will be interested in helping them as persons as well as in treating their disease.

Recommended Reading

Freidson E: *Profession of Medicine: A Study of the Sociology of Applied Knowledge.* New York, Dodd Mead & Co., 1970, Chapts 10 (Illness as social deviance) and 11 (The professional construction of concepts of illness). Freidson is a sociologist who is a leading critic of Talcott Parsons. Although some of his writing is quite unsympathetic to the medical profession, there are very astute observations, such as that physicians are more likely to see illness than to diagnose normality, and this tendency may result in overuse of medical technologies (such as unnecessary surgery). Illness is seen as a social state, and a point is made that an idea about what is normal, desirable, and moral is essential to considering what an illness is.

Parsons T: *The Social System.* New York, The Free Press, 1951, Chap X (Social structure and dynamic process: The case of modern medical practice), pp 428–479. A lucid discussion of the "doctor role" with elaboration on the five expectations. Discussion also of the sick role. A must reading.

Waitzkin H, Stoeckle JD: The communication of information about illness, in Lipowski ZJ (ed): *Advances in Psychosomatic Medicine.* Vol. 8: *Psychosocial Aspects of Physical Illness.* Basel, Karger, 1972, pp 180–215. A good discussion concerning the communication of information about illness to patients. A review article.

References

Bernstein B: Elaborated and restricted codes: Their social origins and some consequences. *Am Anthropol* **6:**55–69, 1964.

Cartwright A: *Human Relations and Hospital Care.* London, Routledge & Kegan Paul, 1964.

Chomsky N: *Aspects of the Theory of Syntax.* Cambridge, MIT Press, 1965.

Davis MS: Variations in patients' compliance with doctors' advice: An empirical analysis of patterns of communication. *Am J Public Health* **58:**274–288, 1968.

Duff RS, Hollingshead AB: *Sickness and Society.* New York, Harper & Row, 1968.

Freidson E: *Profession of Medicine: A Study of the Sociology of Applied Knowledge.* New York, Dodd Mead & Co, 1970.

Parsons T: *The Social System.* New York, The Free Press, 1951, pp 428–479.

Pratt L, Seligman A, Reader G: Physicians' views on the level of medical information among patients. *Am J Public Health* **47:**1277–1283, 1957.

Skipper JK, Leonard RC (eds): *Social Interaction and Patient Care.* Philadelphia, Lippincott, 1965.

Waitzkin H, Stoeckle JD: The communication of information about illness, in Lipowski ZJ (ed): *Advances in Psychosomatic Medicine.* Vol. 8: *Psychosocial Aspects of Physical Illness.* Basel, Karger, 1972, pp 180–215.

ON BEING A PATIENT
Psychophysiological Considerations

Anxiety

1. A 40-year-old woman came to the doctor with the following complaint: "I am afraid my heart will burst and that I will drop dead." On examination, mild hypertension and rapid pulse were noted. After thorough workup and study of the patient, the physician concluded that anxiety was probably the most important factor in producing her symptoms.

2. An attractive female college student, 22 years old, told her physician that she felt she needed sex therapy. She had had difficulty achieving orgasm during intercourse for several years and now found herself withdrawing socially because of fear of getting into sexual relationships in which she would feel frustrated. She was successful and a high achiever in all other areas, for example, scholastic activity, and felt that she should achieve orgasm every time she had intercourse. The physician's impression was that her anxiety concerning sexual performance was interfering with her ability to enjoy it fully.

3. A 30-year-old housewife would develop overwhelming panic whenever she went out of the house alone, particularly in crowded places. She refused to leave her house unless her husband would go with her and stay by her side. Her physician diagnosed her condition as agoraphobia and referred her to a psychiatrist.

Some writers have labeled our present times "the age of anxiety." "Anxiety" is one of our most commonly used words, and virtually everyone is familiar with it. Like pain, everyone has experienced it, and all wish to avoid it. At present, antianxiety medications are the most commonly prescribed drugs in this country. Anxiety is important in medical practice because it constitutes one of the most common, but often unrecognized,

reasons for seeking medical help. In addition to the familiar and easily recognizable symptoms it may produce, it may also contribute to the development of a myraid of physical symptoms that patients may not attribute to it.

While a great deal is known about this ubiquitous and highly important phenomenon of anxiety, we still have not achieved a fully and satisfactorily integrated understanding of its nature or of the pathophysiological mechanisms whereby it may both induce and influence as well as arise from physical dysfunction (disease). In this chapter, we will first discuss some relevant theories and various aspects of anxiety—that is, its phenomenology, central neurophysiology and neurochemistry, peripheral physiology, function, and regulation (and dysregulation)—and then turn to its clinical evaluation, diagnosis, and management. To discuss so many aspects of such a complex phenomenon presents a problem similar to that presented in the fable of the blind men and the elephant: the very same thing can seem so different depending on which aspect one apprehends and on the amount and kind of detail that various approaches can elucidate (i.e., on the relative technical and theoretical sophistication of different disciplines). In discussing the various aspects of anxiety in the sections that follow, we have attempted to balance presentation of detail in relation to sketching overall patterns in such a way as to achieve maximum relevance for the physician.

Phenomenology of Anxiety

As in vignette 1, the most prominent subjective feature of anxiety is identical to what is experienced in the *emotion of fear*—namely, a sense of dread and apprehension. This fearful feeling is usually vague and diffuse, but it may also focus on a specific idea, such as fear of dying, or of cardiac arrest, or of having a dreadful disease such as cancer. When the patient is questioned carefully, one can often determine that the vague feeling of dread came first and was later followed by more specific thoughts and ideas such as those mentioned above. *Physiological changes* are part of anxiety. They are mediated by activation of the central and autonomic nervous systems and of neuroendocrine mechanisms. In a fully developed reaction, all structures influenced by these systems may show functional changes. Thus, the symptoms and signs may include rapid pulse, increased blood pressure, excessive sweating, change in bowel function, changes in appetite, trouble sleeping, and difficulty breathing. In essence, then, *subjective feelings of dread and fear* accom-

panied by *symptoms* and objective *signs* of appropriate physiological changes indicate the presence of anxiety.

Theories of Anxiety and Emotions

Since anxiety is an emotion, we should first take up some general considerations about emotions. *Emotions*, or *affects* as they are sometimes otherwise called, include three main components: (1) a subjective state of mind or feeling tone (e.g., dysphoria, dread, awe), (2) a neurovegetative motor discharge (e.g., increased heart rate), and (3) perception by the person of the bodily sensations caused by the motor discharge (e.g., palpitation of the heart). Various theories relate these three components to one another in different ways.

The James–Lange theory of emotions, proposed in the late 19th century by the famous psychologist William James and the Danish physician Carl Lange, postulated that bodily changes (motor component) follow directly the perception of an exciting event and that the person's perception of the bodily changes (sensory component) is what we call
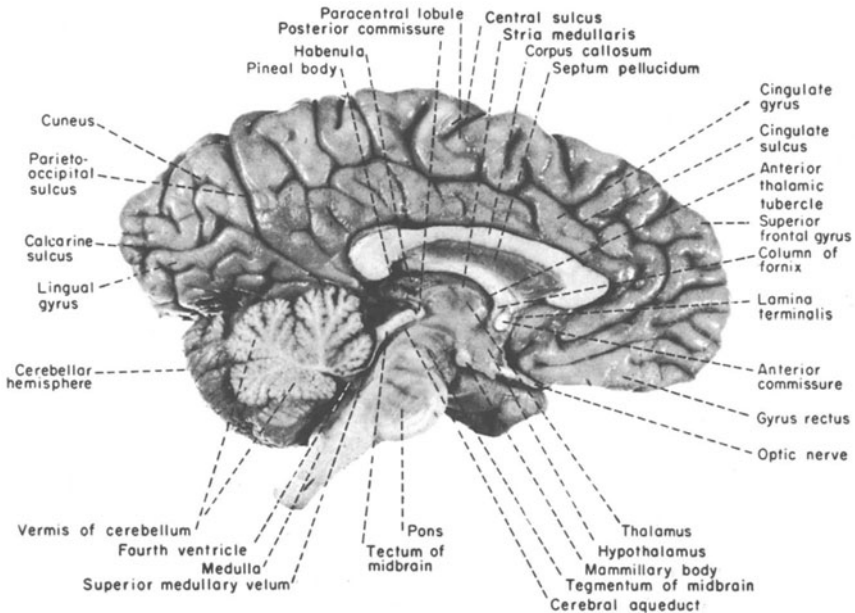
Figure 2. Photograph of the medial surface of the adult brain cut in sagittal section. (From Everett *et al.*, 1971. Copyright 1971 by Lea & Febiger. Reproduced with permission from the publisher and the author's estate.)
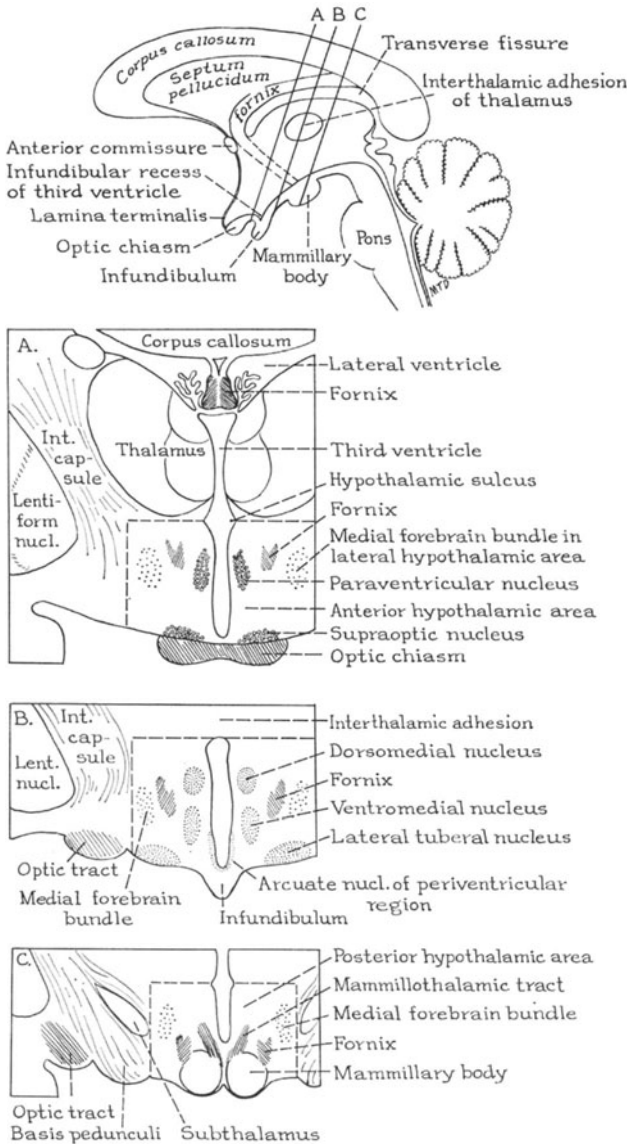
Figure 3. Schematic drawing of a sagittal section of the brain stem showing the relations of the hypothalamus to surrounding brain structures. Lines A, B, and C correspond to the levels at which the three lower drawings were made. Section A is through the supraoptic area, B is through the tuberal area, and C is through the mammillary area of the hypothalamus. (From Everett *et al.*, 1971. Copyright 1971 by Lea & Febiger. Reproduced with permission from the publisher and the author's estate.)

"emotion" (James and Lange, 1922). According to this theory, changes in the viscera or skeletal muscles or both were thought to be essential for the occurrence of emotions. The subjective component was omitted or assumed to be part of the sensory component. These assumptions were later put to test and disproved by Walter Cannon, the famous American physiologist and also William James's son-in-law. He showed that emotions were felt in persons who had had upper cervical spinal cord transections (thus effectively cutting off all the afferents from the viscera and skeletal muscles) and that in normal persons, the latent periods for the bodily changes were much longer than for the felt emotions (i.e., emotions occurred before bodily changes, not after). This led Cannon to believe that perceptions of noxious stimuli at the level of the *thalamus* (Figures 2 and 3) in the brain led to two distinct pathways of excitation, one upward to the cortex, adding affective quality to the experience through associations with memory traces, etc., and a downward discharge from the thalamus effecting redistribution of blood to viscera and skeletal muscles and changes in metabolism (e.g., rise in blood sugar)—all of such a nature as to prepare the animal for the vigorous muscular work involved in attacking (*fight*) or getting away from (*flight*) the danger. Cannon made extensive investigations into the fight–flight responses, which he felt were related to the basic emotions of displeasure, anger, and fear (anxiety) (Cannon, 1932). Cannon's theory concerning emotions was later modified by Papez (1949) and MacLean (1949), placing more emphasis on the limbic brain structures than on the thalamus (see Figure 4). This *limbic system model* forms the basis of modern neurophysiological theories concerning emotions (see the section on Brain Mechanisms of Anxiety).

The two major theories that address the psychological mechanism (higher cortical function of the brain) by which situations may be *perceived* and *appraised as dangerous*, leading to the generation of anxiety as an emotion, are (1) the *learning-theory or conditioning model* of anxiety based on the studies of Pavlov (1927) and (2) the modern *psychoanalytic theory* of anxiety ("signal theory") first articulated by Freud in 1926 (Freud, 1926/1953).

Learning-Theory Model

When a stimulus is inherently dangerous, such as the sight of a growling tiger, a fear response occurs naturally. A stimulus that is neutral and not inherently dangerous, such as open spaces or elevators, can become associated with a dangerous situation through a process called

"conditioning." According to the learning-theory model, then, anxiety is a *conditioned fear response*.

The *conditioning process* by which a neutral stimulus becomes aversive, creating a fear response, may be stated as follows: A neutral stimulus (the sound of a bell) that occurs in close *proximity* to an inherently noxious stimulus (e.g., electric shock) becomes *associated* with the noxious stimulus so that, even in the absence of the noxious stimulus, it acquires the power to *elicit response* in the organism as *though it were the noxious stimulus*.

For example:

1. Electric shock (inherently noxious, and thus called the *unconditioned stimulus*, or US, meaning that no "conditioning process" is necessary to produce the response in question) → fear response (called the *unconditioned response*, or UR, e.g., increase in pulse rate or dilated pupils).
2. Light (usually "neutral" to animals) → no fear response.
3. Light plus electric shock → fear response. In this *pairing* of light and shock, the neutral stimulus is called the *conditioned stimulus*, or CS. If this pairing within a set time interval occurs repeatedly, then . . .
4. Light (CS) alone → fear response. The fear response occurring in response to the CS is called the *conditioned response*, or CR.

Conditioning, of course, occurs to various stimuli, including both pleasant and aversive stimuli. For example, Pavlov's famous experiments involved training dogs to salivate at the sound of a bell using the aforedescribed paradigm.

Ordinarily, conditioned responses will disappear (*extinguish*) if the conditioned stimulus is repetitively experienced without any further pairing with the unconditioned stimulus (reinforcement).* This phenomenon is the basis for a form of behavioral therapy (desensitization) that is particularly useful in treating phobias, where an ordinarily neutral stimulus (e.g., elevator) has acquired the capacity to elicit the anxiety response. In phobias, the patient will go to any length to avoid the phobic object or situation in order to avoid experiencing the anxiety reaction. A patient with a phobia for crowds [and open spaces (agoraphobia)] may ultimately become a virtual recluse (vignette 3). This il-

---

* If the unconditioned stimulus has been strong enough, the conditioned response may resist extinction permanently (Solomon and Wynne, 1954; Wynne and Solomon, 1955). Sailors who had served during World War II, when a bell was used to signal air attacks ("battle stations"), still showed an increased galvanic skin response to the same gong tone 20 years later (Edwards and Acker, 1962).

lustrates an aspect of anxiety with highly important clinical implications, namely, that human beings have little tolerance for free anxiety and that individuals develop behaviors (coping mechanisms, defense mechanisms) to avert or avoid it. In classic learning theory, this is conceptualized as follows: *Anxiety is so unpleasant that a person will repeat behaviors or seek situations that have been associated with its diminution*. Behaviors that lessen anxiety can be learned through instrumental conditioning; that is, relief from anxiety can be regarded as motivation for behavior (see Chapter 19).

Psychoanalytic Signal Theory of Anxiety

As we will see below, psychoanalytic theory assigns central clinical importance to this noxious aversive quality of anxiety and labels the psychological mechanisms that are developed for averting anxiety "ego defenses" (*see* Chapter 5).

In making a distinction between fear, in which the danger stimulus is external and recognized (e.g., an escaped grizzly bear), and anxiety, in which the danger stimulus is internal and unrecognized (e.g., an unconscious conflicted impulse such as a murderous wish), psychoanalytic theory turns our attention to a careful and close consideration of "intrapsychic" phenomena. This signal theory of anxiety* starts with the idea that every human being is endowed with an inborn capacity for experiencing the combined physiological–psychological reaction that we call anxiety. Freud's theory of anxiety, however, concentrates on the importance and role of anxiety in mental life rather than on providing a fundamental explanation of its nature and basic origin (Brenner, 1955). It assigns to anxiety the central role in the neuroses by asserting that it occurs when there is *conflict* between unconscious wishes for pleasurable gratification and the person's opposing mature goals and moral standards.

The theory further specifies that the form of anxiety with which we are most familiar, that is, the response associated with subjective feelings and motor and conscious sensory components, is but one of two forms of anxiety—a clinical form (*free anxiety*). Free anxiety occurs *when psychological defense mechanisms have failed*. It may vary in intensity from relatively mild apprehension to intense disorganizing panic. The *second* form, which Freud called *signal anxiety*, is conceptualized to be so mild

---

* Freud replaced, with this theory, the older "toxic" theory, which is now of historical interest only.

and attenuated as to go unnoticed (or perhaps barely noticed) in consciousness. Nonetheless, it is perceived in the mind and reacted to as a signal of an *impending* danger situation.

The theory specifies a stepwise series of events: (1) when an unconscious wish (that would lead to unacceptable thoughts or behavior or both) is about to attain conscious recognition, an attenuated form of anxiety is generated; (2) this attenuated anxiety reaction then serves as a signal (signal anxiety) indicating that a dangerous situation will develop if the conflictual impulse becomes conscious and gains access to the motor systems that could carry it out in action; (3) in response to the signal anxiety, psychological defense mechanisms come into motion that prevent the dangerous situation from developing by barring access of the threatening impulse to consciousness and the motor systems of the body (see Chapter 5). In other words, the person reacts to the impending emergence into consciousness of a conflictual impulse as to an impending danger and so experiences attenuated anxiety. In response to this attenuated (signal) anxiety, psychological defense mechanisms are mobilized to deal with the offending impulse. In summary, the *attenuated anxiety signals a state of tension within the personality system*—that is, conflict between basic primitive organismic demands (*id*) and demands of the individual's social environment and conscience (*superego*). It is the function of a part of the personality system called the *ego* to appraise and mediate between these opposing forces and to defend against the forbidden impulses (see Chapter 5).

Success of the personality system, more specifically, the ego, is reflected in satisfactory adaptation without experience of noticeable anxiety; partial success results in experience of somewhat attenuated, but felt, anxiety (clinical anxiety states) or of adaptive states marred by neurotic symptoms or behavior or both. Failure is regarded as resulting in panic states (unattenuated anxiety) and perhaps psychotic experience and behavior.

The psychoanalytic theory of anxiety as summarized has been incorporated into clinical psychiatric theory and is regarded as basic and essential for understanding clinical disorders involving the personality system.

Anxiety is also considered to function as a stimulus to growth and to development of adaptive behaviors. This aspect will be discussed in a later section of this chapter. More detailed aspects of the theory dealing with the ontogenetic developmental aspects and metapsychology of anxiety are more controversial but perhaps of less general importance to those working outside of psychiatry.*

---

* Recommended readings by Freud, Brenner, and Reiser at the end of this chapter should be consulted for acquaintance with these more specialized features.

While the learning-theory model and the psychoanalytic theory of anxiety are quite different with respect to formulations concerning the nature and origin of anxiety and of danger situations, there are many aspects of both theories that are congruous, even though they may differ in emphasis. For example, both theories recognize that anxiety is associated with potential danger situations. The emphasis in learning theory is on external danger situations and how neutral stimuli might have become deliberately or accidentally associated with them; psychoanalytic theory emphasizes intrapsychic danger situations arising from psychological conflicts.

Psychological defense mechanisms are mobilized to reduce the unpleasant affect of anxiety in potentially dangerous situations (including being ill and being in the hospital) that threaten the individual's personality system.

Physiology of Anxiety

As already noted, when a stressful event is perceived, the cerebral cortex and its efferent pathways are activated, including the reticular activating system. This increases the arousal level and the motor outflow through the pyramidal and extrapyramidal systems, thereby increasing general muscle tension and causing specific changes in the tone of the facial muscles, giving rise to the tense expression seen in anxiety states. Tension of vocal cords may sometimes seriously interfere with speech. In general, these changes are demonstrable by the use of the electroencephalogram (EEG) and the electromyogram (EMG), the EEG showing fast, low-voltage waves typical of arousal and the EMG showing increased electrical activity of muscles involved in the response.

At the same time, activation of the limbic system (see Figure 4) results in activation of the *hypothalamus* and *autonomic nervous system* (predominantly, but not exclusively, the sympathetic division) and of the hypothalamic nuclei that secrete the *releasing factors* (hypophysiotropic hormones). The hypothalamic releasing (or inhibiting) factors influence the *pituitary gland* and lead to the stimulation or inhibition of the release of various tropic hormones. Autonomic nervous system arousal leads to an increase in circulating *epinephrine* and *norepinephrine* by sympathetic stimulation of the adrenal medulla.

Direct effects of the autonomic arousal in acute anxiety include (1) *circulatory changes*—increase in systolic blood pressure and pulse pressure (diastolic pressure and peripheral resistance may remain the same or fall); increase in stroke volume, heart rate, and cardiac output; increased blood flow to skeletal muscle, heart, lungs, and brain and de-

creased blood flow to the splanchnic vascular bed and skin; (2) exocrine *changes in skin*—increased sweating and skin conductance (cold, clammy hands); (3) dilation of the pupils; (4) changes in rate and depth of respiration; (5) changes in gastrointestinal function—secretion, motility, and mucosal vascularity; and (6) some metabolic changes such as increase in levels of sugar, free fatty acids, and lactic acid in the blood. Imbalance of sympathetic and parasympathetic components may contribute to untoward reactions such as development of disturbances in cardiac rate and rhythm and even fainting, as will be discussed in a later section of this chapter.

Indirect effects of anxiety (via effects of released hypothalamic factors on pituitary tropic function) include increase in the secretion of adrenal cortical hormones, which gives rise to profound metabolic changes (in water and electrolyte balance, in suppression of immune mechanisms, and in catabolic carbohydrate and protein metabolism), and other widespread hormonal changes, for example, in secretion of growth hormone, prolactin, and thyrotropic, gonadotropic, and antidiuretic hormones.

Immune mechanisms are profoundly altered in stressful situations causing anxiety (Locke, 1982; Stein *et al.*, 1976). Lymphoblast transformation (a measure of lymphocyte responsivity to mitogens) was found to be depressed eight weeks following bereavement of spouse (Bartrop *et al.*, 1977). Interestingly, coping ability as measured by an "ego strength scale" was positively correlated with antibody response to influenza vaccine in normal subjects (Roessler *et al.*, 1979).

Lymphoblast transformation was studied in a group of psychiatric residents in Canada preparing for a major qualifying examination, a particularly stressful event (Dorian *et al.*, 1981). The lymphoblast transformation was significantly reduced in the candidates as opposed to an age- and sex-matched control group of physicians. By two weeks after the exam, however, the lymphoblast transformation was higher among the candidates compared to the controls. The level of immune suppression pre-exam was greater in the subjects who expressed high subjective distress as compared to those who expressed low subjective distress. Interestingly, the immunosuppression in the candidates did not seem to be mediated by plasma corticosteroid levels, which were *lower* in the candidates than in the controls.

What might be the mediating mechanisms among stress, anxiety, and immune changes? Certainly hormonal changes, especially corticosteroids, are known to influence immune mechanisms. In addition, the autonomic nervous system may exert influence on the immune system through the adrenergic pathways. In general, substances that increase intracellular cyclic AMP levels, such as histamine and isoproterenol, as

well as β-adrenergic agonists are immunosuppressive, while substances that increase intracellular cyclic GMP levels, such as phenylephrine, acetylcholine, and insulin, as well as α-adrenergic agonists are immunoenhancing (Locke, 1982). There is evidence that the organs that subserve the immune mechanisms, such as the thymus gland and the spleen, are directly controlled by the CNS through neural influences (Bullock and Moore, 1980; Williams *et al.*, 1981). Recently, receptors for β-endorphin and met-enkephalin have been discovered on lymphocytes (Wybran *et al.*, 1979; Hazum *et al.*, 1979). Thus, the immume mechanism seems to be controlled both directly and indirectly by the CNS through direct neural, autonomic, endorphinergic, and endocrine systems.

In general, the longer-lasting endocrine and immune systems are called into play when response to danger is intense and sustained. Then, more profound metabolic effects are added to the more acute autonomically innervated reactions. Selye's now classic studies of stress call attention to the role of the pituitary–adrenal cortical system and the adrenal cortical hormones in the *adaptation syndrome* and point to their importance as probable contributors to pathogenesis of a variety of clinical disorders of (unknown) multiple-factor etiology such as rheumatoid arthritis and essential hypertension (Selye, 1950).

As the preceding discussion shows, consideration of the psychophysiology of anxiety leads quite naturally to a more general look at the psychophysiology of stress and to contemplation of some clinical implications. For the student of medicine, there are two highly important points to emphasize here. The first concerns the distinction between stress and strain. *Stress* consists of an *external* and/or internal *challenge* to the integrity of a structure requiring adaptation or adjustment, whereas *strain* is a measure of the tension or imbalance endured *within* the responding (stressed) structure. A physical analogy would be that of a heavy truck crossing a bridge, the weight of the truck constituting the stress and the disturbance in molecular alignment of the bridge structure constituting the strain. In psychophysiological systems, the stress might be a psychosocial crisis (e.g., death of a close relative); strain would be measured by the degree to which balance of psychophysiological systems is upset and required to adjust in order to restore a previously steady state.

The second point to emphasize is that there is in the human an *inverse relationship between the effectiveness of psychological defenses and the degree of physiological activation* (reviewed by Mason, 1975). When defenses break down or are ineffective, physiological changes are intense; when defenses are highly effective, physiological changes, if any, are minimal. Thus, the effectiveness of psychological defenses is a crucial

variable in determining the degree of strain in the individual. For further discussion of defense mechanisms, see Chapter 5.

Psychosocial stress, then, carries the potential for *inducing* profound and widespread physiological, metabolic, and chemical effects in virtually all systems, organs, and tissues of the body—effects that may influence the balance between health and disease—but the extent and intensity (potential seriousness) of such effects depend on the efficiency of psychological defenses. The physiological, metabolic, and chemical effects referred to above probably influence the health–disease balance in a nonspecific fashion, that is, by affecting the resistance of receptivity of tissues to pathogenic vectors of any type (e.g., bacteria, viruses, metastatic neoplastic cells, and allergens). These are the mechanisms and relationships that account for the profound effects of life change on morbidity and mortality as well as on illness and help-seeking behavior (see Chapters 1 and 13).

Brain Mechanisms of Anxiety

We mentioned earlier that the modern concept of emotion is based on the neuroanatomical model proposed by Papez and MacLean. According to this model, the structures of the limbic system, the inner core of the brain, play a major role in the brain mechanisms of emotions, including anxiety. The word *limbic* refers to a border or a hem. This term was coined by Broca in 1878 to denote the inner brain tissue surrounding the brain stem and lying under the neocortical mantle (Figure 4). The microscopic structures of the limbic brain are presumed to be organized into two layers. The phylogenetically oldest tissue (allocortex) makes up the inner ring, and the outer ring (called the transitional cortex) consists of a peculiar cellular structure, not resembling either the neocortex of the allocortex (Isaacson, 1974). Some portions of the inner aspects of the neocortex and thalamus, although not part of the original "limbic lobe" described by Broca, are often considered as being part of the functional unit, the limbic system. Thus, the structures involved with this system include the *hypothalamus*, and *amygdala*, the *hippocampus*, the *septum*, and the *cingulate gyrus*. These structures are closely related to the *anterior thalamus* and the *reticular activating system*, which runs through the limbic system and the brain stem and extends into the spinal cord (see Figures 2–7).

Various studies, including direct electrical stimulation and surgical ablation of structures of the limbic system, indicate that basic emotions
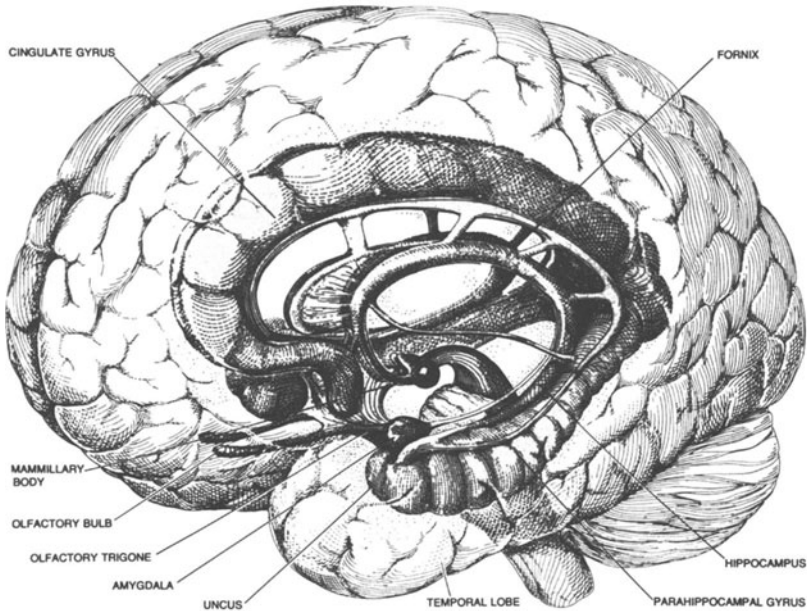
Figure 4. Limbic system (shaded area): a series of evolutionarily primitive regions at the core of the brain that are primarily involved with smelling in lower vertebrates and with the arousal of emotions in humans. (From Synder, 1977. Copyright 1977 by Scientific American, Inc. Reproduced with permission.)

and drives manifested by eating, sexual behavior, drinking, "sham rage," and attacks are controlled by these structures.

In general, pleasurable feelings are produced by stimulation of certain areas of the limbic system and related structures, such as the lateral hypothalamus and the medial forebrain bundle, a neuron system arising from the noradrenergic, serotonergic, and dopaminergic neurons of the brain stem and distributing widely through the limbic system and to the forebrain (see Figures 3 and 6). Lesions of the medial forebrain bundle produce a drop of 90% or more of the norepinephrine levels in the forebrain, which may be of significance in disorders of mood, such as manic or depressive disorders (see Chapter 6). Stimulation of other areas of the limbic brain, such as the medial hypothalamus or the medial portion of the amygdala, the almond-shaped structure lying at the top of the hippocampus, produces aggressive and angry responses.

If an internal or external stimulus is processed by the neocortex and the stimulus is found to be associated with unpleasant or painful memories, then the signal of anxiety would be generated. That is, the neo-
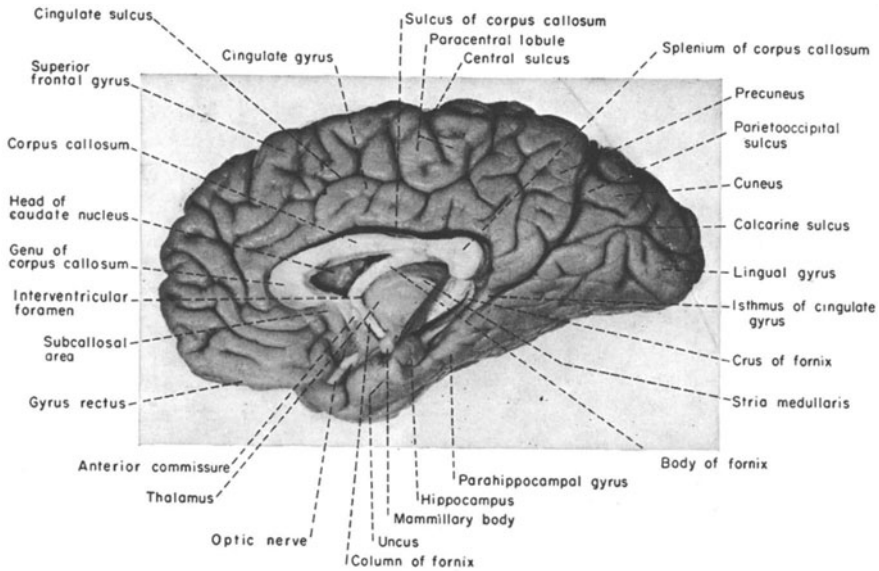
Figure 5. Photograph of the medial surface of the adult brain cut in sagittal section and partially dissected to show the hippocampus and fornix. (From Everett *et al.*, 1971. Copyright 1971 by Lea & Febiger. Reproduced with permission from the publisher and the author's estate.)

cortex would play a major role in the processing and integration of perception and the activation of associative pathways. The result of such activation may be stimulation of certain parts of the limbic system concerned with the feeling of anxiety. In fact, there are extensive connecting pathways between many parts of the neocortex, especially between and among the prefrontal cortex concerned with intention and plans, the temporal cortex concerned with verbal memory, and the amygdala and hippocampus of the limbic system. The limbic system and the neocortex have extensive connections with the brain stem. Nuclei in the brain stem, especially the noradrenergic locus ceruleus (see Figure 6), may have important functions in anxiety–fear mechanisms, as we will discuss in the next section.

The amygdala, hippocampus, and septum all have modifying influences on the functions of the *hypothalamus*, which is the *final common pathway* of limbic system functions. Different areas of the *amygdala* are concerned with searching, curiosity, and aggressive reactions. Complete ablation of both amygdalae in animals tends to produce placidity and lack of fear. This is a part of the *Klüver–Bucy syndrome*, produced by
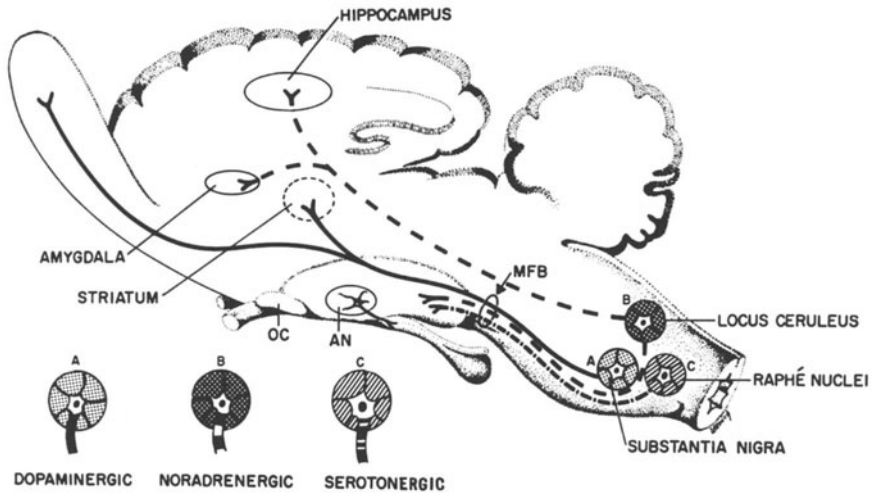
Figure 6. Monoaminergic pathways in mammalian brain. The principal localization of the neurons containing norepinephrine, dopamine, and serotonin is in the mesencephalon and pons. Axons of these cells are distributed to widespread area of the cortex, limbic system, and striatum. The dopaminergic system of the arcuate is an exception to this general scheme of distribution. Abbreviations: MFB, medial forebrain bundle; AN, arcuate nucleus; OC, optic chiasm. (From Martin *et al.*, 1977. Copyright 1977 by F.A. Davis Co. Reproduced with permission from the publisher and authors.)

bitemporal lobectomies. Other features of the Klüver–Bucy syndrome include visual agnosia, a compulsiveness to contact and examine objects, a strong oral tendency, and hypersexuality.

The *hippocampus* is connected to the septum via the fornix, which terminates in the mammillary body of the hypothalamus (Figure 3–5). Destruction of the hippocampus in animals usually results in a greater willingness to undertake new actions and in decreased fear reactions. The hippocampus, together with the septum, forms an inhibitory system, the excitation of which causes behavioral inhibition. The hippocampus is also intimately involved in the conversion of *recent memory* into long-term memory and in the association of stimulus with painful experience in avoidance conditioning (Isaacson, 1974; Gray, 1972). Parenthetically, *psychomotor epilepsy* (also known as temporal-lobe epilepsy) is often associated with extensive scarring of the hippocampus and neighboring structures and with many behavioral and emotional problems.

The *amygdala* and the *septohippocampal system* are considered by many to act as a push–pull balanced control system with regard to anx-
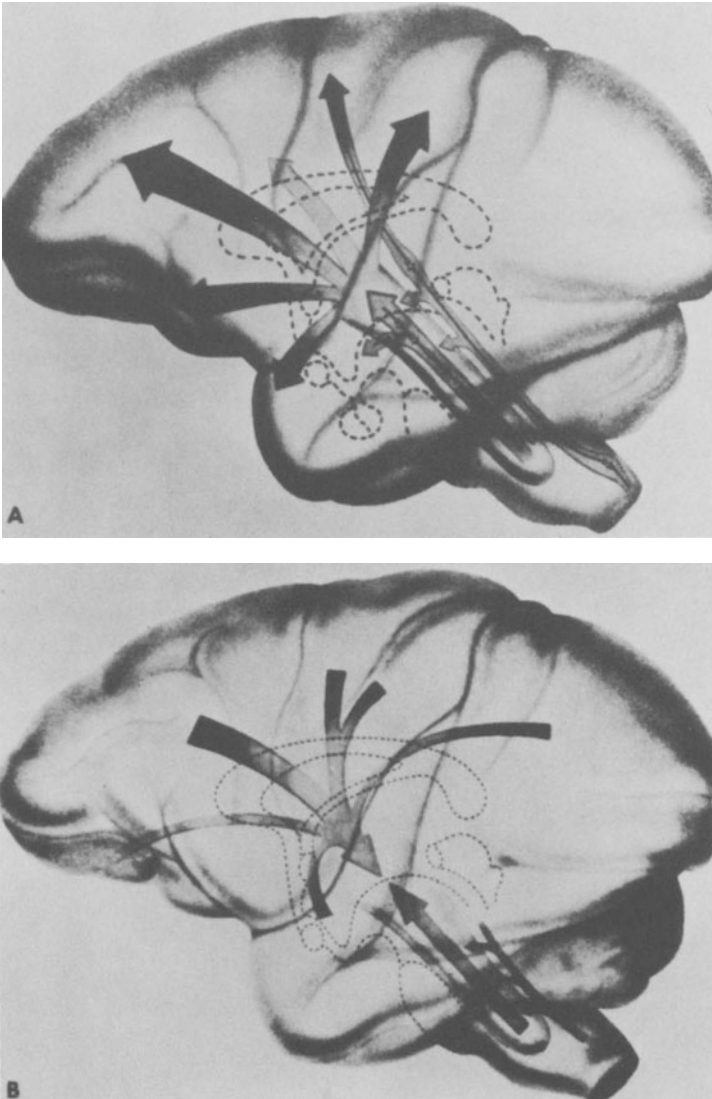
Figure 7. Reticular activating system. A: Ascending reticular activating system schematically projected on a monkey brain. (Originally published in Lindsley: *Reticular Formation of the Brain*. Boston, Little, Brown & Co.) B: Convergence of pathways from the cerebral cortex and from the spinal afferent systems on the reticular activating system. (Originally published in French, Hernandez-Peon, and Livingston: *J. Neurophysiol.* **18:**74, 1955.) (From Guyton, 1976. Copyright 1976 by W.B. Saunders Co. Reproduced with permission from the publisher and author.)

iety. The hippocampus is thought to be primarily responsible for recognition of a "mismatch" between the incoming stimulus and an expected stimulus. Together with the amygdala, the septohippocampal system determines the degree of *uncertainty* in a given situation and determines the overall reaction of the organism via the final common pathway of the hypothalamus.

The *hypothalamus* is the final comon pathway and effector organ of the limbic system. The hypothalamus may be divided loosely into an *anterior, parasympathetic* part and a *posterior, sympathetic* part. The anterior hypothalamus is considered to be "trophotropic," that is, related to energy conservation and pleasurable states. The posterior hypothalamus is "ergotropic," having to do with the interaction between the organism and the environment. Stimulation of the posterior hypothalamus results in sympathetic nervous system activation and anxiety–fear responses as well as anger. Stimulation of the anterior hypothalamus can result in either relaxation or acute distress.

The hypothalamus is sometimes called the *"homeostat"* of the body, since it controls most of the homeostatic mechanism through its outflow into the autonomic nervous system and the endocrine system via the pituitary gland. When the outcome of the excitation of the neocortex and the limbic system structures of the amygdaloseptohippocampal system is excitation of the posterior, ergotropic part of the hypothalamus, a *fight–flight* reaction may be elicited with concomitant emotions of anxiety, fear, and anger. If the outcome is stimulation of the anterior, trophotropic part of the hypothalamus, a *relaxation* response might be elicited. Various gradations of simultaneous stimulation also occur, thus eliciting mixtures of ergotropic and trophotropic responses. Also, in situations of acute fear and distress, the stimulation of the trophotropic, parasympathetic system may be considerable, and sometimes predominant (such as decreased blood pressure, decreased heart rate, and immobilization with depressive or apathetic affect).

In addition to the brain structures described above, the *reticular activating system* plays an important role in anxiety mechanisms (see Figure 7). The diffuse network of nerve cells extending from the neocortex through the limbic system into the spinal cord (called the reticular formation) has both ascending and descending pathways. The major function of this system is the control of the *tone* of the central nervous system (*arousal*). The perception of a potentially dangerous situation results in *activation of the reticular activating system*, thus preparing the brain and the central nervous system for the fight–flight response, and contributes to the emotional experiences of anxiety, fear, and anger.

With the anxiety–fear response, the hypothalamus facilitates the release of certain *"stress" hormones* through its control of the pituitary gland by means of the *hypothalamic releasing and inhibiting factors* (hypophysiotropic hormones).

Once the anxiety–fear response has been elicited by specific activation of the hypothalamus and the pituitary gland, the effect on the brain of the sensory input from the bodily changes due to sympathetic activation, such as increased heart rate and blood pressure, and the direct effect on the brain of the hormones released by the pituitary may form a *secondary component* of the experience of anxiety and fear. This component (the effect on the brain of the bodily changes accompanying stimulus) of emotion had been equated with emotion itself in the *James–Lange theory of emotions*. Although Cannon disproved the equation, further studies have shown that some bodily changes accompanying emotion, such as levels of epinephrine in the blood, can influence the *readiness of individuals* to experience specific emotions, although this reactivity is modified a great deal by cognitive factors, such as what the subject had been told prior to an intravenous infusion of epinephrine (Schachter and Singer, 1962). For example, intravenous infusion of norepinephrine and epinephrine simulates the bodily changes observable in anxiety–fear or rage reactions, and the subject will experience these emotions at the slightest provocation. However, if the subject has had explained exactly what bodily changes to expect, he will not be as ready to react emotionally.

Studies on the direct effects of "stress hormones" on the brain indicate that adrenocorticotropic hormone (ACTH) increases the anxiety–fear response in animals. For example, ACTH injection increased the latency period for animals to enter a cage where they had been shocked previously. Adrenocortical steroids such as cortisol seem to improve avoidance learning in animals, suggesting increased anxiety–fear response. On the other hand, in humans, exogenous corticosteroids tend to cause euphoria, although anxiety symptoms also occur in some subjects. Some studies indicate that ACTH enhances, while cortisol tends to inhibit, the dorsal area of the hippocampus.

Central Neurotransmitters in Anxiety

At present, data concerning the roles of various neurotransmitter substances in the brain during anxiety states are confusing and often contradictory. Almost all known CNS neurotransmitters seem to be involved in the functioning of the various structures involved. The sub-

stances that may be implicated in the anxiety mechanisms include catecholamines (including norepinephrine, epinephrine, and dopamine), the indoleamine serotonin, acetylcholine and γ-aminobutyric acid (GABA), glycine, substance P, and endorphins (enkephalins). The noradrenergic system is involved in the pleasure–reward system as well as in the anxiety–fear system; the serotonergic pathways, in sedation and sleep mechanisms; the cholinergic pathways, in arousal determined by the reticular activating system as well as in the functions of the neocortex and movement. GABA seems to exert a generalized inhibitory effect within the CNS as well as to play a major role in anxiety regulation.

GABA is perhaps the most prevalent neurotransmitter in the brain and seems to exert its inhibitory function by opening chloride channels that are directly linked to the GABA receptors. This increases the influx of chloride ions into the cell, hyperpolarizing it. The antianxiety drugs in the class of *benzodiazepines* (which include diazepam and chlordiazepoxide, the most commonly used antianxiety drugs) bind to specific receptor sites in the brain, the benzodiazepine receptors (Squires and Braestrup, 1977). Recent evidence suggests that the benzodiazepine receptors are functionally linked to the GABA receptors to form a GABA–benzodiazepine–chloride ionophore complex (Paul *et al.*, 1981). Although most benzodiazepines have both sedative and anxiolytic action, it seems that these effects are not necessarily related. For example, benzodiazepines have been discovered that possess anxiolytic action with minimal sedative action. They seem to bind to a specific subclass of benzodiazepine receptors. The imidazodiazepam Ro 15-1788 is a benzodiazepine-receptor antagonist. Ro 15-1788 blocks the anticonvulsant, depressant, and ''antipunishment'' effects of benzodiazepines in animals, but does not possess anxiogenic effects in animals or humans (Hunkeler *et al.*, 1981). The ethyl ester of β-carboline-3-carboxylic acid (β-CCE) is another substance that has a high affinity for benzodiazepine receptors in the brain. In rhesus monkeys, β-CCE infusion produced marked signs of anxiety, including increase in heart rate, blood pressure, and behavioral characteristics, which were reversed by Ro 15-1788 and by diazepam (Ninan *et al.*, 1982). Thus, the GABA–benzodiazepine–chloride ionophore complex seems to be an important mediator in the regulation of anxiety experience.

While the benzodiazepines are effective in most generalized anxiety states, and in anticipatory anxiety to a stressful event, they are ineffective in episodic attacks of panic. It appears that for panic attacks, the norepinephrine system may be more intimately involved.

Stimulation of the *locus ceruleus* in the brain stem (pons) in monkeys results in typical anxiety–fear behaviors, and most antianxiety agents

appear to inhibit the locus ceruleus or its synaptic projections (Redmond *et al.*, 1977; Redmond and Huang, 1979). The locus ceruleus consists of noradrenergic cell bodies that provide more than 70% of the noradrenergic input into the brain. The cells in the locus ceruleus have receptors for GABA, benzodiazepines, and opiates, as well as for norepinephrine. Clonidine, an α_2-receptor agonist, reduces the activity of the locus ceruleus, and is effective in reversing the anxiety–fear behaviors induced by locus ceruleus stimulation. In humans, clonidine is effective in decreasing episodic anxiety attacks, but less effective than other antianxiety agents in reducing generalized nonepisodic anxiety symptoms (Hoehn-Saric, 1982). Antidepressant drugs [tricyclics and monoamine oxidase inhibitors (see Chapter 20)] generally decrease the activity of the locus ceruleus, probably by increasing norepinephrine levels at the autoreceptor sites. They are most effective in reducing panic attacks, but not in generalized or anticipatory anxiety. Thus, it appears that the norepinephrine system is probably involved in some types of anxiety, particularly panic attacks.

The serotonergic system may also be involved in anxiety responses and stress. The antidepressant drugs affect not only the noradrenergic system but also the serotonergic system in the brain. In fact, the long-term effects of tricyclic antidepressant treatment seems to be an enhancement of the serotonergic and α-noradrenergic transmission with a down-regulation of the β-adrenergic transmission in the brain (Aghajanian, 1981). Tricyclic antidepressants with major serotonin-reuptake-blockage effects (clomipramine, zimeldine) have been reported to be effective in panic attacks and phobic anxiety (Hoehn-Saric, 1982).

The endorphine system may also be involved in anxiety and stress regulation. The limbic system and the locus ceruleus have numerous opiate receptors, and stress-induced analgesia is mediated by the endorphinergic system (Emrich and Millan, 1982). Naloxone administration, however, does not cause severe signs of anxiety or autonomic arousal in normal volunteers or in chronically anxious persons, although it does cause mild dysphoria (Hoehn-Saric, 1982).

Function of Anxiety

Anxiety is an unpleasant emotion and certainly a major cause of suffering. One might wonder, then, why we have anxiety at all; that is, why have we not discarded anxiety eons ego in the evolutionary process? The obvious reason is that anxiety serves highly important *adaptive functions*. We have already seen that anxiety prepares the organism phys-

iologically for fight–flight reactions essential for survival, and we have discussed the role of anxiety in learning; that is, it *facilitates avoidance learning*, thus preventing repeated exposure of the organism to dangerous situations. Another highly important function of anxiety that we have discussed is its signal function and role in initiating the *mobilization of psychological defense mechanisms*.

But beyond these more immediate and obvious survival values, anxiety serves broader and more far-reaching functions—it is an important force in stimulating and supporting *personality development*, particularly maturational processes responsible for acquisition and effective performance of *socially valued skills*. Perhaps the easiest way to open discussion of the productive, constructive, or generative function of anxiety is to look at its immediate or short-term role as an influence on skilled performance. Anyone who has had to face the challenge of, say, an important academic examination, or athletic competition, or public speaking knows very well that there is an optimal level of "tension" or anxiety for realizing the best of one's capabilities. With too little anxiety (and insufficient motivation), performance is likely to be lackluster and below par. We all have our own ways of getting "psyched up" for such challenges. On the other hand, if the level of anxiety becomes too high, performance suffers because of the disorganizing effects on finely tuned cognitive and sensorimotor mechanisms—increased incidence of forgetting and of errors, loss of fine motor coordination, loss of confidence, feeling "clutched up," panic, and so on. In vignette 2, it seems likely that excessive anxiety in relation to success in sex may have played a disruptive role in the patient's experience of sexual activities and contributed to her eventual aversive response. The *inverted U relationship between level of drive and performance* (Yerkes–Dodson law) in this way can be seen to apply to anxiety, again calling attention to its "drive" or motivational quality.

Many theories of personality development postulate a similar motivational, generative, and facilitating–inhibiting function for anxiety in cognitive and social development. Psychoanalytic theory (particularly developmental ego psychology) details how frustration generates anxiety and motivates development of the requisite cognitive and defensive ego functions and related social skills on which mature adult adaptive behavior depends (Freud, 1926/1953; Hartmann, 1958, 1964).

Dysregulation of Anxiety

As is the case with other primarily adaptive mechanisms (e.g., immune responses, fever, and inflammation), anxiety is subject to dys-

regulation. Major disruptions in its regulation not infrequently contribute to clinical morbidity—and sometimes even to fatal reactions (Schwartz, 1977). Walter Cannon, fascinated by the phenomenon of voodoo death, reviewed the world literature on it and wrote a now classic paper on the subject (Cannon, 1942). In Africa, some superstitious primitive tribes believed that certain powerful medicine men or witch doctors had the power to kill through the magical ritual of "bone pointing." There were numerous accounts describing how vigorous, healthy individuals, after having had the bone pointed at them and being convinced they were going to die, would leave the tribe and within a few days be dead—of no apparent or usual natural cause.

Cannon speculated that the mechanism might be excessive sympathetic adrenal stimulation. Richter studied an experimental model of sudden functional death in captured wild Norway rats and demonstrated that (1) the pathogenic mechanism was vagal inhibition of the heart and (2) the phenomenon was influenced by sensory and psychological factors. There are, in fact, quite a number of examples of behaviorally induced or associated sudden death or feigned death in animals (Richter, 1957). Sudden death due to fatal arrhythmia is not an unusual occurrence in humans (400,000 deaths each year are attributed to it in the United States) (DeSilva and Lown, 1976). Most studies of this phenomenon implicate the effects of abrupt autonomic imbalance on the cardiac rate-setting and impulse-conducting tissues (Lown et al., 1977). This effect is probably most likely to occur in persons with structural damage to these tissues (e.g., due to arteriosclerosis), but it is not really known whether or not the phenomenon can occur in persons with entirely healthy hearts.

Two other common and clinically important examples of anxiety dysregulation are fainting (syncope) and hyperventilation.

Fainting

In the common form of fainting (*vasodepressor syncope*), the mechanism involves primarily the cardiovascular aspects of the anxiety reaction, specifically as it affects distribution of blood throughout the body. The loss of *consciousness* is caused by inadequate blood supply to the brain even though the anxiety response normally increases cardiac output and redistributes blood so as to increase blood flow to the brain as well as the skeletal muscle, heart, and lungs. In vasodepressor syncope, blood pools in the periphery, particularly in the extensive vasculature of the skeletal muscle of the lower extremities. This results in decreased

venous return to the right side of the heart, decreased cardiac filling, and fall in cardiac output—sufficient to result in inadequate blood supply to the brain. Engel (1962) postulates that the circulatory changes of anxiety prepare for fight or flight, both of which require vigorous muscle activity, which in turn keeps the increased blood supply to muscle circulating and literally massages it out of the muscle bed back toward the heart, ensuring adequate cardiac filling and output. If, however, the danger situation is abrupt, overwhelming, and one that the person is powerless to influence, and if the person would be ashamed to flee (retreat) and does not act, that is, does not move, the large amount of blood sent to the muscles stays there instead of being pumped back to the heart by muscle contraction. Epidemics of vasodepressor syncope commonly occur among army recruits when they are lined up for blood tests by venipuncture. If one recruit faints, it is common for several more to follow suit in short order, illustrating another interesting aspect, namely, social contagion. The issue of pride (shame) preventing retreat in this situation is clear. Another aggravating circumstance is that the subjects are standing up, so that gravity aggravates or accentuates the pooling of blood in the legs.*

Hyperventilation Syndrome

The second clinically important acute dysregulation syndrome is the "hyperventilation syndrome." In this condition, an exaggeration of the respiratory aspect of the anxiety response leads to difficulty, that is, excessive increase in rate or depth, or both, of breathing. This is easily observed by the physician, although the patient most often is not aware of it. With the overbreathing, there is excessive loss of carbon dioxide, leading to *respiratory alkalosis* (reduced pCO_2). The increased blood pH leads to decreased ionization of calcium, which may produce clinical signs of tetany (in which painful muscle contractions occur). In addition to the usual symptom of anxiety, the hyperventilating patient experiences light-headedness (altered blood gases), headache, nausea, tingling around the mouth, tingling of the fingers and toes (hypocalcemia), and, if the overbreathing lasts long enough, even cramping or spasm of muscles in the extremities. All this is enough to make the person even more anxious, resulting in a vicious circle. A patient who has such attacks is usually terrified by the experience. Dramatic reassurance can be given

* For a thorough description and discussion of mechanisms of all forms of syncope, readers are referred to Engel (1962) and medical textbooks.

by demonstrating that the attacks can be reproduced at will by deliberate overbreathing. Then the symptoms can be reversed by having the patient breathe into a paper bag and rebreathe the exhaled air, which has a higher concentration of carbon dioxide. Confirming the diagnosis through this technique can be dramatic and highly gratifying to both patient and physician. Most doctors remember with great pleasure the first time they had the opportunity to use this treatment for hyperventilation. Further evaluation should then be done to understand the underlying cause of anxiety in the patient that leads to hyperventilation.

Stress-Related Disorders

As noted in the section on the physiology of anxiety, when stress (and strain) is prolonged, anxiety responses merge into states in which additional systems, for example, neuroendocrine systems, are mobilized, and extensive metabolic chemical changes eventuate in the central nervous system and throughout the entire body. These changes, in interaction with other factors (e.g., constitutional, specific tissue vulnerabilities, specific pathogens), may contribute to or aggravate many clinical disorders such as hypertension and peptic duodenal ulcer. Although there is growing experimental literature on stress-related disorders, the mechanism of interaction between stress and disease is far from completely understood. We are only beginning to appreciate the multiple interacting and intervening variables that can influence the response of somatic systems to psychological–social stress. For example, let us look at just two (somewhat contradictory) experiments in the vast experimental literature on production of gastric ulcers in animals by exposure to stress.

When monkeys were trained to press bars constantly to avoid electric shock in a complex and highly demanding operant conditioning program with many contingencies, they developed bleeding gastric ulcers and died ("executive" monkeys). On the other hand, yoked control monkeys that were shocked the same amount but did not have to press bars to avoid shock did not develop bleeding ulcers (Brady, 1958). In another experiment, rats that were trained to avoid shock by bar-pressing did not develop bleeding ulcers, while yoked control rats that were exposed to inescapable shock developed bleeding ulcers (Weiss, 1968). In the latter experiment, however, the rats trained to press bars to avoid shock had *feedback* in the form of light—that is, the light indicating impending shock was turned off as the bar was pressed—unlike the "ex-

ecutive" monkey situation, in which there was no direct feedback concerning adequate performance.

One might speculate that the *"executive"* rats had a *sense of control* with performance of bar-pressing, while the monkeys had to perform without this sense of control. In any case, it seems that a sense of control and feedback of success from coping strategies in anxiety states may be one important factor in buffering the physiological strain resulting from the stress and in influencing whether or not a "stress disorder" will result.

Dysregulation of anxiety and stress reactions may occur due to *uncontrollable* and *sustained external or internal anxiety-provoking situations* (e.g., as in battle fatigue or unresolved conflict), or it may occur due to *inherent instability, defect, or malfunction in any of the many structures and systems involved, including the target organs of physiological arousal.*

A somewhat unique side effect of modern medicine is the increase in *iatrogenic dysregulations* of anxiety. This often results when physicians, overlooking the psychosocial origins of the reactions or, in some cases, the adaptive significance of mild to moderate levels of anxiety, prescribe *antianxiety medications at times* and *in dosages* that are *not indicated*. Since antianxiety medications, especially minor tranquilizers such as the benzodiazepines, are habit-forming, a person would have to take increasing doses to maintain anxiety at an imperceptible level. This will be especially marked if the psychological and/or social causes of the anxiety are ignored and allowed to continue operating unabated. As a further complication, the patient will eventually experience exacerbation of the anxiety, due to the effects of withdrawal, when the dose of the medication is lowered. This is *not* to say that antianxiety medications should not be used—there are many situations in which they are indicated. The physician should have, however, a clear idea as to why, and for how long, the medication is to be prescribed.

Classification of Anxiety Dysregulation Syndromes

While it is not within the scope of this book to discuss all stress disorders, a brief discussion of the classification of anxiety dysregulation syndromes is in order. Essentially, anxiety dysregulation syndromes can be categorized according to whether they involve (1) dysfunction in the experience of anxiety (excessive or insufficient); (2) dysfunction in psychophysiological systems, or (3) dysfunction in behavior.

Disorders in the Experience of Anxiety. The *locus* of the problem in this category is often the *brain* and the *personality system.*

Excessive experience of anxiety. Anxiety disorders (neurosis), anxious personality traits, impulsiveness, tendency to become paralyzed with anxiety in the face of moderately stressful situations, and similar disorders, fall within this category. Drug abuse, including alcoholism, may be secondary to this type of anxiety dysregulation.

Panic disorder is characterized by panic attacks occurring with relatively high frequency (e.g., more than one per week). This condition is often associated with agoraphobia (fear of open spaces and crowds). Excessive activation of the locus ceruleus may underlie this disorder. Antidepressant drugs rather than antianxiety drugs are effective in this condition.

Insufficient experience of anxiety. Persons who chronically experience little or no anxiety tend to lack motivation and become underachievers. Some persons, on the other hand, tend to seek extremely dangerous situations to experience arousal (excessive risk-seeking behavior). Inability to learn from unpleasant experiences may be one factor contributing to development of antisocial personality disorders.

Psychophysiological Disorders. Stress disorders in the form of tissue damage or organ dysfunction or both may occur following *sustained physiological arousal* in anxiety. The *locus* of the problem may be in the perceptual apparatus, cerebral cortical structures, and the *personality system* (excessive generation of anxiety), in the *neuroendocrine* and *autonomic nervous systems* (instability or excessive excitation), and/or in the *target organs* (vulnerability). Psychophysiological disorders may occur in relatively healthy and disease-resistant persons with intact personality systems and central nervous and endocrine systems if the organism–environmental interaction is such that the physiological component of anxiety is activated for prolonged periods in an uncontrollable way. If disease (such as essential hypertension, peptic ulcer, migraine, the depressive syndrome, or schizophrenia) eventuates, the selection of the affected system probably rests on *constitutional* (genetic and developmental) *predisposing* factors related to the organ system involved.

Dysfunction in Behavior. Heterothetic behaviors. As we discussed in Chapter 1, some persons who find themselves in anxiety-provoking situations seek medical help for minor physical symptoms without recognizing that the help-seeking behavior is motivated by anxiety. An example is the woman who, when family problems arose, came to see her physician for varicose veins she had had for 20 years. Prompt recognition

of the presence of heterothetic behaviors is important for the physician to prevent "addiction to sick-role behavior" or unnecessary and potentially dangerous medical procedures or both (see Chapters 2 and 17).

Cognition–action dissonance. This occurs when a person takes no action to alleviate an anxiety-generating situation, even when the situation is readily identifiable and the means of avoiding danger is readily available. The *locus* of the problem in this case is obviously in the personality system and, secondarily, in the organism–environment interaction. This syndrome can eventually contribute, of course, to development of any of the disorders discussed above. "Learned helplessness" may be one possible explanation for this disorder (Seligman and Maier, 1967). Animal and human experiments show that repeated prior exposure to situations in which behavior has no effect (learning helplessness) may influence individuals' behavior in future situations. They may tend to "give up" the quest for ways of coping, even in new situations in which their behavior could have an effect. Such learned helplessness is often encountered in depression (see Chapter 6).

Evaluation of Anxiety

The diagnosis of anxiety is based on the signs, symptoms, and behavioral changes discussed in previous sections of this chapter.

Once the presence of anxiety is established, the evaluation should proceed to the contexts of its occurrence, that is, the questions of the meaning and significance of anxiety (What is the danger situation?), the kind of individual (Prone to anxiety? Tending to deny anxiety?), the cultural–social matrix (What is the method of expressing anxiety the patient is accustomed to? Complaining of physical symptoms? Taking medications?), and the reasons the patient is seeking help now (Limit of tolerance? Heterothetic? Occurrence of psychophysiological disorder?). These questions should be considered in terms of the current, recent, and background contexts of the anxiety.

Contexts of Anxiety

Current Context. Presenting symptoms. What are the immediate circumstances (and accompanying thoughts) under which the anxiety is experienced? Include considerations of patient's antianxiety medications or withdrawal therefrom. How effective are the psychological defenses?

Recent Context. What is the danger situation? Why is the situation seen to be dangerous in the light of the patient's experience? Any cumulative effect of stressful events in the recent past? Has the body been weakened by physical illness (e.g., recovering from an infection, presence of chronic disease)? Are there any physical illnesses or vulnerabilities that may tend to dysregulate anxiety at central nervous system, neuroendocrine, or target organ levels?

Background Context. Is the patient habitually prone to experience anxiety? What psychological defenses does he ordinarily use? How is anxiety handled in the patient's cultural and social class matrix (e.g., by somatization or suppression)? If a physical symptom related to anxiety is present, what is the patient's early experience concerning such symptoms either in himself or in a relative?

A fuller discussion on the systematic evaluation of patients will be presented in Chapter 11.

Differential Diagnosis of Anxiety States

Any *disease* of any part of the brain, autonomic nervous system, or neuroendocrine system associated with the anxiety mechanism may mimic anxiety states, as may some diseases of target organs. It is important to *rule out* such diseases. Physical examination and psychiatric evaluation, including mental status examination and appropriate laboratory test, will usually clarify the diagnosis. To establish the psychiatric diagnosis of anxiety disorder (neurosis), it is not sufficient to "rule out organic disease" by negative physical examination and laboratory findings. It is also necessary to adduce, by psychiatric evaluation, evidence for a positive psychiatric diagnosis, such as clarification of the psychosocial context or the danger situation. Some diseases capable of mimicking anxiety states are thyrotoxicosis, pheochromocytoma, carcinoid syndrome, hypoglycemia, seizure disorders, drug withdrawal states, brain tumors, and Cushing's syndrome. Some major psychiatric disorders such as schizophrenia and affective disorders are often accompanied by anxiety, but in addition, one would find evidence of other psychiatric difficulties such as a thought disorder in patients with schizophrenia and altered neurovegetative function and sleep disorders in patients with the depressive syndrome.

Management of Anxiety

Careful evaluation and appraisal of the patient with anxiety should lead naturally to formulation of a rational management plan. The first order of business is to determine whether or not the experienced anxiety is excessive, that is, whether it threatens to paralyze or decrease *coping* and adaptive *abilities* of the patient or to cause other dysregulation syndromes such as psychophysiological disorders. If excessive anxiety is present, then prompt reduction of such anxiety by means of appropriate reassurance and antianxiety medications is indicated. (Sometimes even hospitalization is desirable as a way of getting the patient away from a stressful life situation and providing a supportive setting.) In prescribing antianxiety agents such as diazepam (Valium) or chlordiazepoxide (Librium), the physician, being aware of the habit-forming qualities of the medications, should take care that they are used only *temporarily*, almost as an emergency measure. Major tranquilizers such as perphenazine (Trilafon) or haloperidol (Haldol) in small doses (e.g., 2 mg Trilafon b.i.d. or t.i.d. p.r.n.) may be equally effective in reducing anxiety without addictive qualities, but they can also produce serious and irreversible side effects in some patients if used excessively and over prolonged periods. For the treatment of panic disorder and agoraphobia, antidepressant drugs [tricyclics and monoamine oxidase inhibitors (see Chapter 20)] are effective. Clonidine, an α_2-auto-receptor agonist acting on the locus ceruleus, has also been reported to be effective. In some cases, in anxiety states with predominant symptoms of sympathetic arousal (e.g., palpitations), β-blockers such as propranolol are useful. (A more comprehensive discussion of these and other medications will be found in Chapter 20).

Having determined the severity of anxiety present, and having made a decision as to whether or not to *treat the manifest anxiety per se*, the physician should next attempt to define the *causes* and *contexts* of the anxiety and, having done so, to institute appropriate treatment modalities. A few examples follow.

Careful evaluation of the contexts of anxiety may reveal that the main danger situation is an *intrapsychic* one (intensification of psychological conflicts, for example, between basic drives and learned inhibitions). In this instance, *psychotherapy* is indicated. The danger situation may be an *external* one, such as threats to health or occupation. *Medical treatment* or *counseling* may be necessary in these situations. On occasion, it may be necessary to advise *environmental change* or suggest *new coping strategies* (especially in the presence of action–cognition dissonance). If

the anxiety-provoking situation is pervasive and seems to be a result of *faulty learning, behavioral treatment modalities*, such as desensitization or learning of relaxation techniques, may be helpful.

If a target-organ disorder, such as hypertension, is present, *it should be treated medically in parallel with the management of the psychological and social factors* that might have contributed to it. Excessive anxiety determined predominantly by *intrinsically unstable brain structures* (acute toxic or structural brain damage) may be managed successfully by teaching the patient new coping strategies and relaxation techniques.

Summary

Anxiety, together with pain, is one of the most common major causes of help-seeking behavior. The experience of anxiety involves *subjective* feelings of fear and dread that are usually vague, although on occasion the patient may complain of specific fears such as that of dropping dead. *Physical examination* will usually reveal signs of sympathetic nervous system activation, including rapid pulse rate, elevated blood pressure, and excessive sweating.

Anxiety can be regarded as a *warning response* to impending danger that may be external or intrapsychic. The term "signal anxiety" in psychoanalytic theory refers to the special case in which the person is not aware of the nature of the danger situation (i.e., the impending situation is internal, psychological, and "unconscious"). Anxiety may develop as a conditioned response to previous exposure to unpleasant or dangerous situations. Anxiety serves the adaptive *functions* of preparing the organism for fight or flight, of mobilizing psychological defense mechanisms, and of facilitating performance.

All parts of the *brain* participate in the anxiety mechanism. The most important parts of the brain include the neocortex for processing of information, the limbic system for the emotional reactions leading to the neural and endocrine discharge via the hypothalamus, the brain stem nuclei, especially the locus ceruleus, which may play a major role in the generation and suppression of the anxiety–fear response, and the reticular activating system for the arousal levels of the central nervous system accompanying anxiety.

Many neurotransmitters are involved in anxiety modulation. The GABA–benzodiazepine–chloride ionophore complex plays a particularly important role. In addition, norepinephrine, serotonin, and endorphins probably play major roles in anxiety regulation.

Activation of specific parts of the hypothalamus in anxiety–fear re-actions results in specific patterns of excitation of autonomic and en-docrine systems. Anxiety is usually associated with excitation of the autonomic nervous system and altered function of neuroendocrine sys-tems.

Although anxiety serves a useful function in moderate levels, it may, so to speak, go out of kilter, resulting in "anxiety dysregulation syndromes." Dysregulation may have multiple causes, and any com-bination of the following may be involved: the perceptual apparatus, the brain and personality system, the neuroendocrine and autonomic nervous systems, target organs, organism–environment interaction, and iatrogenic factors.

Anxiety dysregulation syndromes may be classified broadly into (1) disorders in the experience of anxiety (excessive or insufficient), (2) psy-chophysiological disorders, and (3) dysfunction in behavior, including heterothetic behaviors and cognition–action dissonance.

Evaluation of anxiety should include the determination of the pres-ence of the *state* of anxiety by means of indicators including subjective reports and an evaluation of the *contexts* of anxiety, which include cur-rent, recent, and background contexts. The recent context includes de-termination of the possible danger situation generating the anxiety, re-cent life events, and stresses. The background context includes the cultural factors, early learning factors, and the *trait* of the patient in experiencing anxiety.

The *management* of anxiety should naturally follow from the infor-mation obtained in the evaluation phase. Excessive anxiety may be suc-cessfully treated by medication, behavioral treatment modalities, and reassurance. The danger situation should be identified and coped with, for which psychotherapy or counseling is often indicated. Target-organ disorders may need specific medical treatment.

In general, antianxiety medications should be used only temporarily to alleviate massive and paralyzing anxiety while evaluation and treat-ment continue to deal with the situational and psychological factors that are generating the anxiety.

Implications

For the Patient

Since anxiety is an unpleasant and vague feeling the cause of which is usually not obvious, some patients may tend to *attribute the cause of*

dysphoria to a physical illness or may displace their concern onto physical sensations associated with minor disorders. Thus, the presence of anxiety is one of the most common reasons for help-seeking behavior, *ostensibly for other physical symptoms* (heterothetic, or problems of living presenting as a symptom). In such instances, the fact that physical examination and laboratory tests reveal no serious medical condition will not completely reassure the patient, since the cause of the anxiety was not related to the physical symptoms in the first place. If the situation (usually interpersonal or intrapsychic or both) that is generating the anxiety is not explored by the physician, the patient often interprets continuing high levels of anxiety as evidence that the physican either lacks competence or "does not care."

For the Physician

The physician should be aware that the patient is *suffering* when anxiety is present. Alleviation of this suffering can be achieved only by comprehensive evaluation and management of the patient. Although anxiety often results in heterothetic behaviors, the physician should also be aware that (1) actual physical illness, especially chronic illness, may increase the patient's propensity to experience anxiety and (2) on occasion, some medical illnesses *simulate* anxiety states. Any disease affecting the brain and peripheral structures concerned with anxiety mechanisms may mimic anxiety states. A rational management plan can be formulated only after a thorough evaluation of the patient according to the principles outlined in this chapter. Emphasis should be placed on understanding the recent context, or the danger situation to which the patient may be responding with anxiety. To elicit this information, the physician has to ask *specific questions* about recent events, such as the patient's *relationships* with spouse, relatives, and friends, the financial situation, and the health status of people close to the patient. Questions should be asked about the patient's *demographic data* and early experiences, especially in relation to anyone who had a disease or symptom similar to the one the patient is complaining of now, in order to elicit information concerning the background context. The patient should also be asked whether or not he has a tendency to feel anxious or to get upset easily, whether he has had many physical illnesses and complaints in the past, etc. (the "trait" of the patient). An evaluation of the current context should include the question, "What do you think is causing your present symptoms?" (e.g., palpitations, dizziness, or whatever). Often, the patient may tell you, "I think it's because I have cancer" (or heart

disease, or leukemia, etc.). This should be followed by the question, "Why do you think that?" or "Do you know of anyone who had cancer?" (or heart disease, or leukemia, etc.). This will often give the physical information concerning what the *meaning* of the symptom might be for the patient and, sometimes, what the danger situation might be, given the patient's unique experiences and exposures ("priming factors").

For the Community and the Health-Care System

We are often told that we live in an age of anxiety. We have seen that one of the important elements in reducing undesirable effects of anxiety (or perhaps anxiety itself) in animals and humans is a *sense of control or mastery* and *feedback* indicating that the attempt to control or master the situation generating anxiety has been successful. At the level of social systems, it seems imperative to facilitate, as much as possible, the sense of mastery available to the individual members and to provide feedback of such mastery. More responsive decision-making and administrative structures with a minimum of bureaucratic red tape (an excellent device to reduce or delay feedback) at all levels of the social system would be highly desirable. This would apply particularly to the health-care system, including the hospital, the ward, and the treatment team.

Patients with anxiety disorders can become more anxious, frustrated, and sick when their requests for p.r.n. ("when necessary") medications (or bedpan or whatever) remain unanswered or ignored for hours.

Medical curricula should include the evaluation and management of anxiety and anxiety dysregulation disorders. Special emphasis should be placed on the role of anxiety in facilitating heterothetic help-seeking behaviors and in the treatment of target organ disorders, as well as on the psychopharmacological management of acute anxiety. Drug treatment of anxiety should always be accompanied by an attempt to understand and cope with the danger situation and underlying factors generating the anxiety.

Recommended Reading

Brenner C: *An Elementary Textbook of Psychoanalysis*. New York, International Universities Press, 1955, pp 81–92. An exceptionally lucid, accurate, and detailed synopsis of the theory of signal anxiety. The entire volume is recommended as an excellent and authoritative introduction to psychoanalytic theory.

Freud S: Inhibitions, symptoms, and anxiety, in Strachey J (ed): *The Standard Edition of the Complete Psychological Works of Sigmund Freud*. London, Hogarth Press, 1953, Vol 20, pp 71–175. (Originally published in 1926). A classic psychoanalytic view of the mechanisms of anxiety and symptom formation.

Hilgard ER, Bower GH: *Theories of Learning*. Englewood Cliffs, New Jersey, Prentice-Hall, 1975. This is a comprehensive textbook on various learning theories and theoreticians. The chapters on Pavlov's classic conditioning and Skinner's operrant conditioning are well worth reading if you have not already read them in a psychology course. A good reference concerning any questions about learning theory and conditioning.

Isaacson RL: *The Limbic System*. New York, Plenum Press, 1974. A review of the structure, functions, and experiments concerning the limbic system. Recommended for those interested in the specific structures.

Izard C: *Patterns of Emotions*. New York, Academic Press, 1972. A comprehensive psychological analysis of anxiety and depression. The author believes that anxiety is a complex emotion consisting of fear, distress, and anger.

Kollar EJ, Alcalay M: The physiological basis for psychosomatic medicine: A historical view. *Ann Intern Med* **67**:883–895, 1967. A nice and reasonably comprehensive review of the psychophysiology of emotions.

Levi L: *Emotions: Their Parameters and Measurement*. New York, Raven Press, 1975. This is a multiauthored comprehensive volume on the biological and psychophysiological aspects of emotion, including anxiety. Quantification of emotion is also discussed extensively. An excellent reference.

Reiser MF: *Mind, Brain, Body: Toward a Convergence of Psychoanalysis & Neurobiology*. New York, Basic Books, 1984. Chapters 7, 11, and 12 of this book deal with the psychoanalytic theme of signal anxiety. In a detailed way Chapter 7 compares, contrasts, and relates it to classical Pavlovian theories and suggests ways of relating these two theories to newer empirical research in infant development and to contemporary thinking in the fields of cognitive psychology and evolutionary biology.

Spielberger CD (ed): *Anxiety: Current Trends in Theory and Research*. New York, Academic Press, 1972. This two-volume multiauthored book deals more specifically with anxiety, its nature, measurement, biological aspects, and relationship to stress.

Warburton DM: *Brain, Behavior and Drugs*. London, John Wiley & Sons, 1975. A comprehensive review of the neurochemistry of behavior, including mood, homeostasis, sleep and dreams, and memory.

References

Aghajanian GK: Tricyclic antidepressants and single-cell responses to serotonin and norepinephrine: A review of chronic studies, in Usdin E, Bunney WE, Davis JM (eds): *Neuroreceptors—Basic and Clinical Aspects*. New York, John Wiley & Sons, 1981, pp. 27–35.

Bartrop RW, Luckhurst E, Lazarus L, *et al.*: Depressed lymphocyte function after bereavement. *Lancet* **1**:834–836, 1977.

Brady, JV: Ulcers in "executive" monkeys. *Sci Am* **199**:95–100. 1958.

Brenner C: *An Elementary Textbook of Psychoanalysis*. New York, International Universities Press, 1955.

Bullock K, Moore RY: Nucleus ambiguus projections to the thymus gland: Possible pathways for regulation of the immune response and the neuroendocrine network (abstract). *Anat Rec* **196**:25, 1980.

Cannon WB: *The Wisdom of the Body*. New York. WW Norton & Co, 1932.

Cannon WB: "Voodoo" death. *Am Anthropol* **44**:169–181, 1942.

DeSilva RA, Lown B: Ventricular premature beats, stress, and sudden death. *Psychosomatics* **19**(11):649, 1976.

Dorian BJ, Keysteone E, Garfinkel PE, *et al.*: Immune mechanisms in acute psychological stress (abstract). *Psychsom Med* **43**:84, 1981.

Edwards AB, Acker LE: A demonstration of the long-term retention of a conditioned GSR. *Psychosom Med* **24**:459–463, 1962.

Emrich HM, Millan MJ: Stress reactions and endorphinergic systems. *J Psychosom Res* **26**:101–104, 1982.

Engel GL: *Fainting*, ed 2. Springfield, Illinois, Charles C Thomas, 1962.

Everett NB, Sundsten JW, Lund RD: *Functional Neuroanatomy*, ed 6. Philadelphia, Lea & Febiger, 1971.

Freud S: Inhibition, symptoms, and anxiety, in Strachey J (ed): *The Standard Edition of the Complete Psychological Works of Sigmund Freud*. London, Hogarth Press, 1953, Vol 20, pp 77–175. (Originally published in 1926.)

Gray JA: The structure and emotions of the limbic system, in *Physiology, Emotion and Psychosomatic Illness. Ciba Foundation Symposium 8 (New Series)*. New York, Ciba Foundation, 1972, pp 87–130.

Guyton AC: *Textbook of Medical Physiology*. Philadelphia, WB Saunders Co, 1976.

Hartmann H: *Ego Psychology and the Problem of Adaptation*. New York, International Universities Press, 1958.

Hartmann H: The mutual influences in the development of ego and id, in *Essays on Ego Psychology: Selected Problems in Psychoanalytic Theory*. New York, International Universities Press, 1964.

Hazum E, Chang KJ, Cuatrecasas P: Specific nonopiate receptors for beta-endorphin. *Science* **205**:1033–1035, 1979.

Hoehn-Saric R: Neurotransmitters in anxiety. *Arch Gen Psychiatry* **39**:785–742, 1982.

Hunkeler W, Mohler H, Pieri L, *et al.*: Selective antagonists of benzodiazepines. *Nature (London)* **290**:514–516, 1981.

Isaacson, RL: *The Limbic System*. New York, Plenum Press, 1974.

James W, Lange CG: *The Emotions*. Baltimore, Williams & Wilkins, 1922.

Locke SE: Adaptation and immunity: Studies in humans. *Gen Hosp Psychiatry* **4**:49–59, 1982.

Lown B, Vemier RL, Rabinowitz SH: Neurologic and physiologic mechanisms and the problems of sudden cardiac death. *Am J Cardiol* **39**:890–902, 1977.

MacLean P: Psychosomatic disease and the "visceral brain." *Psychsom Med* **11**:338–353, 1949.

Martin, JB, Reichlin S, Brown G: *Clinical Neuroendocrinology*. Philadelphia, FA Davis, 1977.

Mason JW: Clinical psychophysiology: Psychoendocrine mechanism, in Reiser MF (ed): *American Handbook of Psychiatry*, ed 2 Vol 4: *Organic Disorders and Psychosomatic Medicine*. New York, Basic Books, 1975, pp 553–582.

Ninan PH, Insel TM, Cohen RM, *et al.*: Benzodiazepine receptor-mediated experimental "anxiety" in primates. *Science* **218**:1332–1334, 1982.

Papez JW: A proposed mechanism of emotion. *Arch Neurol Psychiatry* **38**:725–743, 1949.

Paul SM, Marangos PJ, Skolnick P: The benzodiazepine–GABA–chloride ionophore receptor complex: Common site of minor tranquilizer action. *Biol Psychiatry* **16**:213–229, 1981.

Pavlov IP: *Conditioned Reflexes*. London, Oxford University Press, 1927.

Redmond DE Jr, Huang YH: New evidence for a locus ceruleus–norepinephrine connection with anxiety. *Life Sci* **25:**2149–2162, 1979.

Redmond DE, Maas JW, Huang YH, *et al.*: Does the locus ceruleus mediate anxiety and fear? Presented at the meeting of the American Psychiatric Association, Toronto, 1977.

Richter CP: On the phenomenon of sudden death in animals and man. *Psychosom Med* **19:**191–198, 1957.

Roessler RL, Cate TR, Lester JW, *et al.*: Ego strength, life change and antibody titers. Paper presented at the Annual Meeting of the American Psychosomatic Society, Dallas, Texas, March 25, 1979.

Schachter S, Singer JE: Cognitive, social, and physiological determinants of emotional state. *Psychol Rev* **69:**379–399, 1962.

Schwartz GE: Psychosomatic disorders and biofeedback: A psychobiological model of dys-regulation, in Maser JD, Seligman MEP (eds): *Psychopathology: Experimental Models.* WH Freeman, San Francisco, 1977.

Seligman ME, Maier SF: Failure to escape traumatic shock. *J Exp Psychol* **74:**1–9, 1967.

Selye H: *The Physiology and Pathology of Exposure to Stress.* Montreal, Acta, 1950.

Solomon RL, Wynne LC: Traumatic avoidance learning: The principles of anxiety conversion and partial irreversibility. *Psychol Rev* **61:**353–365, 1954.

Snyder SH: Opiate receptors and internal opiates. *Sci Am* **236:**45, 1977.

Squires RF, Braestrup C: Benzodiazepine receptors in rat brain. *Nature (London)* **266:**732–734, 1977.

Stein M, Schiavi R, Camierino M: Influence of brain and behavior in the immune system. *Science* **191:**435, 1976.

Weiss JM: Effects of coping responses on stress. *J Comp Physiol Psychol* **65:**251–260, 1968.

Williams JM, Peterson RG, Shea PA, *et al.*: Sympathetic innervation of murine thymus and spleen: Evidence for a functional link between the nervous and immune systems. *Brain Res Bull* **6:**83–94, 1981.

Wybran J, Appelboom T, Famey JP, *et al.*: Suggestive evidence for receptors for morphine and methionine-enkephalin on normal human blood T-lymphocytes. *J Immunol* **123:**1068–1070, 1979.

Wynne LC, Solomon RL: Traumatic avoidance learning: Acquisition and extinction in dogs deprived of normal peripheral autonomic functioning. *Genet Psychol Monogr* **52:**241–284, 1955.

Psychological Defense Mechanisms

1. A 45-year-old man was quite surprised to discover that he had completely forgotten his doctor's appointment one Monday until his wife asked him about it that same evening. He had suffered severe chest pain a week before, but after it subsided, he decided not to call the doctor because his routine checkup appointment had already been scheduled for that Monday.

2. A young physician admitted a 29-year-old woman to the surgical service but could not understand his patient. Earlier the same day, she had gone to her family doctor's office complaining of a sore throat. When her doctor examined her, he found an area of ulceration on her breast and a hard lump under the ulcerated area. On admission to the surgical service, she told the admitting physician that she entered the hospital because of a sore throat— completely ignoring the breast condition. When asked about it, she said that she had noticed it three months ago but had not paid any attention to it.

3. A respected surgeon specializing in the surgical treatment of cancer always appears cheerful and optimistic. When a colleague commented to him that it must be difficult to maintain such cheerfulness while treating cancer patients, he said, "All cancer patients want to be cheerful and optimistic. So, I am cheerful, and they are cheerful." One of his patients, however, was not always cheerful and was referred to the psychiatrist. She told the psychiatrist, "I feel like a failure. I cannot keep up a cheerful front for my doctor."

4. A patient in the coronary care unit who had witnessed the death of the patient in the next bed was asked what had happened. He said, "The guy

in the next bed improved and was transferred to the medical floor, since he doesn't need to be on intensive care any more."

5. An intern proposed a crippling radical operation for a cancer patient, even though the cancer was widespread throughout the body and could not be ameliorated by the proposed surgery.

6. A 45-year-old man underwent open-heart surgery for replacement of the mitral valve. He was recuperating satisfactorily in the recovery room until the second postoperative day. At noon, the nurses noted that he was becoming confused and restless. Shortly thereafter, he was openly agitated and attempted to pull out his intravenous tubing. He insisted that the women in the room (nurses) were plotting to kill him by injecting poison through the intravenous apparatus. On mental-status examination, he was noted to be grossly disoriented as to time and place and unable to remember even simple things such as a three-digit number.

In the first five cases presented above, either the patient or the doctor avoided or minimized a potentially unpleasant situation and feelings—for example, finding out that he or she might have a serious disease (vignettes 1 and 2), having to discuss the gravity of the illness with the patient, or recognizing that patients may sometimes feel discouraged (vignette 3). This kind of avoidance or minimization is effective only temporarily and, in the long run, is often *detrimental*—unduly delaying help-seeking behavior and prompt medical treatment. On the other hand, as a short-term reaction, it can be *beneficial* when the *danger situation abates spontaneously* or in situations when *everything that can be done to deal with the danger is already being done* and when the *physiological change of anxiety could be harmful*. For example, patients who deny feeling frightened or apprehensive in the coronary care unit for the first few days after suffering from a heart attack have been shown to have better survival rates than patients who admit feeling frightened or apprehensive (Hackett *et al.*, 1968). Understanding the psychology of defense mechanisms can help in developing an appreciation for some of the (sometimes inscrutable) adaptive and maladaptive ways that patients and doctors behave when exposed to the myriad of unpleasant situations and emotions connected with illness and death.

Distress, Anxiety, and Defense Mechanisms

As we discussed earlier, help-seeking behavior occurs when a person has reached his limit of tolerance of anxiety because of a symptom or disability. Anxiety arising from problems of living may also result in

help-seeking behavior by lowering the person's threshold for tolerance (e.g., of pain), increasing his awareness of discomfort associated with the symptom, or motivating him to seek comfort through interpersonal contact with the doctor. In each of these situations, the common denominator is a *felt distress* experienced by the patient (or, in some instances, by other persons involved with the patient) that moves him to seek help.

When perceptual and psychological stimuli activate *memories* associated with dangerous situations or are themselves perceived and evaluated as being *directly threatening*, then *anxiety* results. The brain structures as well as the physiological and psychological mechanisms involved in the generation of anxiety and the anxiety response have been discussed in Chapter 4. We have seen that anxiety serves as a signal to the organism that it has to somehow cope with a potentially dangerous situation. Human beings may deal with anxiety (and thus the danger situation) in two ways: (1) by fleeing from or eliminating the source of the stimuli, that is, *fight or flight*, or (2) by internal adjustments in the central nervous and psychological systems whereby the anxiety sensation is reduced and the potential danger situation "avoided," although the stimuli may still be present. This is always the case when the stimuli are internal rather than external, since we cannot flee from internal psychological–motivational sources of potential danger. The internal adjustment mechanisms are called *psychological defense mechanisms.*

When the stimuli associated with danger come from outside the person, elimination of the source would be most desirable, but is not always possible. Sometimes we have to make *inner adjustments to ignore unpleasant events* outside because we are not powerful enough or are otherwise unable to change them. If the danger situation exists internally—that is, if a psychological conflict has been activated that, if allowed to run its course, threatens to overwhelm the person emotionally—the only resource lies in the use of *internal adjustments*. For example, certain past memories or mental representation of forbidden conflictual drives may be rendered inaccessible to consciousness; this is called *repression* in psychological terms. Of course, there must be changes in the central nervous system that subserve these phenomena, such as, perhaps, attenuation of associative pathways in parts of the brain involved in memory storage and retrieval. We do not yet have the technology that would permit study and description of these different aspects in a unified way. The best we can do, then, is to refer to states and changes in the brain that manifest themselves in both the physio-

logical and psychological realms and to describe separately and in parallel the changes in the two realms insofar as they are known.

We should emphasize that the defense mechanisms come into being *automatically* ("unconsciously"), that is, without conscious "willing" on our part. According to psychoanalytic theory, the generation of a small amount of anxiety as a signal is considered to usher in the defense mechanisms, which, in turn, avoid the development of clinical anxiety. There are individual variations in the kind of defense mechanisms habitually used in the face of potential danger. Some persons usually or preferentially cope with danger situations by using internal adjustment processes (defense mechanisms), others use mainly outer-directed defensive activity, and some use both.

During the course of development, each person develops a repertoire of defenses, some of which are called into play (separately or in combinations) so habitually that, in effect, they become part of the person's character style. For example, if someone habitually attributes inner unacceptable feelings to others (this defense is called projection; see below), we may speak of him as a paranoid character. Since habitual defense mechanisms can be expected to be utilized when a person encounters threatening events, including discovery of symtoms or signs of disease, these character styles have special implications for medical management, as we will see in Chapter 17.

It seems likely that developmental environmental influences, especially learning experiences within the family, contribute to the development of defenses and defensive repertoires, but there is still a great deal to be learned about the ontogeny of defenses.

As the term "defense" implies, these mechanisms are usually quite adaptive and are necessary in daily life, serving useful functions, just as inflammation serves useful physiological defense functions. On the other hand, exaggerated or persistent use of certain defensive maneuvers may lead to psychopathology, just as persistent inflammation may itself contribute to certain diseases.

When defenses break down, increasing conscious experiences of free anxiety and awareness of danger develop and may lead to help-seeking behavior. In addition, as we saw in Chapter 4, when the physiological activation is severe and prolonged, the stage may be set for precipitation or exacerbation of stress-related illnesses (Sachar *et al.*, 1970). The variety of effective defenses and combinations of defenses that are available for flexible and appropriate mobilization when needed serve as one highly important measure of a person's capacity to adapt

successfully and maintain health in a dynamically stressful environment. This assessment is a major consideration that a psychiatrist takes into account in appraising the strength ("ego strength") of the personality.

Our discussion of defenses is oriented toward the medical reader; we have tried whenever it is appropriate to emphasize the ways in which defenses manifest themselves so as to influence the sick role, illness, and professional behaviors. Our main concern is their relevance to medical practice in all its aspects—diagnosis, treatment, research, and prevention.

Classification of Defense Mechanisms

Despite the importance of their functions, there is, unfortunately, no standard system for classifying defenses—nor is there, for that matter, any standard comprehensive list of them. The classification systems that are offered differ from one another. Anna Freud (1966) proposed that repression be regarded as the primary defense, with the others then to be regarded as reinforcing it. Meissner *et al.* (1975) (following Vaillant) classify defense mechanisms as narcissistic, immature, neurotic, and mature defenses. Others classify them as successful or unsuccessful, still others as defenses that are directed primarily toward outer dangers and those that are primarily inwardly oriented. In the discussion of the defenses that follows, we have grouped them according to a scheme that is compatible or syntonic with the general systems approach that we have used in the rest of the book.*

The personality system can be seen as consisting of *input, internal processing–decider,* and *output* apparatuses or subsystems for handling information. General system concepts can be applied to classify the defense mechanisms on the basis of the subsystem most affected by the defensive process (see Figure 8) (Leigh and Reiser, 1982). "Input" refers to the perceptual apparatus, "internal processing" refers to the associative and memory connections, "decider" refers to the hypothetical "executive" apparatus (of the ego), and "output" refers to the devices subserving motor (including verbal) function. Obviously, for any defense mechanism to be mobilized, there must be some perception of a danger situation, and the decider, through input from the internal pro-

---

* This is for the sake of consistency and continuity and to facilitate transfer of concepts and understanding between different sections of the book and permit extrapolations from one section to another.
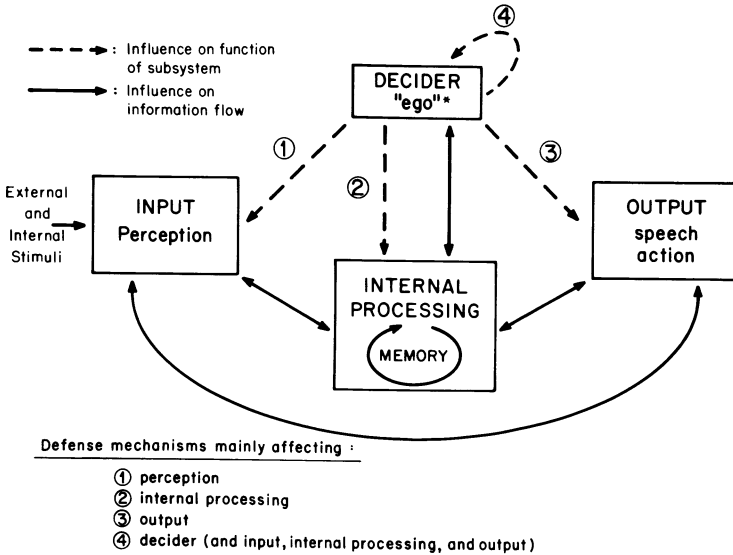
Figure 8. General systems diagram for defense mechanisms. Executive and defensive functions of the "ego."

cessing apparatuses, mobilizes defense mechanisms, which in turn act on the functions and channels leading to any of the subsystems. Since the personal system is an open system, and all the subsystems have mutual feedback, any change in one subsystem usually affects all other systems more or less, and so, to some degree, all are involved (see Figure 8). The scheme we are using here classifies or groups defenses according to which subsystem—input, internal processing, output, or decider itself—is the one the altered function of which constitutes the presenting or most obvious manifestation of the defensive operation. Thus, *denial* of an external danger manifests itself mainly in the input (perceptual) subsystem. *Acting out* manifests itself mainly in the output subsystem. *Rationalization* manifests itself mainly in the internal processing subsystem. This scheme, then, simply highlights the subsystem manifestly most affected when the defense mechanism is in operation. A subsystem's function can be either increased, decreased, or altered, for example, by a reversal process in which an output device may send out a message that is opposite in character from the information contained in the internal processing subsystem. (As a result, a person may, for example, express hate in place of love or suspicion instead of trust.)

Defenses Listed According to Their Main Subsystem Effects*

1. Defense mechanisms that manifest their main effects in the *input* subsystem. These mechanisms reduce anxiety by changing the perception of a stimulus field that heralds the development of a potential danger situation by:

    a. Diminution or negation of its perception (*denial*); this may even occasionally take the extreme form of negative hallucinations.
    b. Switching the direction of feelings toward and/or thoughts about a highly important and meaningful person onto another, less meaningful, one (*displacement*), thereby enabling the individual to feel comfortable and safe with the negative thoughts or emotions.†
    c. Perceiving certain feelings, characteristics, or thoughts as belonging to someone other than oneself (*projection*) or as being located within the self rather than in another person (*introjection*).
    d. Reducing the perceptual sphere in a massive fashion (*constriction of awareness*).

2. Defense mechanisms that manifest their main effects in the *internal processing* subsystem. (Those affecting mainly the executive or decider subsystem are discussed separately below.) The mechanisms we have included in this category involve mainly changes in cognitive, associative, and integrative functions of the brain:

    a. A stimulus or percept that reactivates and would ordinarily stimulate conscious recall of an old forgotten memory may fail to do so because of *repression*, a defense that attenuates association such that stored memories do not reach conscious awareness.
    b. If the meaning of the motives underlying a particular action has been repressed, another reasonable but more socially and personally acceptable meaning may be substituted and considered by the person to constitute the motivation for that action (*rationalization*).

---

* Illustrative examples of each of the defense mechanisms will be given in a later section of this chapter.
† This mechanism, which may also involve considerable alterations in output and internal processing mechanisms, is grouped here with those involving perception as their primary mode of presentation, since the object of the negative thought or feeling is *perceived* to be different from the original object.

c. *Intellectualization* refers to use of intellectual activity in an intense effort to master feelings through objective understanding.

d. When conflictual memories and ideas enter conscious thought or are verbalized without activation of appropriate affects, the defense of *isolation* is being utilized.

e. *Fantasy and daydreaming* involve associative processes that evoke pleasant feelings and may aid or reinforce denial by diverting attention away from anxiety-provoking stimuli in the environment.

f. The affective quality associated with a particular idea or action may be turned into its opposite (*reaction formation*); for example, a craving may be reversed and perceived as a revulsion.

3. Defense mechanisms that manifest their main effects in the *output* (action) subsystem. These mechanisms reduce anxiety by:

a. Concealing meanings from recognition by expressing them as actions rather than as thoughts (*acting out*).

b. Attempting to achieve mastery of a danger by direct active confrontation; for example, the lion tamer exposes himself to the very situation he fears and achieves a sense of security through mastery (*counterphobic maneuvers*).

c. Doing something that is considered to negate or "cancel out" a dangerous or guilt-producing action already perfomed (*undoing*).

d. Engaging in an activity that is socially acceptable and worthwhile in its own right but that at the same time may covertly gratify a conflictual forbidden desire (*sublimation*).

e. Finally, *activity* of any sort may serve in a nonspecific way to distract one's attention from the anxiety-producing situation and thus provide some relief.

4. Defense mechanisms the main effects of which are more evenly distributed in the subsystems. The effects of regression and identification uniformly pervade all the functional subsystems through the *decider* or executive functions.

a. In the face of anxiety, we may have a tendency to revert to earlier modes of thinking, feeling, and acting in an effort to feel as we did at an earlier epoch of life when we were free of the kinds of anxiety-producing situations that now confront

us or when earlier developmentally age-appropriate modes of behavior were successful in dealing with the kind of situation that is producing anxiety at present (*regression*). When this mechanism is manifest, we may, at least in part, perceive things as though we were a young child, concern ourselves increasingly with issues that were important in childhood, and behave and act in less mature ways. Regression may, of course, be maladaptive. In many situations, however, it serves adaptive purposes, especially when regulated and controlled, as, for example, in psychotherapy and creative activity. It can facilitate a hospitalized patient's adaptation to the sick role. When regression is excessive and uncontrolled, it can seriously impair the capacity for rational, mature adaptive behavior.

b. Another mechanism that affects all the input, internal processing, and output apparatuses is *identification*. By modeling much of one's own behavior and self-image on another person's ways of perceiving, thinking, acting, dressing, speaking, and so forth, one may unconsciously feel as though one *is* the other person. This mechanism may protect against the anxiety over loss of, or fear of separation from, a loved person. It is also extensively involved in healthy growth, maturation, and learning.

Illustrations of Defense Mechanisms as They May Be Encountered in Medical Practice

Defense Mechanisms That Manifest Their Main Effects in the Input Subsystem

Denial. This is a mechanism by which a person manages to remain unaware of potentially anxiety-provoking external situations, perceptions, or emotional states. Some patients who have suffered heart attacks can in this way minimize the anxiety-provoking aspects of the situation and remain calm.

As noted earlier, this may have a salutary effect on the patient's clinical course during the early acute phase in the coronary care unit—probably by sparing the damaged heart muscle from physiological burdens that may accompany anxiety, such as increased demand for cardiac work or stimulation of irritable injured ventricular muscles by circulating epinephrine. It can be over-done, though; for example, some patients,

as soon as the pain subsides, refuse to believe that they have suffered a heart attack and sign out of the hospital against medical advice—only to be brought back a few hours later pronounced "dead on arrival." Similarly, too much denial during the recovery and rehabilitation phase of illness may interfere with a patient's ability to follow a prescribed regimen of carefully graded return to activity.

Some women who have found lumps on the breast may, through the use of this mechanism, delay consulting a physician (see vignettes 2 and 4).

Physicians may also use denial both adaptively and maladaptively. In dealing with a patient who is terminally ill, a certain amount of denial (of the seriousness of the illness) may be manifested as optimism in the physician's attitude, and this may help the patient to sustain the hope that is so essential in coping with his condition. Excessive denial, however, may strain the patient's credulity or capacity to respond (vignette 3) or may lead to unrealistic planning (vignette 5). Denial is one of the most common and important defense mechanisms encountered in medical practice, in both patients and physicians. It may be reflected in help-seeking behavior and in physicians' behavior and may affect the course, treatment, and outcome of disease.

Displacement. In this process, conflictual feelings, thoughts, or impulses are directed away from an emotionally significant person and instead perceived as belonging to another person. For example, a patient who was actually insulted by the doctor felt offended by the nurse and was abusive toward her; a resident physician who had been criticized by his chief of staff became angry with his patient.*

Displacement occurs more easily when the secondary object is in some way actually or symbolically related to the original one. A patient who has unresolved angry feelings toward his mother may be prone to feel angry with the nurses because the nurturant, caretaking (mothering) aspects of the nurse's role evoke conflictual memories and feelings about the mother.

Displacement is operative in heterothetic patient behavior. The emotional suffering and anxiety arising from problems of living may be shifted to a physical symptom in instances where contacting the physician for a physical symptom is more acceptable to the patient than doing so for emotional reasons.

---

* Behavior akin to displacement also appears in animals (Tinbergen, 1953; Lorenz, 1966). For example, a cat that has been made more aggressive by a dog's barking may attack an inanimate object instead.

Projection. This is a mechanism by which unacceptable feelings and thoughts within oneself are attributed to and perceived as belonging to someone else. A patient may insist that he is consulting the physician only because his wife is upset about his symptoms, though he "knows" that "the symptoms are of no consequence."

Projection is the major defense involved in the development of paranoid feelings and ideas. A patient who develops erotic feelings toward the physician may accuse him of being seductive or of making sexual advances. Confusing environmental cues (such as those encountered in intensive care units and recovery rooms) or lowered capacity to put together such cues and percepts (as in organic brain syndromes) facilitate projection and formation of paranoid persecutory delusions (see vignette 6). A physician uncomfortable about discussing the prognosis of a serious or terminal illness may feel that the patient does not wish to "know" (see vignette 3).

Introjection. This process, whereby the qualities of an emotionally important person are perceived as being within or belonging to the self, may lead a patient to regard a part of his body (such as a diseased organ) as though it were someone else. For example, a patient with cancer felt as though the cancer within her were her mother persecuting her from within her own body (see Chapter 21). This may occur at a conscious or an unconscious level. If conscious, it constitutes a frank delusion. If unconscious, it may give rise to neurotic attitudes about the illness or its treatment.

Constriction of Awareness (Constriction of the "Ego"). This is a process whereby the person reduces his sphere of awareness to exclude unpleasant situations from becoming conscious. This is usually achieved by focusing all one's awareness on some limited aspects of one's life at the expense of other aspects. For example, a patient with extensive and severe burns may focus his attention exclusively on his pain to avoid becoming aware of the possibility of being seriously disfigured.

Terminally ill patients often use constriction of awareness—being concerned and preoccupied with minor symptoms allows them to be unconcerned with (the implications of) the illness itself. Students are often surprised at the excessive concern over minor discomforts and seeming lack of concern about prognosis in seriously ill patients.

Defense Mechanisms That Manifest Their Main Effects in the Internal Processing Subsystem

Repression. This is the mechanism that underlies forgetting of unpleasant things—it bars unwanted impulses, memories, desires,

thoughts, and other feelings from consciousness. Mental content so "forgotten" subjectively no longer seems to exist, but that it does remain in existence in the "unconscious" is evidenced by the fact that there are circumstances under which it may again become conscious, such as:

1. When critical functions of the mind are weakened, as in toxic states of the brain (e.g., drugs, severe fatigue, fever). For example, a patient with organic brain syndrome may use obscene language and may alarm the nurses with "improper suggestions." (Normally unacceptable impulses are expressed due to reduced repression.)
2. When the critical functions of the mind are suspended, as in sleep (dreaming) or under hypnosis.
3. When the balance of forces between strength of repression and strength of the impulses is upset by an upsurge in pressure of the impulses such as occurs in puberty, after long periods of abstinence, or when life circumstances are particularly stimulating or seductive.

Repression operates unconsciously. There is a (probably related) conscious mechanism called *suppression* that refers to a conscious (intentional) effort to forget something or put it out of one's mind. The accesibility or recovery into consciousness of forgotten material may vary from being *relatively* recoverable (e.g., momentary forgetting; see below) to being deeply and firmly barred from recall, as is the case with highly charged traumatic events. The latter may exert considerable influence on behavior, including that involved with health and illness. One striking example is the phenomenon known as the *"anniversary reaction,"* in which it has been observed that patients may become ill (e.g., suffer a heart attack) on the forgotten anniversary date of a highly charged painful event such as the death of a loved one (Weiss and English, 1957).

The simplest example of repression at work is *momentary forgetting*— the forgotten thought is often shown to be related to unpleasant feelings, either directly or indirectly. Other somewhat longer, lasting examples would be the patient in vignette 1 who conveniently forgot an important doctor's appointment and the case of a patient who, on Monday, was told by his physician that he had cancer and, by Tuesday, had forgotten that he had seen his physician the previous day. Further discussion of this defense and its ramifications would take us beyond the scope of this work; appropriate references should be consulted by those who are interested.

You can imagine for yourself the myriad ways in which emotionally determined forgetting could affect illness and sick-role behavior (e.g., forgetting to take medication).

Rationalization. This is a process whereby a person "explains away" anxiety-provoking situations or meanings. He may advance rational-sounding explanations for behaviors that were motivated by unaccept-able or undesirable feelings or thoughts. The "rational explanations" usually have some degree of validity.

A woman who felt a lump in her breast did not consult a physician, having rationalized that it was due to the menstrual period she was having at the time of discovery. A patient with organic brain syndrome and memory difficulty may attribute his faulty memory to the medica-tions he is taking (which may or may not be partially true). A hospital may require unrealistic working hours and case loads of its house staff "for their training experience," when actually this may be motivated by the need for service.

Intellectualization. This defense is closely related to rationalization and refers to the use of intellectual processes to master anxiety-provok-ing information and situations. After developing a serious illness, a pa-tient may spend much of his time reading about the illness in medical journals and textbooks. Under similar circumstances, another patient may read philosophy books in an attempt to develop a stoic attitude toward the illness.

Physicians invariably and naturally use intellectualization in their professional activities. Without it, constant exposure to illness and death might well be unbearable. Furthermore, it is knowledge and under-standing that render the physician capable of practicing medicine. The wisdom, the skill, and the professionalism of the physician, as well as the objective and impartial attitude toward patients, all presuppose and, to an extent, require the use of intellectual defenses.

Since intellectualization is often an effective defense, physicians may wish to encourage some patients to use it and to provide them with detailed information about the disease, references to appropriate books, and other encouragement.

Isolation. Isolation as a psychological defense mechanism refers to the process whereby unpleasant feelings are dissociated from the cog-nitive process. Thus, a person recognizes a situation or memory without experiencing painful or anxious feelings associated with it. Through this mechanism, a patient may be able to give the physician a detailed nar-

rative of a painful experience, such as protracted pain, without becoming overly emotional. Survivors of catastrophes such as accidents and fires often use isolation when remembering the experience—and provide calm, newspaper-report-like accounts of frightening experiences.

This defense is obviously utilized a great deal by physicians. On occasion, excessive use of isolation by the physician gives the patient the impression that the physician is uncaring and hostile. Excessive isolation on the patient's part sometimes gives the physician the impression that the patient is really not suffering or that he is displaying "bizarre" affect.

Fantasy and Daydreaming. These are mechanisms through which (partial) gratification is achieved through imagination. They may also serve to distract attention away from unpleasant reality. Fantasy may concern or be stimulated by real situations; for example, a physical examination may stimulate the fantasy that the physician will meet the (unfulfilled) sexual needs of the patient. Although the content of daydreams is conscious, not all fantasies are. Patients are often unaware of the fantasy gratification they derive from medical experiences and procedures; for example, painful procedures may satisfy unconscious guilt or masochistic wishes. In extreme cases, this may even lead to the patient's repeatedly seeking operative procedures as though addicted to them. In such cases, the fantasy is acted out through a substitute activity that disguises the true nature of the underlying wishes and needs.

Reaction Formation. Through reaction formation, an unacceptable thought, feeling, or impulse generates its intensely felt opposite in consciousness. Thus, strong affection may be felt instead of hate, great respect instead of contempt, strong affinity instead of repulsion. A patient with unrecognized hostile feelings toward his doctor may become especially cooperative and even ingratiating. A physician may show extra concern toward a patient about whom he might feel hostile. On the other hand, it is also possible for hostility to replace or defend against love or erotic attraction that must be repressed. It should be emphasized again that these mechanisms operate without awareness—what the person actually feels is the (often exaggerated) opposite of the feeling against which he is defending himself.

Defense Mechanisms That Manifest Their Main Effects in the Output (Action) Subsystem

Counterphobic Maneuvers. These refer to the process by which a person exposes himself to or becomes involved in the very activity or sit-

uation he fears. The anxiety may be replaced or made up for by a sense of mastery. The lion tamer in the circus and the reformed alcoholic who has become an abstemious bartender are extreme examples. The patient who reads voluminous books about his illness is behaving counter-phobically and using intellectualization as a defense.

Another example is that of an advanced cancer patient who, on being informed of her diagnosis, established a local chapter of the American Cancer Society within a short time and contributed greatly to a cancer early-detection program in the community. Still another is that of a patient with angina pectoris who regularly jogged every day, against his doctor's advice—he felt reassured when his chest pain after jogging did not reach unbearable intensity.

Undoing. This refers to an act that symbolically nullifies a previously committed act or thought. Rituals and ceremonial acts may be considered to be examples of undoing.

A physician may become oversolicitous or may overmedicate a patient in whose treatment he has made an error.

For some patients, the suffering that accompanies illness may serve the role of "undoing" actions or feelings about which they feel guilty.

Sublimation. Through this mechanism, wishes and impulses that were originally unacceptable can find gratification through acceptable channels. Thus, aggressive drives may be channeled into such socially acceptable activities as surgery; sexual curiosity may find expression in the practice of psychiatry. Sublimation is also important in many creative activities, including intellectual and artistic endeavors.

In the course of development, sublimations may become independent of their defensive (and substitute-gratification) role and continue to be pursued and perfected in their own right—for pleasure, for accomplishment, and for productivity. Each time the surgeon operates, each time the sculptor "attacks" a block of marble, he or she is not necessarily deriving covert gratification of aggressive impulses, even though such may once have been the case and can on occasion again be so.

Activity and Acting Out. Engaging in any sort of activity may help relieve or reduce anxiety or tension in a nonspecific way, and this may be very useful and adaptive. The term "acting out" refers to a specific form of covert gratification, or discharge of unconscious wishes, or reenactment of forgotten experiences through acting that is unaccompanied by explicit recognition of its inner meaning. As in pantomime, the inner

meaning may be obvious to the observer, but in acting out, it is not recognized by the "actor." A patient who convinces (seduces) doctors repeatedly to perform unnecessary exploratory operations for esoteric and vague symptoms may be acting out a forgotten conflictual memory of having been painfully and sadistically attacked or molested.

Withdrawal and Avoidance. These are psychological mechanisms by which a person avoids the occurrence of painful emotions by declining to engage in new interpersonal relationships and disengaging from old ones. Behavioral withdrawal and social isolation often follow. Some patients stop interpersonal contact after being informed of a serious illness because they anticipate and fear social rejection; others actively withdraw by choice to avoid ending up later in a position where the disability will leave them no choice and *force* them to limit interpersonal relationships. Withdrawal and avoidance may enable a patient to achieve a state of "comfortable" emotional detachment. When this is not effective, however, painful affective states such as depression may ensue; in fact, withdrawal and depression are often observed to occur together when the defense is only partially effective.

Defense Mechanisms the Main Effects of Which Are More Evenly Distributed in All the Subsystems by Affecting the Decider or Executive Subsystem Itself

Regression. In this mechanism, cognitive and behavioral aspects of earlier stages of development are reexperienced or reenacted, usually in the mode of an epoch during which the individual enjoyed rather full gratification and relative freedom from anxiety. When regressed, one may in many ways feel and act as though one were a child, or even an infant. Regressive behavior is a prominent feature of the psychopathology of some major psychopathological disorders, for example, in certain stages of schizophrenia. It can also be observed in medical patients, virtually invariably when the illness is severe or of long duration or both. Since the patient is expected to entrust his life and welfare to the health-care personnel (doctor, nurses, and others), he must assume a childlike dependent role. The medical-care setting is especially conducive to the appearance of this phenomenon; in fact, hospitalized patients find themselves literally infantilized—deprived of privacy, being bathed and fed, having to carry out excretory functions in bed, and made almost wholly dependent on others. Given all this, added to the basic

anxiety of being ill and in a hospital, a certain amount of regression is in fact probably necessary to function in the patient role in the hospital.

Along with regression, forgotten memories, conflicts, and feelings of childhood often reappear and lead patients into inappropriate acting out of childhood or infantile conflicts. A patient who without provocation accuses a particular nurse of being hostile to him may be reexperiencing old conflicts over dependency on a mother whom he had as a child perceived as uncaring and hostile. In such a case, the particular nurse might even have some characteristics of appearance or temperament that are reminiscent of the patient's mother; this would facilitate the process but is not a necessary condition for its occurrence. The experiencing of feelings that were associated with an important person in childhood as belonging to someone in the present is the *transference phenomenon*. Transference phenomena are regressive in nature and may utilize additional mechanisms such as displacement and projection.

Identification. Through this mechanism, a person takes on (with only partial or no realization that he is doing so) the characteristics of another person; that is, he *becomes like* the other person—perceiving, processing information, and behaving like the other. This is considered to be a major defense against the anxiety connected with separation from the loved person; that is, if you are (like) the person you love, you do not lose him. *Imitation* is seen as a precursor to identification. Through identification, a person learns a wide variety of values, attitudes, mannerisms, and behavioral characteristics, including illness behavior, sick-role behavior, and help-seeking behavior.

Identification may also be with a feared person (*identification with the aggressor*), in which case the motivating force may be to reduce the fear of the person by becoming like him. Another motivating force for identification may be guilt; taking on a hated person's suffering or symptoms may serve as "just" or "deserved" punishment and so relieve feelings of guilt. An example of this is a patient who developed unexplained shortness of breath and chest pain shortly after his mother's death from chronic lung disease.

As noted earlier, identification plays an important role in healthy growth, development, and learning. The educational development of the physician includes a large element of progressive identification with faculty who act as preceptors and role models. Patients also tend to form partial identification with their physicians, for example, in adopting the physician's attitude and outlook concerning the illness. (In vignette 3, many patients of the surgeon identified with him, taking on his cheerfulness.)

Defense Mechanisms, Anxiety, Character, and Coping Styles

Although the psychological mechanisms intrinsic to the defenses are extensively used in reducing anxiety, most of them—some more than others (e.g., fantasy)—seem to be inherent in human beings and may manifest themselves even in the absence of anxiety or anxiety-provoking situations. Some defense mechanisms are more closely related to particular personalities and psychiatric disorders than others. However, everybody uses some of the defense mechanisms in a habitual way, and, as noted earlier, this habitual pattern of defensive maneuvers forms the basis of "character." The term "personality" involves all the behavioral characteristics of a person, including the "character." These terms, however, are often used synonymously (see Chapter 17 for further discussion).

The defense mechanisms are also closely related to the concept of "coping styles." The latter refers to a person's habitual ways of dealing with clearly defined, conscious anxiety-provoking situations, such as hospitalization or impending surgery. Many defense mechanisms, alone or in combination, enter into the development of the coping styles (see Chapter 14).

Summary

Help-seeking behavior often occurs in the presence of anxiety. Human beings have a tendency to avoid feeling anxiety by the automatic (unconscious) use of certain psychological processes called "defense mechanisms." The function of the defense mechanisms is to reduce or prevent the generation of anxiety, a common pathway to distress, by keeping stimuli that signal or could lead to danger situations out of conscious awareness or by inducing a sense of mastery.

The processes can be classified, on the basis of the functional subsystems primarily affected, into (1) those that affect input (perception), (2) those that affect internal processing (cognitive and affective processes), (3) those that affect output (action), and (4) those that affect all subsystems evenly by affecting the "decider" or "executive" function of the personality system.

Defense mechanisms commonly used in medical settings include denial, displacement, projection, introjection, constriction of awareness, repression, rationalization, intellectualization, acting out, counterphobic

maneuvers, undoing, sublimation, nonspecific activity, regression, and identification.

Implications

For the Patient

The defense mechanisms that a patient uses exert a major influence on when and whether he seeks help for a particular symptom, how he will respond emotionally to the medical treatment and to information provided by the health-care team, and how he will cope with illness, procedures, and recovery process. For example, excessive use of denial and repression is likely to result in delayed help-seeking behavior, non-compliance with the doctor's instructions, and frequent conflict with the health-care personnel. On the other hand, the ability to deny anxiety to a limited degree and under certain circumstances, for example, in the acute phase of myocardial infarction, may exert a salutary effect.

For the Physician

In Terms of Understanding the Patient. An assessment of the habitual defense mechanisms used by the patient is essential for understanding the patient and for planning treatment approaches. The recognition that defense mechanisms are geared to ward off anxiety (and thus distress) should help the physician in learning how to cope with them. Defenses should not be attacked frontally—this will only cause excessive mobilization of anxiety and distress. The physician in vignette 2 would have understood his patient better if he had been acquainted with the concept of denial, and he might even have wondered whether the excessive denial on the patient's part could have been related to a "priming experience," such as exposure to someone else who had a lump or ulceration on the breast. Regression is a common phenomenon in the hospital, where a certain amount of actual infantilization takes place. Recognition of this phenomenon will help the physician understand why some patients may manifest infantile or childlike behavior in the hospital and also why some patients develop irrational feelings concerning members of the health-care team, including the doctors and nurses. This understanding is important if the unrealistic or irrational feelings expressed by patients are not to be "taken personally" but rather to be understood as part of the process of being ill and as being directed

not at the current health-care personnel but at important figures in the patient's past. Habitual defense mechanisms may serve as guides to *strategies in clinical management*. For example, patients who show a tendency to intellectualize may respond well to the provision of reading material concerning the illness or proposed procedure; patients who tend to show denial may respond better to blanket reassurances than to detailed information when anxiety reduction is indicatd; patients who use repression excessively may not remember the doctor's instructions or explanations; patients accustomed to using activity as a major defense mechanism may find it unbearable to be confined in the hospital without activity, and for such patients, physicial therapy may be indicated as well as early ambulation whenever possible.

In Terms of Understanding Self. In the doctor–patient relationship, the defense mechanisms used by the physician also play a major role. In dealing with patients, it is helpful for physicians to be aware of their own habitual defense mechanisms and to understand how they may interact with the patient's defensive style. One clue that may alert us to the fact that defense mechanisms may be "overworking" (i.e., that we are really quite anxious) is to feel a strong overwhelming conviction without being able to explicate the reasons for it. For example, a physician who feels strongly (and stubbornly) that radical surgery should be performed on a patient when, in fact, the objective indications for the operation are equivocal may be using the mechanisms of denial and activity as a way of reducing feelings of helplessness, impotence, and anxiety (vignette 5). Self-awareness could help him to refrain from insisting on an unnecessary, and perhaps dangerous, surgical procedure. Learning to *wait*, to *observe*, and to *listen*—rather than feeling compelled to *do* something (anything as long as it is active)—is one of the hardest "skills" physicians have to acquire. The social expectation that physicians will not exploit patients for their own needs includes an implicit dictum against treatments that serve mainly as ways of reducing the physician's anxiety.

For the Community and the Health-Care System

Why do millions of people still smoke cigarettes despite the quite visible and rational warnings by the surgeon general and the medical profession? Surely, denial, counterphobic maneuvers, rationalization, and other defenses must play a role in the otherwise inscrutable smoking behavior of many individuals.

Refinement and extrapolation of principles based on understanding of psychological defenses in individual behavior in order to design programs of group and public health education, health-care delivery, and preventive medicine constitute a major challenge in our society.

Recommended Reading

Freud A: *The Ego and the Mechanisms of Defense*. New York, International Universities Press, 1966. A classic monograph describing the phenomenology and development of defense mechanisms as an "ego function." Easy to read, succinct, and full of actual illustrations of defense mechanisms in action.

Hackett TP, Cassem NH, Wishnie HA: The coronary care unit: An appraisal of its psychologic hazards. *N Engl J Med* **279**:1365–1370, 1968. The psychological environment of the coronary care unit. Patients who used major denial had a better hospital course than those who were not using much denial.

Meissner WW, Mack JE, Semrad EV: Structure of the psychic apparatus, in Freedman AM, Kaplan HI, Sadock BJ (eds): *Comprehensive Textbook of Psychiatry*, ed 2. Baltimore, Williams & Wilkins, 1975, Vol 1, Chapter 8.1 (Classical psychoanalysis), pp 528–540. An up-to-date account of the psychoanalytic concept of the psychic apparatus: the id, ego, and superego. Defense mechanisms are discussed in terms of the development of the ego and functions of the ego. The ego is a hypothetical construct of the mind representing a "coherent organization of functions," the task of which is to avoid displeasure through the regulation of the "instinctual drives" and the demands of external reality. This chapter contains a classification of defense mechanisms into narcissistic, immature, neurotic, and mature defenses.

Sachar EJ, Kanter SS, Buie D, *et al.*: Psychoendocrinology of ego disintegration. *Am J Psychiatry* **126**:1067–1078, 1970. In a psychoendocrinological study of four acute schizophrenic males, the authors found great activation of the adrenocortical hormones when the defense mechanisms were failing. When defense mechanisms were functioning, even if they were pathological defenses such as projection and delusion formation, the hormonal activity was not increased.

Reiser M: *Mind, Brain, Body: Toward a Convergence of Psychoanalysis and Neurobiology*. New York, Basic Books, 1984. Chapters 7 and 11 deal with defenses as they are conceptualized in psychoanalytic theory.

References

Freud A: *The Ego and the Mechanisms of Defense*. New York, International Universities, 1966.

Hackett TP, Cassem NH, Wishnie HA: The coronary care unit: An appraisal of its psychologic hazards. *N Engl J Med* **279**:1365–1370, 1968.

Leigh H, Reiser MF: A general systems taxonomy for psychological defense mechanisms. *J Psychosom Res* **26**:77–81, 1982.

Lorenz K: *On Aggression*. New York, Bantam Books, 1966.

Meissner WW, Mack JE, Semrad EV: Classical psychoanalysis, in Freedman AM, Kaplan HI, Sadock BJ (eds): *Comprehensive Textbook of Psychiatry, II*, ed 2. Baltimore, Williams & Wilkins, 1975, Vol 1, pp. 482–566.

Sachar EJ, Kanter SS, Buie D, *et al.*: Psychoendocrinology of ego disintegration. *Am J Psychiatry* **126:**1067–1078, 1970.

Tinbergen N: *Social Behavior in Animals*. New York, John Wiley & Sons, 1953.

Weiss E, English OS: *Psychosomatic Medicine: A Clinical Study of Psychophysiologic Reactions*. Philadelphia, WB Saunders, 1957, p 214.

CHAPTER 6

Depression, Mania, and Suicide: Affective Disorders

1. A 43-year-old married woman, the mother of two children, ages 21 and 18, consulted her physician at the urging of her husband. She complained of fatigue, headaches, and vague aches and pains in many parts of her body. She had developed progressive difficulty in sleeping over the last month or so, with the onset of the vague symptoms. She would often wake up at 3:00 A.M. and think about how empty and meaningless life was. She would feel sad and burst into tears, but lately she felt that her tears had dried up. She neglected her personal appearance and seemed to feel extremely guilty about "not being a good mother, or a wife, or even a housemaid." Her sexual activity decreased to zero. In the last month, she lost 15 pounds. When urged to eat, she would say that food had no flavor, like cardboard. She was also convinced that she had a fatal disease, "Cancer of an internal organ, maybe the stomach." She would not see a doctor, however, because she "might as well be dead, being such a no-good person . . . anyway, the cancer has spread all over me, there is nothing they can do."

2. While making rounds in the morning, a 28-year-old medical resident was noticed to be loud, inappropriate, and irritable. For example, he cracked intimate jokes with the patients in full view of other patients and staff, asked questions of the attending physician in a condescending manner, and proposed radical, potentially dangerous, and unnecessary procedures for the patients. When the proposals were not accepted, he became irritable and accused the others of being incompetent, again in front of patients and staff. When he further stated that he now possessed a secret method of healing that would cure any disease without the need for any diagnostic tests, his colleagues decided that he needed emergency psychiatric help.

99

On admission, the patient was talking incessantly in a loud voice. He stated to the psychiatrist that he came to the psychiatric unit so that he could teach the staff how to cure schizophrenia using his secret method. He stated that he would also give electroshock treatments to all the patients on the unit with his bare hands, which were "charged with electricity." He also asked for a dictaphone in his room because he was in the middle of writing five articles at once, including his proposal to the hospital on how to make more money, a "Nobel-prize-winning article on new, secret medical therapeutics for all diseases," and an autobiography. During the first day of hospitalization, he attempted to date all the nurses he saw on the unit.

3. A 64-year-old successful executive of a major company became depressed and despondent after his wife's death about three years ago. His work efficiency decreased, and he began drinking excessively. He often talked about life being meaningless and about how empty success was. The only things in life that really gave pleasure, according to him, were things of beauty, like the collection of original paintings he had in his study, which was his lifetime hobby. For about the last two years, however, he had not purchased any more paintings, which was unusual for him. He had seemed to be especially brooding and generally irritable for about the last three months and was absent from work for several days, which was, again, unusual. Since about a week ago, his spirits seemed to have lifted considerably. He tidied up his office, and he seemed to socialize more, calling up his friends and giving them as gifts his prized painting collection. He even gave his secretary an expensive original painting that was hanging in his office. Last night, he shot himself in the head.

4. A 50-year-old married woman was admitted to the psychiatric unit because of tearfulness, suicidal thoughts, and fatigue. For approximately four or five weeks prior to admission, she had become progressively depressed, with less interest in such activities as watching television, bridge, and reading—all of which she had enjoyed doing in the past. She developed a feeling of impending doom—as though everything would crumble about her. She lost all interest in sex, and food had no taste or flavor. She lost ten pounds during this period. She had difficulty in falling asleep and also in staying asleep through the night. She felt guilty about minor things and felt that she was not a worthwhile person to be allowed to live. She withdrew from her family and friends.

On admission to the psychiatric unit, she was treated with antidepressant medications with equivocal results. She continued to lose weight. A thorough medical workup in the hospital revealed that the patient had a carcinoma of the pancreas. Her depression abated following surgical removal of the diseased pancreas.

5. A few days ago, a 25-year-old medical student began to feel sad, blue, and dejected. He felt like crying from time to time and seemed to feel less pleasure in usual activities. During conversation with a friend, it suddenly

occurred to him that the anniversary of his mother's death had just passed. He had been so busy with schoolwork that this had completely escaped his mind. With this recognition, he understood the reason for his sadness.

Affect, Mood, Depression, and Mania

Like anxiety, depression in its milder form is a ubiquitous experience. We feel sad in the face of loss and sometimes helpless and inadequate when we realize that we are not up to completing the tasks required of us. On the other hand, when we have accomplished a task well, we feel joyous, and even elated. In the company of a good friend, and perhaps with a glass of good wine, we feel content and euphoric.

The emotional feeling tone of an individual, such as sadness, joy, depression, and elation, is called an *affect* (see Chapter 4 for further discussion of affect or emotion). When the affect is prolonged and colors the whole emotional life of the person, it is called a *mood*. Thus, a person may be in a blue mood, an elated mood, or a depressed mood.

Very simply, one can consider good and bad moods. On the bad side, such descriptions as feeling down, blue, sad, miserable, depressed, down in the dumps, are used, while, on the other hand, one can think of being happy, high, joyous, euphoric, elated, exulted, ecstatic, and manic. While all of us experience varying gradations of these moods, the extremes of moods, the depressive syndrome and mania, are not experienced except in pathological conditions. Thus, they are collectively called affective or mood disorders.

Phenomenology of Depression and the Depressive and Manic Syndromes

The subjective feeling of sadness is the most common experience of depression in everyday life. This feeling is usually experienced after suffering a loss or failure—the loss of a loved one, possession, or prestige. The loss may be purely imaginary, and even the anticipation of a loss may cause sadness. Depression, in this sense, is closely related to grief, the specific emotional suffering related to loss, and separation, a common antecedent to grief. Separation and grieving processes will be discussed in a later section of this chapter.

The term *depression* is a confusing one, because it is used to denote the whole gamut of unpleasant moods, and exactly what degree of severity is referred to is not clear from the term itself. When psychiatrists

use the term "depression," it almost invariably means the *depressive syndrome*, which is very different, indeed, from feelings of sadness associated with, say, the moving away of a good friend. This is somewhat similar to the difference in the use of the term *hypertension* between the lay public and the medical profession. When a patient talks about having "hypertension," he may mean (1) that he has high blood pressure, (2) that he tends to become tense and nervous, or (3) both. Of course, there are precise blood pressure levels and procedures for the diagnosis of hypertension in medicine. What, then, is meant by depression when the term is used in a medical context?

The *depressive syndrome* [as in major affective disorder according to the *Diagnostic and Statistical Manual of Mental Disorders (DSM III) of the American Psychiatric Association, 1980] is characterized by a period of either depressive mood* or a *pervasive loss of interest or pleasure*. The patient, as in vignette 1, often feels *sad, hopeless, helpless,* and *empty. Guilt* feelings are prominent, and there is a loss of *self-esteem*. Feeling discouraged and "down in the dumps" is common. The patient typically *withdraws* from family and friends, and activities and hobbies that used to give him pleasure no longer interest him. There is usually some *sleep disturbance*, usually early-morning awakening (EMA), but middle-of-the-night awakening (MNA) and difficulty in falling asleep (DFA), are not uncommon, especially if anxiety is also prominent. *Loss of appetite* is quite common, with concomitant weight loss, although in some patients, there may be an increase in eating with weight gain. The patients often show *psychomotor agitation* or *retardation*. In agitation, pulling out hair, pacing, wringing hands, inability to sit still, incessant talking, and shaking of hands and feet often occur. Psychomotor retardation is characterized by slowing of speech, slowed body movements, or even muteness.

In the depressive syndrome, patients often manifest *cognitive disturbances* (disturbances in thinking). This includes inability to concentrate, indecisiveness, and generally slowed thinking processes. Often, patients feel that they do not have enough energy to think out a simple problem. They feel tired, fatigued, and exhausted in the absence of physical exhaustion. They may experience vague pains, aches, and discomfort, without any physical basis; headaches, toothaches, backaches, and muscle aches are especially common.

Patients often suffer from feelings of *inadequacy, worthlessness,* and sometimes completely unrealistic *low self-esteem*. The smallest task may appear impossible or monumental. There may be excessive guilt feelings concerning current or past failings, most of them minor, or even delusional conviction of sinfulness or responsibility for some untoward tragic event.

Suicidal ideas are frequent and may take the form of fears of dying, the belief that the person himself or others would be better off if he were dead, or suicidal desires or plans. Often, there is a *diurnal variation*—the symptoms are worse on waking up in the morning and improve slightly as the day progresses.

When the symptoms are mild, temporary improvement often occurs in the presence of positive environmental stimuli. In several cases, the syndrome is not affected by environmental change to any extent. (Specific diagnostic criteria for the depressive syndrome can be found in the section on Evaluation of Depression.)

At the opposite pole of the depressive syndrome in mood is the *manic syndrome*. Just as sadness and grief are experienced by most people from time to time, so do pleasurable moods of euphoria and elation, short of mania or hypomania, fall within the normal range of mood. In euphoria, there is a positive feeling of emotional and physical well-being. In elation, there is a definite feeling of joy with increase in self-confidence, motor activity, and energy level. These states can be induced by drugs such as alcohol, narcotics, and amphetamines.

On the other hand, mania and hypomania (which is a somewhat less severe form of mania), like the depressive syndrome, form a syndrome with definite features and signs. The essential feature of the *manic syndrome* is a distinct period when the predominant mood is elevated, expansive, or irritable and is associated with other symptoms of the manic syndrome. They include hyperactivity, excessive involvement in indiscreet and foolish activities without recognition of the high potential for painful consequences, pressure of speech, flight of ideas, inflated self-esteem, decreased need for sleep, and distractability (DSM III, 1980).

The patient may describe the *elevated mood* as being euphoric, unusually good, or high. The good mood may have an infectious quality, so that the physician or others in contact with the patient may find themselves feeling expansive and often humorous. The patient may show *indiscriminate enthusiasm* in relating to people or planning things, so that he may start a dozen projects at once, call up distant relatives and bare acquaintances all over the globe, and go on a buying spree. On the other hand, the mood may be characterized by *irritability* rather than joyfulness, especially when the patient's expansiveness is thwarted. The patient then becomes touchy and domineering. The *hyperactivity* is often generalized, including participation in multiple activities that may be sexual, occupational, political, or religious. The patients often have *poor judgment*, and the activities are disorganized, flamboyant, and bizarre (see vignette 2).

Manic speech is usually *loud, rapid,* and *difficult to understand.* It is often full of jokes and puns and is theatrical, with singing and rhetorical mannerisms. In the irritable mood, there may be hostile comments and angry outbursts. Abrupt changes from topic to topic based on understandable associations and distracting stimuli often occur (flight of ideas). When severe, the speech may be incoherent. Distractibility is usually present.

Self-esteem is usually inflated, with unrealistic and uncritical self-confidence and grandiosity. For example, the patient may give advice on matters about which he has no expert knowledge whatever—such as how to perform an operation or how to run the Federal government. Grandiose delusions may occur, such as, "I have a special hot line to God."

When a person has episodes of *both* depression and mania, this is called manic–depression or *bipolar illness.*

Separation, Bereavement, and Grief

Phenomenology

The phenomenology of acute grief following separation and bereavement has much in common with that of depression. Lindemann (1944) described the symptomatology of acute grief in his classic paper based on bereaved persons, including those who lost their relatives in the Coconut Grove fire. The symptoms of normal grief include sensations of *somatic distress* occurring in waves lasting from 20 minutes to an hour at a time, a feeling of tightness in the throat, shortness of breath, frequent sighing, an empty feeling in the stomach, lack of muscular power, and an intense subjective distress described as tension or mental pain. These waves of distress can be precipitated by mere mention of the deceased. There is often a loss of appetite.

In addition to the somatic distress, grieving persons have an *intense preoccupation with the image of the deceased*—to the point where they may look for the deceased person in a crowd or almost feel the presence of the deceased in the room. *Guilt feelings* are also common. The bereaved searches the time before the death for evidence of failure to do the right things. Self-accusation of negligence and exaggeration of minor omissions are common. The bereaved tends to become *irritable and hostile* to friends and relatives who are making a special effort to show sympathy.

There is often a change in the *patterns of conduct.* The bereaved finds it difficult to initiate and maintain organized patterns of activity, simply

going through the motions of carrying out normal activity. The bereaved is surprised to discover how large a part of his customary activity was done in some meaningful relationship to the deceased and has now lost its significance. The bereaved feels restless and unable to sit still. He tends to move about in an aimless fashion, as though in search of something to do.

In addition to the five symptoms described by Lindemann to be pathognomonic for acute grief, some bereaved persons develop an *identification* with the deceased, manifested by the taking on of the manner or speech or gait of the deceased or even the development of symptoms of the deceased's last illness in the absence of evidence of the disease.

Course

The *course* of bereavement was studied extensively by Parkes (1972). He described *three phases* of bereavement: the phase of numbness, followed by the phase of pining, followed in turn by the phase of depression.

The first phase described by Parkes, *numbness*, may last for a few hours up to a few days. This phase may be punctuated by moments of panic and distress, including the somatic distress described above. The second phase *pining*, is characterized by *anxious searching*, with preoccupation with the thoughts and images of the deceased. This phase includes the somatic distress described by Lindemann, as well as feelings of anger and guilt. Guilt alternates with and eventually gives way to feelings of depression. The anxious searching phase peaks in two to four weeks and leads to the phase of *depression and despair*. This period is also characterized by apathy and aimlessness, with loss of patterns of control.

The course of normal acute grief is approximately four to eight weeks (Huston, 1975), but a substantial proportion of the bereaved continue to report distress up to one or two years after the death of a spouse.

Pathological Grief Reactions

Lindemann described two types of pathological grief reactions, the *delayed* grief reaction and the *distorted* grief reaction.

The delayed grief reaction is characterized by a postponement of the grieving process. When a person is in the middle of performing important tasks, or when it is necessary to maintain the morale of others, there may be no overt sign of grief for weeks, months, or even years

after the loss. The full-blown picture of acute grief may be precipitated, however, by mention of the deceased or by a situation that reminds the bereaved of the loss. Such precipitating events may be the anniversary of the loss or the individual's attaining the same age as the deceased's at the time of death.

Distorted reactions may occur during any phase of grief or as a delayed reaction. They include overactivity, no sense of loss, the acquisition of symptoms of the last illness of the deceased, severe alteration in relationship to friends and relatives, excessive hostility against specific persons, lasting loss of initiative, self-destructive activities, and the development of the full-blown depressive syndrome.

Separation in Children

Spitz (1942) described the phenomenon of anaclitic depression in infants in the latter half of the first year on separation from their mothers. This syndrome consisted of crying, apprehension, withdrawal, psychomotor slowing, dejection, stupor, insomnia, anorexia, weight loss, and gross retardation in growth and development. "Anaclitic" means dependence, and the depressive syndrome was presumed to be due to the absence of the mother, a figure to depend on.

Bowlby studied the effects of separation from mothers of somewhat older children (Robertson and Bowlby, 1952; Bowlby, 1960) and described three phases. The first is a *protest* phase, characterized by a frantic searching for the mother with restlessness and tearfulness, followed by a *despair* phase in which the child withdraws from others and appears apathetic. Parkes's later staging of the grief reaction in adults, discussed above, has much similarity to these stages described by Bowlby. When the child is reunited with the mother, some children ignore the mother or actively reject the mother for a period. This was called the *detachment* stage by Bowlby.

Although separation in infants results in a syndrome similar to grief in adults, a causal relationship between early separation and later vulnerability to depression is as yet unproven (Akiskal and McKinney, 1975). Further, there is evidence that substitute mothering can, to a large extent, alleviate whatever damage may occur following separation from the mother.

Separation in Infant Monkeys

The separation reaction described by Bowlby occurs in nonhuman primates as well. Separation of seven-month-old infant monkeys from
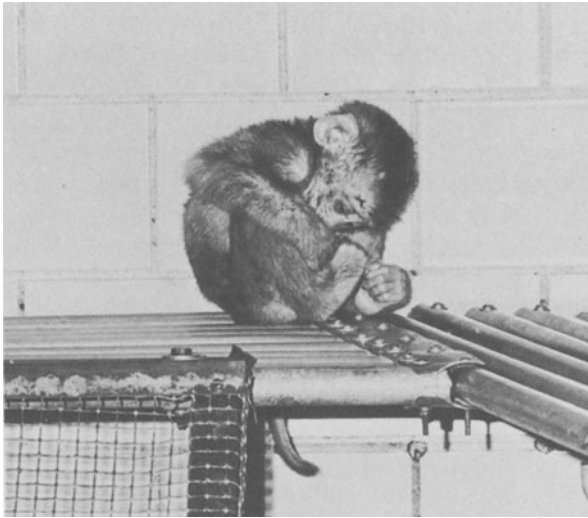
Figure 9. Depressed captive pigtail infant monkey showing characteristic posture of despair including head between legs. (From Kaufman and Rosenblum, 1967. Courtesy of the authors. Reproduced with permission.)

the mother resulted in initial *violent protest* with crying and searching behavior, followed by a *despair* phase with decreased activity, withdrawn behavior, and occasional crying (Figure 9) (Seay and Harlow, 1965; Kaufman and Rosenblum, 1967).

Harlow and Harlow demonstrated the effects of isolation from mothers and peers from birth in rhesus monkeys (Harlow and Harlow, 1962, 1966a,b). The Harlow's experiments showed that tactile sensation was an important element of attachment behavior. Some monkeys were given inanimate "surrogate mothers" made of terrycloth or wire. The infant monkeys would choose "terrycloth mothers," which were mechanical devices with terrycloth, over "wire mothers," which were equipped with a feeding apparatus but without any soft terrycloth. Monkeys that were socially isolated in this way early in life developed difficulties in later life, especially in socialization and sexual activity. These difficulties were alleviated in large measure, however, if peers were present during infancy and childhood.

Function of Depression

The phenomenology of mild forms of depression is indistinguishable from that of grief reactions to losses of varying degrees. Thus,

depression as an affect can be conceptualized as a response to loss or separation, actual or symbolic, that may have an adaptive function. The depressive syndrome, in this light, may be seen as a dysregulation syndrome of the depressive affect.

The feeling of depression signals that a loss or separation has occurred or is in the making. In cases of threatened loss, anxiety will occur together with depressive affect and prepare the individual to deal with the loss (including, in certain instances, avoidance of the loss).

The protest phase of the separation reaction in infants and young monkeys serves the funtion of drawing the attention of the mother or other members of the social group to the separated infant. The crying of the baby may suddenly remind the mother that it has crawled into another room while she was not watching. *Social support* is often mobilized for an infant who is lost or separated from its mother.

When the separation is prolonged, the infant proceeds into the stage of despair, with reduction in activity and behavioral withdrawal. This state may serve the *conservation* function previously described—in the face of adversity, the organism might withdraw and tend to conserve energy and strength by reduction of activity, so that there might be strength at a later date. Thus, the behavioral patterns of depression may have biologically and socially adaptive functions.

The affect of depression related to helplessness in childhood and threat of loss may play an important role in the *socialization* and *learning* of infants and children. Somewhat like anxiety, which has an optimal level for performance, certain amounts of depressive experience may help the learning process.

Significance of Bereavement, Depression, and Affective Disorders

Many recent epidemiological studies indicate that the experience of bereavement or grief is associated with an increased risk of becoming ill or, in fact, dying. Grief was accepted as a cause of death in prescientific medicine, as evidenced by Table 2. The pathophysiological mechanism by which the experience of bereavement, grief, or, for that matter, depression leads to morbidity and mortality by such diseases as coronary disease and cancer remains unclear (see Chapter 4).

The i*ncreased mortality rate* in the bereaved seems to be 2–7 times that of the expected rate in age- and sex-matched nonbereaved persons during the first year of bereavement. The risk of mortality is greater for men than for women for all ages. The major effect of bereavement in

Table 2. Dr. Heberden's Classification of the Causes of Death in London during the Year 1657[a]

Flox and smailpox	839
Found dead in the streets, etc.	9
French pox	25
Gout	8
Griefe	10
Griping and plague in the guts	446
Hang'd and made away 'emselves	24

[a] As quoted by Parkes (1972), Chapter 2. Copyright 1972 by Tavistock Publications, Ltd. Reproduced with permission from the author and Associated Book Publishers, Ltd., London.

terms of mortality seems to occur in the first two years after the death of a spouse and, in men, almost exclusively in the first six months (Jacobs and Ostfeld, 1978). The cause of death of men is accounted for by tuberculosis, influenza and pneumonia, cirrhosis of the liver and alcoholism, suicides and accidents, and heart disease. For women, elevated mortality occurs from tuberculosis, cirrhosis of the liver and alcoholism, heart disease, and cancer.

In addition to increasing the risk of morbidity and mortality from *medical diseases* such as tuberculosis, heart disease, and cancer, *bereavement precipitates a depressive syndrome* in a significant number of persons. Clayton and her associates studied 109 randomly selected bereaved subjects (Clayton *et al.*, 1972; Bornstein *et al.*, 1973). At one month after bereavement, 35% of the sample were definitely or probably depressed; at 13 months, 17%. Thirty-nine percent had a depressive syndrome at some time during the period.

Depression unassociated with bereavement has also been shown to be associated with increased morbidity and mortality. For example, in one study, subjects who had acute myocardial infarction and died had higher depression scores on the Minnesota Multiphasic Personality Inventory (MMPI) than those who had acute myocardial infarction and survived (Lebovitz *et al.*, 1967).

Preoperative depression is known to be a predictor of increased postoperative behavioral problems and mortality in cardiac surgery patients (Kennedy and Bakst, 1966; Reiser and Bakst, 1975). On the basis of studies of illness-onset situations, Schmale and Engel postulate that a feeling of helplessness and hopelessness (the *giving up–given up complex*)

may provide a "permissive" setting for the development of a medical disease (Schmale, 1972). This permissive setting, which has many features of depression, was found to occur in patients who had leukemia, lymphoma, diabetic ketosis, cervical carcinoma, bronchial asthma, and other diseases.

In one study, the mortality during the first decade after hospitalization of those who had been admitted to a psychiatric service for depression and mania was more than two and three times, respectively, the mortality rate of control subjects who had been admitted for surgical reasons (Tsuang *et al.*, 1979)

Epidemiology of Affective Disorders

Just how common are depression and mania? As we discussed, transient depressive or euphoric mood is ubiquitous. What about the depressive and manic syndromes?

In a comprehensive review, Boyd and Weissman (1981) report that the lifetime prevalence of affective disorders in general is 17–20%, and a one-year prevalence is 5–8%. Affective disorders, then, are the most common psychiatric disorders. In the United States and Europe, an estimated 8–11% of male adults and about 18–23% of female adults have had a depressive syndrome at some time (DSM III). Approximately 6% of the females and 3% of the males have had depression severe enough to require hospitalization. Thus, the sex ratio for depressive syndrome for females and males is 2:1.

As for the bipolar illness, it is estimated that about 0.4–1.2% of the adult population have had at least one manic episode. Unlike the depressive syndrome, bipolar disorder is equally common among men and women.

In an extensive review, Hirschfeld and Cross (1982) described the epidemiological factors that seem to increase the prevalence of affective disorders, which include the full-blown and incomplete depressive syndromes. The incomplete depressive syndrome, which they called "depressive symptoms," was characterized by the presence of dysphoria and was often accompanied by other associated features of the depressive syndrome but did not quite meet the criteria for the complete syndrome (see the criteria in Table 3). Their findings are summarized below.

Sex: Female/male ratio of 2:1 for both complete and incomplete unipolar depressive syndrome. No sex difference for bipolar disorder.

Age: There seems to be a higher prevalence for both complete and incomplete unipolar depressive syndrome in the younger adult age groups (18–40 years of age) than in older groups. Typical age of onset of unipolar depression is in the late 30s, as opposed to the late 20s in bipolar depression or mania.

Marital Status: Unipolar depression (both complete and incomplete syndrome) is more common among separated and divorced persons, and married groups have the lowest rates. There is no marital-status difference in the prevalence of bipolar disorders.

Residence: Although there is some suggestion that depressive symptomatology is greater in urban areas, there is no conclusive evidence that residence affects the prevalence of affective disorders.

Race: There is no evidence for race differences in affective disorders.

Religion: There is no difference in prevalence of unipolar depression among different religions. However, bipolar disorder has been reported to be particularly common among Ashkenazi Jews and among Hutterites in the northern Midwest.

Social Class: Unipolar depression seems to be more common among persons of lower socioeconomic status. On the other hand, an unusually high rate of unipolar (38–50%) depression has been reported among professional women (Welner *et al.*, 1979). As for bipolar disorder, the rate seems to increase with increasing socioeconomic status, with the highest rates among professionals.

Life Changes: As would be expected, unipolar depression is more common among persons who have had stressful life changes, particularly after bereavement.

Presence of Other Medical and Psychiatric Disorder: Depression is often associated with a medical disease or another psychiatric disorder (e.g., substance abuse).

Significant Others and Family Resources: Significant others and supportive family members seem to provide a protective effect against unipolar depression.

Personality Characteristics: Not surprisingly, the "personality profiles" of patients with unipolar depression show increased introversion,

neuroticism, obsessionality, dependency, and low self-esteem. In bipolar disorder, however, the personality profiles are mostly normal except for higher levels of obsessionality.

Brain Mechanisms of Affective Disorders

Certain areas in the brain are known to be associated with the experience of pleasure and displeasure. Among others, electrical stimulation of the medial forebrain bundle is considered to be pleasurable and reinforcing to the animal, while electrical stimulation of the periventricular system is considered to be unpleasant (see Figures 10–12). It is reasonable to suppose, then, that these structures are somehow involved in the experience of mania and depression (Akiskal and McKinney, 1975). The biogenic amines norepinephrine and serotonin are both involved in the modulation of the function of the medial forebrain bundle and the periventricular system.

Biogenic Amines

Much information on the brain mechanisms of affect is based on the study of patients with the fully developed depressive and manic syndromes and their pharmacological treatment. The two classes of antidepressants, tricyclics and monoamine oxidase inhibitors (MAOIs), increase the functional levels of biogenic amines in the brain. The biogenic amines thought to be involved in the brain are the catecholamines (mainly norepinephrine and dopamine) and the idoleamine serotonin. Reserpine, used in the treatment of hypertension, depletes biogenic amines (both catecholamines and indoleamines) in the neurons and can cause the depressive syndrome in 10–15% of patients receiving it. The catecholamine hypothesis of depressive disorders (Schildkraut and Kety, 1967) postulated that in mania, there is an increased functional level of norepinephrine in the noradrenergic synapses in the brain, whereas in depression, there is a decreased functional level of norepinephrine in the brain. The cell bodies of the norepinephrine-containing neurons are in the brain stem, especially in the locus ceruleus in the pons [see Figures 6 (Chapter 4) and 10]. The axons ascend in the medial forebrain bundle to supply the hypothalamus and areas of the limbic system as well as the entire cerebral cortex (Sweeney and Maas, 1978).

The pharmacological evidence may be equally strong for a *serotonin* theory, which is especially widely accepted in Great Britain. The cell
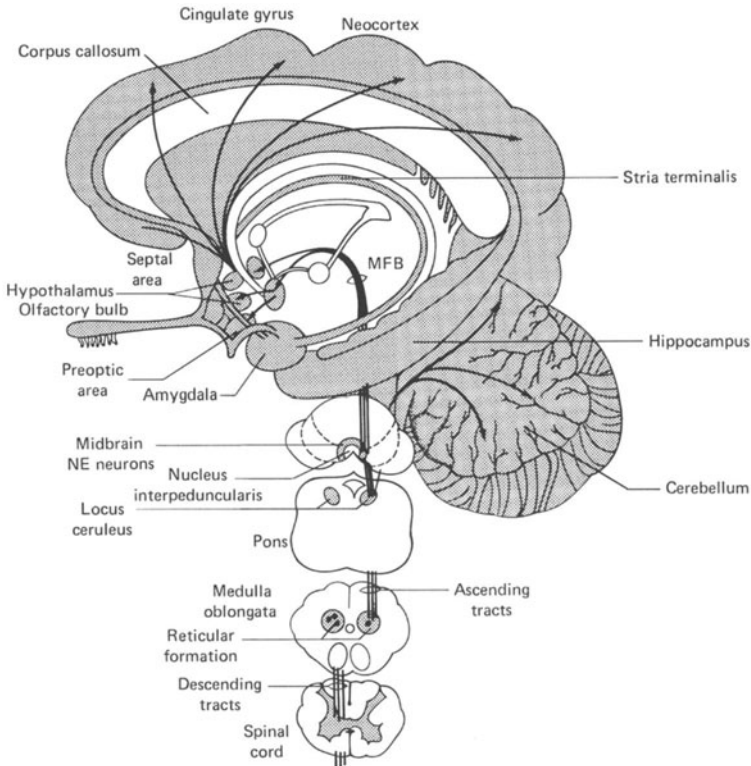
Figure 10. Noradrenergic (NE) systems. Sagittal projection of NE pathways arising from cell groups in medulla oblongata and pons. The NE tracts are both descending and ascending. Descending fibers arise from cell bodies in the medulla oblongata and innervate the gray matter of the spinal cord. Ascending neurons are derived primarily from cell bodies in the medulla oblongata (reticular formation) and pons (locus ceruleus) and enter the medial forebrain bundle (MFB). These fibers innervate the hypothalamus, stria terminalis, preoptic area, septal area, amygdaloid cortex, cingulate gyrus, and neocortex. The cerebellum is also innervated by NE neurons from the medulla and pons. A smaller number of NE neurons arise from an area surrounding the nucleus interpeduncularis. (From *Neuropsychopharmacology, 1: The Monamine Systems*, 1976. Reproduced with permission from Roche Laboratories, Nutley, New Jersey.)

bodies of the serotonergic neurons are in the median raphe in the mesencephalon and upper pons (Figure 12). Their axons ascend in the medial forebrain bundle, giving off terminals to various parts of the brain, including the hypothalamic areas and the cerebral cortex. There is evidence that depressed patients have low levels of 5-hydroxyindoleacetic acid, a serotonin metabolite, in the cerebrospinal fluid (Bowers *et al.*,
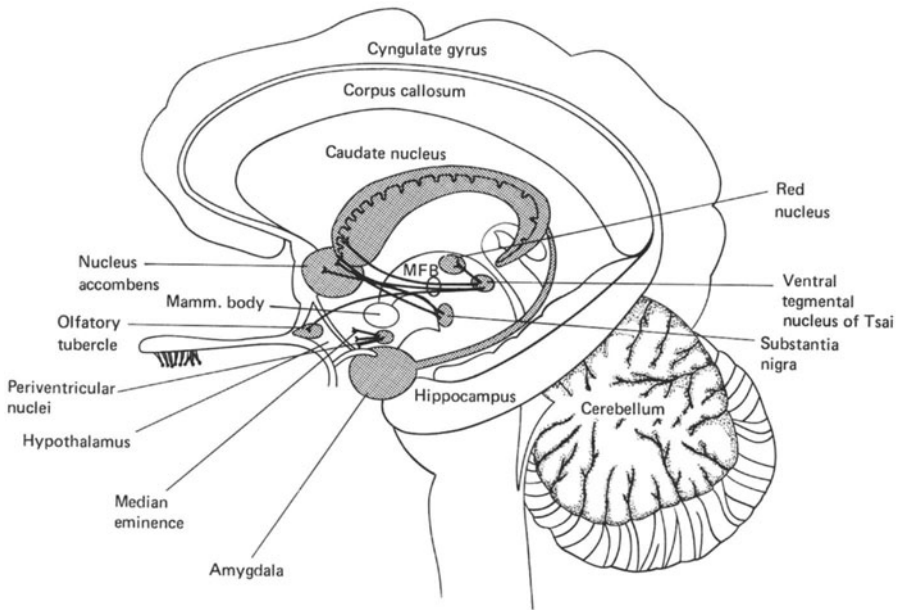
Figure 11. Dopaminergic (DA) systems. Sagittal projection of the DA pathways arising from cell groups in the midbrain. The DA neurons form at least three definable systems. One pathway consists of the nigrostriatal tract, which arises from cell bodies in the substantia nigra and terminates on small interneurons of the neostriatum (caudate nucleus and putamen). (Note: Putamen cannot be seen from sagittal view.) A second major pathway arises from the ventral tegmental nucleus of Tsai (located about the cranial portion of the nucleus interpeduncularis), enters the medial forebrain bundle (MFB), and terminates anterior to the caudate nucleus in the nucleus accumbens (nuclei nervi vestibulocochlearis), olfactory tubercle, and red nucleus of the stria terminalis (the limbic striatum). A third pathway involves DA cell bodies from the arcuate and anterior periventricular nuclei that terminate in the median eminence of the hypothalamus and appear to have a role in regulating gonadotropin secretion. (From *Neuropsychopharmacology, 1: The Monoamine Systems*, 1976, with modification. Reproduced with permission from Roche Laboratories, Nutley, New Jersey.)

1970; Åsberg *et al.*, 1973). Low levels of serotonin and 5-hydroxyindoleacetic acid were found in the autopsied brains of suicidal patients (Davis, 1977). L-Tryptophan is the precursor of serotonin in the brain, and the brain level of serotonin seems to be determined by the blood level of L-tryptophan. There is evidence that L-tryptophan may improve the symptoms of both mania and depression in some patients (Davis, 1977). Prange *et al.* (1974) postulated a *permissive theory* of affective disorders that states that in all affective disorders, there is an abnormally *low level of functional serotonin* in the brain; in addition, in the case of
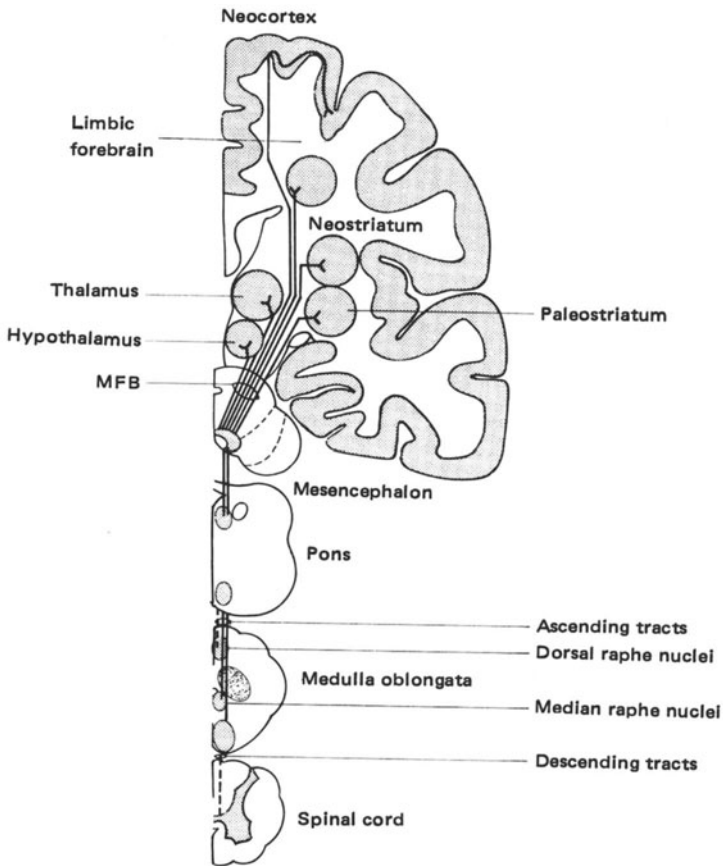
Figure 12. Serotonergic (5-HT) systems. Diagrammatic representation of 5-HT pathways arising in the mesencephalon, entering the medial forebrain bundle (MFB), and innervating rostral nuclei along with NE neurons. The 5-HT neurons tend to have a distribution similar to the NE system. Descending pathways innervate the gray matter of the spinal cord; cell bodies are located primarily in the lower raphe nuclei of the pons. Ascending neurons arise from cell bodies in the medulla, primarily the medial and dorsal raphe nuclei, and accompany the NE neurons to the hypothalamus, limbic forebrain, neostriatum, paleostriatum, and neocortex. (From *Neuropsychopharmacology, 1: The Monoamine Systems*, 1976. Reproduced with permission from Roche Laboratories, Nutley, New Jersey.)

depression, there is a *decrease* in the functional level of norepinephrine in the brain and in *mania* an *increase* (see Chapter 10 for serotonin's role in sleep).

Other investigators believe that there may be *two different kinds of depressive syndromes*, one associated with *decreased functional levels of norepinephrine* in the brain and the other with *decreased functional levels of serotonin*. The urinary excretion of *3-hydroxy-4-methoxyphenylethylene glycol* (MHPG), a metabolite of norepinephrine largely produced in the brain, is found to be low in some depressed patients, but not in others. Those who have low levels of MHPG tend to respond better to imipramine, a tricyclic antidepressant that increases the brain levels of norepinephrine and, to a lesser extent, of serotonin, whereas those who do not have low levels of MHPG tend to respond better to amitryptyline, a tricyclic that increases brain serotonin levels more than imipramine (Maas, 1978).

More recent studies indicate, however, that the CSF MHPG levels are increased rather than decreased in depressed patients. In addition, the urinary excretion of epinephrine and norepinephrine and their metabolites was increased in depressed and manic patients as opposed to controls (Koslow *et al.*, 1983). Thus, a hyperfunctioning rather than hypofunctioning noradrenergic system is suggested in affective-disorder patients.

Receptor Sensitivity

More recent evidence suggests that changes in the sensitivity of postsynaptic receptors to norepinephrine and serotonin may play a more important role in affective disorders than the functional levels of the biogenic amines (Charney *et al.*, 1981, 1982).

Antidepressant therapy in depressed patients requires several weeks to take effect, while the functional levels of biogenic amines at synapses increase almost immediately following drug administration through the blockade of their reuptake into the presynaptic neuron. Long-term antidepressant therapy has been shown to change the sensitivity of postsynaptic receptors in complex ways: increased sensitivity to serotonin and α-adrenergic stimulation and decreased sensitivity to β-adrenergic stimulation.

Acetylcholine

In addition to the biogenic amines norepinephrine and serotonin, acetylcholine is also probably involved in depression. There is evidence

to indicate that the brain cholinergic function may be high in depression and low in mania (Janowsky *et al.*, 1972, 1973). Physostigmine, a substance that inhibits cholinesterase, the enzyme that breaks down acetylcholine, was shown to convert mania to depression and to make depression worse.

Thus, three major neurotransmitters in the brain, acetylcholine, norepinephrine, and serotonin, seem to be involved in at least some forms of depression. Another way of conceptualizing the relationship between the neurotransmitters and depression is that the central nervous system must function as a system in which there is an equilibrium of neurons using different transmitters. Such a system may become dysregulated if any of the components malfunction, and the overall equilibrium of the neurotransmitters may shift, producing abnormalities in more than one subsystem. At the same time, in one dysregulated system, one may find particularly reduced function of one component (say, norepinephrine), while in another dysregulated system with the same behavioral characteristic, one may find reduced function of another component (such as serotonin).

Brain noradrenergic function may be reduced in cats in states of heightened emotionality, regardless of whether the state is pleasurable or aversive (Bliss and Zwanziger, 1966; Bliss *et al.*, 1966).

Other Putative Neurotransmitters and Neuromodulators

Numerous other substances, among them the endorphins and enkephalins (see Chapters 4 and 7) and, possibly, histamine, have been suspected of being involved in mood states, but the findings are still too preliminary to warrant full discussion.

Intracellular Sodium

In both depression and mania, there seems to be an increase in residual sodium (intracellular sodium plus the sodium in bone). In depression, the magnitude of increase is 50% and in mania, 100%. This increased intracellular sodium (hypothesized for the brain) may lower resting membrane potential and increase neuronal irritability and thus "arousal." There may be central hyperarousal in both depression and mania, as evidenced by insomnia, especially reduction in the delta (deep) sleep, and lowered threshold for arousal (Whybrow and Mendels, 1969).

Parenthetically, alcohol also increases intracellular sodium and potentially aggravates depression.

Psychological Aspects

Early psychoanalytic formulations concerning depression focused mainly on object loss and *turning inward of hostility* that had originally been directed toward an ambivalently loved person who had been lost through death or separation (actual, threatened, or symbolic) (Freud, 1917). Later, "ego psychologists" such as Bibring (1965) added the formulation that depression is a state in which the individual recognizes his inadequacy and *helplessness*—loss of self-esteem. Beck (1967) postulates an altered *cognitive style* characterized by negative expectations to be the basis of depressive states. His theory, then, gives primacy to the cognitive or thinking aspects of depression—negative conception of the self, negative interpretations of one's experiences, and a negative view of the future. From these cognitive changes arise helplessness and hopelessness and the mood of depression. The "learned helplessness" model of Seligman (Seligman and Maier, 1967) provides an interesting model for depression. Many animals that had been exposed to multiple sessions of inescapable shock showed impaired ability to avoid shock even when it was avoidable—as though they had "learned" that the shocks were not avoidable.

According to Wolpe (1971), chronic anxiety resulting from chronic frustration in one's personal or professional life causes *hopelessness* concerning the reduction of anxiety. Related to these theories are the observations that antecedent to a depressive episode, there is often a lack of environmental rewards (positive reinforcement) for formerly successful behaviors.

A Hypothetical Integrated Model (Figure 13)

Depression may be conceptualized as expressing a state of the central nervous system with accompanying neuroendocrine and physiological changes. This state of depression may have a *final common pathway*, which might be the state of brain neuronal systems concerned with reinforcement and pleasure. Environmental, symbolic, and genetic–chemical factors would contribute to and influence this final common pathway to varying degrees. For example, the basic vulnerability

DEPRESSION
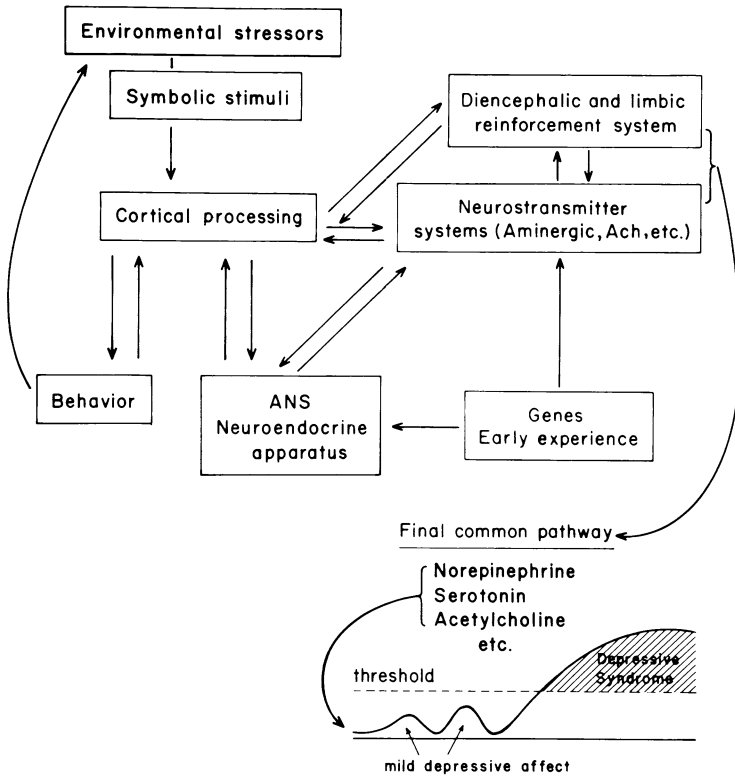
Figure 13. An integrated model of depression.

of the neurotransmitter (and perhaps reinforcement) systems in the brain may be determined by genetic (and early experiential) factors.

Psychosocial stresses such as separation and loss might be translated in the brain into neuronal impulses and neuroendocrine responses, which in turn may change the activity of neurons in the neurotransmitter and reinforcement–pleasure systems. Vulnerability to depression may be conceptualized as related to a hypothesized threshold in the central nervous system. Given a large *genetic loading* (low threshold) for depression, only a minimal amount of symbolic stimuli may be sufficient to trigger a severe depressive state. On the other hand, in some individuals with no genetic loading (high threshold), even catastrophic losses conceivably might not result in a detectable depressive state. Once a de-

pressive state occurs, it may further aggravate depression through behavioral changes (e.g., social withdrawal) and cognitive changes (e.g., pessimistic outlook).

On the basis of clinical studies, it seems that once a severe *depressive syndrome* has developed, no matter what the precipitating event, it reaches a phase that has an autonomous course that can be modified only by time or somatic intervention, such as antidepressants or electroshock therapy. Thus, one might postulate a threshold for the final common pathway for depression—once the threshold has been reached for the depressive syndrome, a process similar to a stress-induced disease in other organs (such as stress-induced peptic ulcers) may ensue. This threshold might speculatively be related to the functional level and sensitivity of noradrenergic, serotonergic, and cholinergic systems related to reinforcement and pleasure. Seen in this light, depression would be a stress-related disease with the brain as the target organ (Figure 13).

Physiology and Endocrinology of Affective Disorders

Depression is associated with a specific pattern of facial muscular patterning (Izard, 1977) that is identifiable in monkeys and humans and across cultures. This characteristic facial expression, together with the specific behavior patterns (such as protest and despair), is probably associated with the adaptive significance of depression discussed above.

In states of *sadness*, there seems to be agreement that the parasympathetic nervous system is activated, with concomitant physiological alterations. The latter include lacrimation and decrease in blood pressure and heart rate (Izard, 1972). In the *depressive syndrome*, however, the physiological changes are quite complex. In patients with established depressive syndrome, the heart rate may be increased, and increased blood pressure has been associated with depression (Heine *et al.*, 1969; Altschule, 1953). The increased sympathetic activity found in the depressive syndrome may be associated with the central arousal and anxiety that often accompany the syndrome.

Neuroendocrinology

Endocrine changes have been studied primarily in patients with the full-blown depressive syndrome. A number of typical endocrine changes accompanying depression have now been identified, and it is also known that primary endocrinopathies may cause secondary depressive

syndromes or depressive symptomatology (see the section on Evaluation of Depression). In general, the endocrine changes found in the depressive disorder (depressive syndrome) are the kinds of changes that might be anticipated on the basis of the effect of functionally decreased levels of brain norepinephrine on the hypothalamic releasing and inhibitory hormones.

Adrenocorticotropic Hormone and Cortisol. Adrenocorticotropic hormone (ACTH) seems to be synthesized and released in response to the hypothalamic corticotropin-releasing factor (CRF). Under normal conditions, CRF secretion appears to be tonically inhibited by the higher brain centers through a noradrenergic (norepinephrine) influence.

In the depressive syndrome, there may be a severe *disinhibition of the hypothalamic–pituitary–adrenal* function. Thus, in depression, there is a marked elevation in the plasma cortisol levels throughout the day and night, obliterating the normal circadian rhythm of cortisol secretion (Ettigi and Brown, 1977; Sachar, 1975). Depressive patients with elevated cortisol secretion often do not show normal suppression of cortisol production after the administration of *dexamethasone*, a potent synthetic corticosteroid. Dexamethasone normally suppresses pituitary ACTH secretion and thus lowers plasma cortisol. The depressive patient's *dexamethasone suppression test* usually returns to normal when depression improves (Carroll, 1972).

Although nonspecific emotional arousal can also result in increased cortisol levels, the increase accompanying the depressive syndrome seems to be fundamental and not secondary to arousal, as evidenced by elevated cortisol levels in sleeping patients and apathetic patients.

Growth Hormone. Growth hormone secretion is probably controlled by a hypothalamic releasing factor (GHRH) and an inhibiting factor (somatostatin). Growth hormone release normally occurs in response to hypoglycemia, exercise, stress, arginine infusion, and slow-wave sleep.

There is evidence to suggest that catecholamines (norepinephrine and dopamine) mediate growth hormone secretion. In depression, there appears to be a *decreased growth hormone response* to hypoglycemia.

Other Hormones. In addition to corticosteroids and growth hormone, the levels of which are probably altered in depression, other hormone systems, such as luteinizing hormone, thyroid hormone, and prolactin, might also be altered in depression, although this is not yet well established.

The endocrine changes in mania are not well established and are at best controversial.

Physiological Signs of Depression (Depressive Syndrome)

In severe depression (depressive syndrome), there is often a loss of appetite, decreased sexual interest and drive, profound loss of interest in and experience of pleasure, constipation, and weight loss. In some patients, there may be an increase in appetite and weight gain, especially when depression is not severe. Sleep disturbances are common, especially early-morning awakening. There may be profound psychomotor retardation or agitation. Vague physical symptoms such as aches and pains may be present.

Evaluation of Depression

When the physician suspects that a patient is depressed, specific types of further evaluation should be made. The kinds of things the physician must decide in the course of this evaluation are the *severity* and *nature* of depression, *suicidal risk, advisability of hospitalization*, and *management plans.*

We mentioned earlier that depressive affect is usually generated in response to a loss and that it may have adaptive significance. In cases of mild depression (or, rather, sadness), recognizing the loss suffices in the evaluation, since the loss can be dealt with by the patient alone or with the support of his family, friends, or the physician. The same is true in grief reactions, in which the nature of the loss is clear. Grief reactions often occur in the medical setting in anticipation of the *loss of function* or of an organ, such as in amputations. Grief over the patient's own impending death also occurs. Duration of depression is an important factor to be considered. A patient who has become seriously depressed in the last month, for example, needs a different management plan from another patient who has been depressed for the last five years.

The following specific questions, then, should be answered in the comprehensive evaluation of a patient with suspected depression:

1. Questions concerning the *phenomenology* of depression
      Sad affect, loss of interest
      Difficulty with concentration
      Crying

        Guilt feelings
        Hopelessness and helplessness
        Low self-esteem
        Decreased libido
        Anorexia
        Constipation
        Dry skin
        Dry mouth
        Vague aches and pains
        Sleep pattern
        Suicidal thoughts or plans

2. Observation of *behavior and appearance*
        Sad or apathetic appearance
        Evidence of self-neglect
        Agitation or psychomotor retardation
        Cognitive disturbances

3. Questions concerning *history of depression*
        Duration of the depression
        Past history of depression or mania
        Family history of depression, mania, suicide, or alcoholism

4. Questions concerning *medical history*
        Possible concurrent disease
        Drugs

5. Questions concerning *environmental factors*
        Losses
        Separations
        Anniversary of above

Once mild sadness and grief ractions have been ruled out, the physician should determine whether the depression is *chronic* or *acute*.

Chronic depressive disorder (dysthymic disorder) is defined as moderate to severe depression with a duration of *longer than two years without remission*. With these patients, sad feelings and some of the other psychological and physiological signs of depression seem to be a *character style*. Depression in this case, then, is a trait that cannot be expected to be easily removed by treatment.

If the patient's depression does not fall under the chronic depressive disorder, then the presence or absence of the depressive syndrome (depressive episode) has to be determined. An acute *depressive syndrome* can be diagnosed by the criteria shown in Table 3.

Once the diagnosis of *depressive syndrome*, complete or partial (although not all criteria are met, sufficient criteria are fulfilled to cause

Table 3. Diagnostic Criteria for Depressive Syndrome (Depressive Episode)[a]

1. Dysphoric mood or loss of interest or pleasure in all or almost all usual activities and pastimes. The dysphoric mood is characterized by symptoms such as the following: depressed, sad, blue, hopeless, low, down in the dumps, irritable, worried. The disturbance must be prominent and relatively persistent but not necessarily the most dominant symptom. It does not include momentary shifts from one dysphoric mood to another dysphoric mood, for example, anxiety to depression to anger, such as are seen in states of acute psychotic turmoil.
2. At least four of the following symptoms:
   a. Poor appetite or weight loss or increased appetite or weight gain (change of one pound a week or ten pounds a year when not dieting)
   b. Sleep difficulty or sleeping too much
   c. Loss of energy, fatigability, or tiredness
   d. Psychomotor agitation or retardation (but not mere subjective feelings of restlessness or being slowed down)
   e. Loss of interest or pleasure in usual activities or decrease in sexual drive
   f. Feelings of self-reproach or excessive or inappropriate guilt (either may be delusional)
   g. Complaints or evidence of diminished ability to think or concentrate, such as slow thinking or indecisiveness (do not include if associated with obvious signs of schizophrenia)
   h. Recurrent thoughts of death or suicide, or any suicidal behavior, including thoughts of wishing to be dead
3. Duration of period of illness of at least two weeks from the time of the first noticeable change in the individual's usual condition
4. None of the following that suggest schizophrenia is present:
   a. Delusions of being controlled or thought broadcasting, insertion, or withdrawal
   b. Hallucinations of any type throughout the day for several days or intermittently throughout a one-week period unless all the content is clearly related to depression or elation
   c. Auditory hallucinations in which either a voice keeps up a running commentary on the individual's behaviors or thoughts as they occur or two or more voices converse with each other
   d. Delusions or hallucinations for more than one month at some time during the period of illness in the absence of prominent affective (manic or depressive) symptoms (although typical depressive delusions, such as delusions of guilt, sin, poverty, nihilism, or self-deprecation, or hallucinations of similar content are permitted)
   e. Preoccupation with a delusion or hallucination to the relative exclusion of other symptoms or concerns (other than delusions of guilt, sin, poverty, nihilism, or self-deprecation or hallucinations of similar content)
   f. Marked formal thought disorder if accompanied by either blunted or inappropriate affect, delusions or hallucinations of any type, or grossly disorganized behavior
5. Not due to any organic mental disorder (organic brain syndrome)
6. Not superimposed on schizophrenia, residual subtype (history of schizophrenia)
7. Excludes simple bereavement following loss of a loved one if all the features are commonly seen in members of the individual's subcultural group in similar circumstances

[a] Quoted, with minor modifications, from the *Diagnostic and Statistical Manual of Mental Disorders* (DSM III) (1980). Reproduced with permission from the American Psychiatric Association.

Table 4. Partial List of Medical Conditions Often Associated with Depression

Endocrinopathies
  Hyperparathyroidism (hypercalcemia)
  Cushing's syndrome
  Hypothyroidism
  Premenstrual tension syndrome
Viral disease (often during incubation and convalescence)
  Influenza
  Infectious mononucleosis
  Infectious hepatitis
  Any other viral infection
Malignancies
  Occult abdominal malignancies, especially cancer of the tail of the pancreas
  Any other malignancy
Drugs that may cause or aggravate depression
  Corticosteroids and ACTH
  Oral contraceptives
  Reserpine
  α-Methyldopa (Aldomet)
  Propranolol (Inderal)
  Alcohol
  Benzodiazepines (e.g., diazepam or chlordiazepoxide)

reluctance in ruling out this possibility), is made it is helpful to make a further decision as to whether or not the depressive syndrome is associated with a known antecedent or cause such as a medical disease, exogenous toxins (e.g., drugs), or psychological stress such as bereavement. Table 4 presents a partial list of medical conditions that must be ruled out in a depressed patient.

It is because of this frequent association of the depressive symdrome with serious medical diseases that a thorough *medical workup* with physical examination and laboratory tests is a *must* in the evaluation of a depressed patient. Once depression secondary to a medical condition is established and the underlying disease diagnosed, the depressive syndrome usually abates when the underlying disease has been satisfactorily treated (vignette 4). If a thorough medical evaluation rules out possible underlying disease, then the possibility of primary depression should be entertained.

Depressive syndrome is an integral part of a psychiatric disorder called *major affective disorder* (primary depressive illness, endogenous depression, manic–depressive illness). In this disorder, the symptomatology of the patient is usually severe enough to meet fully the criteria

for diagnosis of the depressive syndrome. In addition, there is often a positive *family history* of depression, mania, suicide, or alcoholism. About 15–20% of the first-degree relatives of patients with major affective disorders have histories of similar illnesses. History of a previous depressive episode is common, since in 95% of patients, one or more episodes of depression can be expected to occur within ten years after the initial episode. The first episode may occur at any age, but is most common in middle age. In some patients, depressive episodes may alternate with, or be associated with, manic episodes (bipolar affective disorder). It appears that the *unipolar* and *bipolar disorders* have different genetic vulnerability, so that a depressed patient who has a relative who had manic episodes may eventually have a manic episode as well.

A depressive episode due to major affective disorder is usually self-limiting, the average duration without treatment being about six to nine months (in bipolar disorder, somewhat shorter; in unipolar, somewhat longer).

Dexamethasone Suppression Test

As previously mentioned, patients with depressive syndrome due to major affective disorder, particularly the unipolar type, tend to secrete high levels of cortisol in the blood, and the dexamethasone suppression test (DST) is often abnormal in such patients. The DST may thus be used to confirm the diagnosis of depressive syndrome (Carroll *et al.*, 1981; Schatzbert *et al.*, 1983).

In an outpatient setting, 1 mg dexamethasone is taken orally at 11 P.M., and blood is drawn at 4 P.M. the following day for cortisol level determination by the radioimmunoassay method. A 4 P.M. cortisol level of 5 μg/dl or above is considered to be a positive result (nonsuppression).

One should be aware that a number of medical conditions, including, of course, Cushing's syndrome, cause nonsuppression in the DST. Simple weight loss may be associated with a positive DST. Many drugs also interfere with the DST. The DST may also be positive in other psychiatric conditions including dementia.

When a depressed patient is successfully treated, the DST seems to revert to normal. Failure of the DST to normalize despite vigorous antidepressant drug therapy may be a poor prognostic sign concerning the depressive syndrome.

As clinical experience accumulates, it appears that the DST may not be as specific as originally thought. Final appraisal of the DST as a clinical diagnostic test awaits further research.

Physical Symptoms in Depressive Syndrome Associated with Major Affective Disorders

Certain physical and physiological symptoms and signs are often present in the depressive syndrome. In fact, some of the physiological signs, such as loss of appetite and weight loss, sleep disturbance, and loss of sexual interest, are part of the diagnostic criteria. In addition, many patients have *vague pains* and *discomfort* in various parts of the body, perhaps associated with a turning inward of attention with depression. To complicate matters even more, some patients who have the depressive syndrome have *primarily* physical symptoms, with relatively mild to moderate depressive affect, and may seek help for physical symptoms such as vague toothaches, headaches, and back-aches (related concepts are "depressive equivalents" and "masked depression"). In some, this may be a heterothetic help-seeking behavior (see Chapter 1). In patients complaining of vague physical pains and discomfort whose medical workup is negative, the physician should consider the possibility that the symptoms may be associated with the depressive syndrome. Careful questioning about affect and other associated symptoms and signs and history will usually disclose more features of the depressive syndrome.

Evaluation of Suicide Potential

An important consideration in evaluating a depressive patient is the suicide potential. Approximately 15% of depressive patients ultimately commit suicide (Pokorny, 1977). In a study of 134 consecutive suicides, Robins and associates found, on the basis of interviews with relatives and medical records, that about 45% of the suicides probably had a depressive disorder (Robins *et al.*, 1959a,b).

Risk Factors

In a classic book called *Le Suicide*, the French sociologist Emile Durkheim (1897/1966) studied the sociological factors influencing suicide rates in different cultural conditions. He found that suicide rates were higher in the Protestant areas of Europe as opposed to the Catholic areas, which he attributed to the spirit of free inquiry and individualism among the Protestants. Durkheim postulated that loosening of the individual's ties with the society, as in the case of Protestants and unmarried persons,

tends to lead the individual to question the purpose of life itself and, thus, to a heightened risk of suicide This type of suicide was called the egoistic suicide." Durkheim also described the "altruistic suicide," occurring in an opposite cultural setting. Altruistic suicide occurs in settings of excessive lack of individualism, such as the suicide of women on the death of their husbands in some cultures. In this type of suicide, there is a sense of duty, a social prescription for the suicide. Durkheim also included religious suicides, such as self-immolation occurring in certain Buddhist sects, in the category of altruistic suicide.

Anomie and *anomic suicide* are important concepts proposed by Durkheim. He noted that the suicide rate increased at times of relative peace and financial prosperity. He also noted that the suicide rate was high in the European countries where the divorce rates were high. He postulated that at times of peace, relative financial prosperity, and following divorce and separation, there is a loss of regulation in one's life—that is, the individual feels lost without a sense of purpose and demands. He called this state of lack of regulation "anomie" and the resultant suicide occurring in some individuals "anomic suicide" (Durkheim, 1897/1966).

Current statistics show that suicide is the second leading cause of death for white males between the ages of 10 and 55 in the United States. It is one of the ten leading causes of death for both men and women up to the age of 75. The rate of actually committed suicide is higher for men than for women (3 to 1), but more women than men attempt suicide (2 to 1).

Suicide is common among certain professions, especially physicians. Under the age of 40, suicide is the leading cause of death among physicians, psychiatry leading other medical fields (Schneidman *et al.*, 1975).

In the United States, the rate of suicide in the *general population* is approximately 1 in 10,000. The risk for successful suicide is higher in *single, separated,* or *divorced persons;* in *men;* and in *older persons.* Suicide risk is higher in patients who have pain, who are *depressed,* or who have a history of a depressive syndrome. Patients who live *alone,* who drink *heavily,* and who have experienced a *recent stressful event* are also at a higher risk for suicide.

About 80% of persons who commit suicide give *definite warnings about their intent* (Motto, 1975). Suicides often occur during a phase *when the person's depression seems to be lifting.* When a depressed patient has decided to commit suicide, there may be a sudden lifting of depressive affect, with a sense of resolution (vignette 3). With antidepressant therapy, the suicide risk rises initially as the patient becomes more energetic

Table 5. Suicide Risk Factors

1. Presence of a depressive syndrome, including suicidal thoughts; especially, has the patient planned how he would do it?
2. Demographic risk factors: religion (high in non-Catholics), marital status (higher in single, divorced, and separated persons), older age, male sex
3. Presence of a painful condition or other medical disorder
4. Living conditions—living alone increases risk
5. Alcohol use—heavy use increases risk
6. Behavioral warnings of suicide—seeking help (including medical), talking about suicide, giving away possessions, putting personal affairs in order, hoarding drugs, buying weapons, and other similar acts
7. Apparent lifting of depression—suicide occurs more often following this
8. Ready availability of the means of suicide, for example, large quantities of prescribed medication or a rifle hanging in the den
9. Previous suicide attempt, history of depression, family history

and able to contemplate the execution of a suicide plan. Many persons intending to commit suicide indicate this decision by such actions as making out a will, getting insurance, and giving away personally valued possessions (as in vignette 3).

Many people who ultimately commit suicide have recently *engaged in help-seeking behavior*. (Seventy percent of depressed persons committing suicide were in touch with a physician within 30 days of their death, and nearly half during the preceding week.)

To recap, then, suicide potential should be evaluated by asking specific questions about the risk factors listed in Table 5.

Suicide Attempt

The physician usually encounters the suicidal patient in two contexts: the patient who is depressed and therefore a suicide risk and the patient who comes to the physician's attention because of a suicide attempt. The latter is an especially important problem encountered in emergency rooms and intensive care units.

It used to be said that suicide attempters seldom actually commit suicide—this is *not* true. Approximately 1% of suicide attempters who were admitted to the hospital complete suicide *each year* (Weissman, 1974). Although not all suicide attempters ultimately wind up committing suicide, the suicide attempt is usually an indication of serious suffering, which may be alleviated if proper help is offered by the physician.

In contrast to completed suicides, suicide attempters tend to be *young* (peak age, 20–24 years), 50% of attempters being under 30 years of age (Weissman, 1974). Again, unlike completed suicides, women outnumber men by 2 to 1 among suicide attempters.

Suicide attempt often occurs in the context of *interpersonal difficulties*. Although it is more frequent among separated and divorced persons, suicide attempt also occurs among married persons. Suicide attempt is most frequent among young persons, who also tend to be single. Although, as Durkheim pointed out, suicide mortality is lower in countries that have a large number of Roman Catholics, suicide attempt does not seem to be infrequent among Catholics (Weissman, 1974).

The most common form of suicide attempt is drug overdosage (70–90% of all attempts). Barbiturates, tranquilizers, and antidepressant medications are among the drugs commonly used in suicide attempts.

Evaluation of Suicide Attempt

In evaluating a patient who has attempted suicide, we have to consider (1) the current context, including the immediate reason for the attempt; (2) the recent context or recent events or changes that culminated in the patient's attempt; and (3) the background factors, such as the patient's personality, cultural forces, and the meaning of suicide. (For a detailed discussion of these contexts, see Part III.)

Current Context Factors. Method of suicide attempt. The lethality of the method is an important consideration. A patient who attempts suicide by using a gun is obviously more serious in his attempt than someone who ingests ten tablets of aspirin. However, one has to consider the patient's own ideas about the lethality of the method used. For example, a patient who attempts suicide with aspirin believing it to be very lethal is more serious than someone who takes an overdose of antidepressants believing that antidepressants could not seriously endanger life. (In fact, antidepressants are highly lethal.)

Although ingestion of drugs may not be as *immediately* lethal as shooting, this is still the most common method of suicide attempt and completed suicide. Whether the patient has any *further supply* of the drug (which might be used in a repeat attempt) and *where the drug was obtained* should be ascertained. In one study, over half the patients who committed suicide by overdosage had received prescriptions for fully lethal amounts of hypnotic medication that they ingested, or unlimited prescriptions, from their physicians (Murphy, 1975a). The *physician*

should be alerted to the patient's suicide attempt as soon as possible if the patient is brought directly to the hospital.

Emotional state of the patient. This includes the seriousness of *suicidal intent*, presence or absence of *intoxication* or *overwhelming emotion* at the time of the attempt, and degree of impulsivity. The seriousness of the suicidal intent is best evaluated by direct questions of the patient, such as, "What did you have in mind when you took the pills? Did you seriously want to die? What ideas did you have about what would happen after you died?" Alcohol or other drug intoxication, presence of rage, or an impulsive suicide attempt militate against the patient's suicidality being prolonged beyond the current attempt. However, the presence of depressive affect or apathy on a continuing basis indicates continuing suicide risk.

Patient's behavior and psychological state around and after the attempt. If the patient went to a secluded place before ingesting an overdose, the seriousness of the suicide risk should be considered grave, even if the method of attempt was less serious (e.g., aspirin). Making a phone call to a friend after ingestion of an overdose probably indicates the presence or ambivalence and a wish for rescue.

Recent Context Factors: Recent Events, Changes, Stresses

Depression. Eighty percent of suicide attempters are *clinically depressed* (i.e., have the depressive syndrome) at the time of the attempt (Silver *et al.*, 1971). The patients should be *asked specific questions* concerning mood, loss of interest, libido, sleep patterns, appetite, weight loss, fatigue, etc., to determine whether the suicide attempt is a part of the depressive syndrome.

Interpersonal conflict. This includes recent marital problems, arguments with lovers, and separations. In certain situations, the interpersonal conflict may be resolved after a suicide attempt, for example, marital reconciliation. In other situations, no interpersonal change occurs, and the *risk may continue.*

Help-seeking behavior and pain. Suicide attempt occurs often in patients with *chronic or severe pain*. Pain can also be a symptom of a depressive syndrome. Help-seeking behavior of any kind occurs often before a suicide attempt. Understanding the help-seeking behavior pattern of the patient will help the physician to recognize future suicide potential. For example, increased frequency of visits to the doctor with complaints of vague pains may be the most prominent symptom of increasing depression for a patient, culminating in a suicide attempt.

Termination behavior. Persons who have decided on suicide often engage in activities indicating their intention, such as giving away prized possessions and making out a new will (vignette 3).

Previous suicide attempts. Over two thirds of those who eventually commit suicide have histories of suicide attempts or threats (Murphy, 1975b). Positive findings in any of the recent-context factors indicate that the attempt was a serious one requiring thoughtful management.

Background Context Factors: Personality, Constitution, Culture

Demographic data related to the risk factors. The risk for completed suicide increases (if the patient attempts suicide again) with increasing age; in men; in persons whose marital status is single, divorced, or separated; in non-Catholics; and in certain professions (e.g., physicians).

Cultural views on suicide. Culture often influences the desirability of suicide as an option (e.g., in Japanese culture) and the method of suicide. The latter is obviously related to the lethality of the attempt. Culture and early history also often influence the psychological meaning of suicide. For example, death by suicide may mean reunion with a loved one, liberation from pain and suffering, or eternal fire and brimstone.

Personality of the patient. Impulsive personality style tends to increase the risk for impulsive suicide. On the other hand, patients who tend to be orderly and controlling (see Chapter 17) are more likely to be successful in a suicide attempt because of their tendency for careful planning. In the past, "hysterical personality" was considered to have a low risk for actual suicide—this is *not* true. Suicide attempts are very common in hysterical personality, and completed suicide is not uncommon.

Constitutional or genetic factors. These are important in that the depressive syndrome has a genetic predisposition. Family history of suicide increases the risk for the patient's having a major affective disorder and for suicide.

The background context factors provide information on the broad background factors for assessing a patient's suicide risk.

Management of Depression

A comprehensive evaluation of the depressed patient is the first step toward management. This includes the nature and severity of the depressive affect as well as the life situation of the patient.

Mild depressive affect occurring in response to loss or separation usually does not require specific treatment. In severe *grief reactions*, a generally supportive environment and empathic attitude by the family,

friends, and physician usually allow the "grief work" to proceed to resolution in time. On occasion, the grieving person may be *frightened* about the very vivid images of the deceased that he or she may experience or, occasionally, by *hallucinations* concerning the deceased. Reassurance by the physician that these are normal phenomena occurring in the course of bereavement or grief is effective and enlightening. Antidepressant medications are generally *not* indicated and ineffective in uncomplicated grief reactions. If the sleep disturbance is severe or anxiety symptoms are prominent, mild sedatives or tranquilizers may be of transient benefit, for example, 15 mg flurazepam at bedtime for sleep when necessary.

For *chronic depressive disorder*, the recognition of the chronic nature of the depression as a personality style is the key to management. Depressive ways of looking at things, relating with people, and feeling are unlikely to change easily or promptly. The health-care personnel should consider this as a limiting factor in management. Long-term psychotherapy or restructuring of the patient's life may be effective in some instances, but this requires extensive investment of time, money, and effort. When the physiological symptoms and signs of depression are prominent in a chronic depressive disorder, antidepressants may be transiently effective, but the chronic characterological aspects are unlikely to respond.

Once the presence of an *acute depressive syndrome* has been recognized, the physician should determine whether it is due to or associated with a medical disease such as an occult carcinoma or an endocrinopathy. If this is the case, the *underlying medical disease* should be treated. In the meanwhile, if the depressive symptomatology is very severe, including serious suicidal ideations, a *protective psychiatric hospitalization* may be necessary. (The medical disease should be treated in the psychiatric unit, or, if surgery is needed, the patient could be transferred for the operation after initial admission to psychiatry.) Antidepressants are not usually effective in depressive syndromes due to medical diseases.

If the diagnosis of a *major affective disorder* is made in a patient with the depressive syndrome, then specific treatments for this disorder are to be considered.

Before embarking on a specific treatment regimen for the depressed patient, a decision should be made as to whether the patient should be *hospitalized*. If the patient is at high risk for suicide, has active suicidal thoughts or plans, is unable to function at work or home, or cannot care for himself, then hospitalization is definitely indicated.

Psychotherapy as a general measure is valuable and often by itself sufficient in milder cases. Psychotherapy in this context should begin as a supportive relationship with a physician who understands the patient's experience of the illness, who has a hopeful and confident attitude, and who encourages the patient to share and discuss his problems. More ambitious or active psychotherapy with such patients should be undertaken only by a specialist. The frequency of such psychotherapy can be flexible, but initially, it should be at least once a week. If the patient is to be treated as an outpatient, the physician should determine whether the patient needs hospitalization at the time of each therapy visit, as the need may arise in the course of treatment.

In addition to the type of psychotherapy described above, specific treatment for the depressive syndrome that is a part of the major affective disorder may include *antidepressants, lithium carbonate,* or *electroconvulsive therapy.* In patients with bipolar disorder, lithium is helpful in treating the acute manic episode and especially in the prevention of recurring manic episodes. Lithium also has the beneficial effect in unipolar depressions of reducing the frequency and severity of recurrent depressive episodes.

Antidepressants include the *tricyclic antidepressants* and the *monoamine oxidase inhibitors.* Because the monoamine oxidase inhibitors have many interactions with other medications and certain foods (any food containing tyramine, such as aged cheese, wine, and pickles), tricyclic antidepressants are more commonly used, especially in patients with concurrent medical problems. The use of antidepressants and lithium is described in more detail in Chapter 20.

Electroconvulsive therapy. (ECT) is an effective treatment for depression. It is usually performed on hospitalized patients who are very agitated and suicidal. Contrary to popular belief, ECT therapy is quite painless. (The patient is anesthetized with sodium pentothal, and muscles are relaxed with a relaxant such as succinylcholine during ECT). ECT is a safe procedure. The only absolute contraindication to ECT is increased intracranial pressure or recent cerebrovascular accident.

Generally, *antidepressant medications* should be tried first in conjunction with psychotherapy to treat the depressive syndrome. Antidepressant drugs should be used in adequate doses for an adequate amount of time (at least two to three weeks) before the effectiveness is determined. Since antidepressants are potentially lethal medications, however, the prescription for an outpatient should not exceed more than one week's supply (another reason the doctor should see the patient at least every week—to give new prescriptions). If the antidepressant is

not effective, or if the patient is acutely suicidal and agitated, the patient may need hospitalization and, possibly, ECT.

With antidepressant therapy or ECT or both, 80–90% of patients with the depressive syndrome will respond dramatically within four to six weeks of treatment. Antidepressant drugs should, however, be continued for at least six to nine months (for the duration of the natural history of the depressive syndrome, which is about nine months without treatment). For some patients, more intensive long-term psychotherapy for underlying chronic unresolved conflicts that may contribute to depressive episodes may be indicated.

The management of *suicidal states* should also be based on comprehensive evaluation. Depression, intoxication, and other acute psychological and medical states should be managed with specific treatment, protection, and general supportive measures. Acutely suicidal patients should be hospitalized, by commitment if necessary. Long-term management and prevention should be based on each of the factors described in the section on Evaluation of Suicide Potential.

Summary

Sadness is an affect usually associated with loss, failure, or separation. Although sadness and grief are normal experiences, the extreme form, depressive syndrome, is a pathological condition. At the opposite pole from the depressive syndrome in mood is the manic syndrome, also a pathological condition. There are specific features and criteria for the diagnosis of the depressive syndrome.

Affective disorders are the most common psychiatric disorders, affecting approximately 20% of the general population. Unipolar affective disorders are much more common than bipolar affective disorders. Women are affected twice as much as men by unipolar depression, while the sex ratio for bipolar disorder is 1:1.

Grief reaction is a response to acute loss. The symptomatology includes somatic distress, intense preoccupation with the image of the deceased, guilt feelings, irritability, and change in patterns of behavior. The course of bereavement or the grief reaction has been described as consisting of three phases: numbness, pining (anxious searching), and depression and despair. The course of the normal acute grief reaction is approximately four to eight weeks, but a substantial portion of the bereaved continue to feel some distress up to one or two years after the death of a spouse. Pathological grief reactions consist of delayed and distorted grief reactions. Depressive syndrome may also ensue.

Studies of young children separated from their mothers show three phases: protest, despair, and detachment. Phenomena similar to those occurring in the protest and despair phases have also been described in infant monkeys that are separated from their mothers. Monkeys that are socially isolated from early life also develop difficulties in later life, especially in social and sexual activity.

There is an increased morbidity and mortality due to many causes in depressed patients and in the bereaved population.

Depressive affect may be adaptive. It serves as a signal of a loss or separation. The typical expression and behavior pattern (such a protest or despair) may mobilize social support and nurturance. Reduction of activity and withdrawal may facilitate conservation of energy and resources in the face of adversity. In childhood, the threat of sadness resulting from loss of love and approval by the parents and others may help the socialization and learning process.

The *brain mechanisms* of depression are not completely understood. Presumably, there is a dysfunction of the brain areas involved with pleasurable and unpleasurable affects, including the medical forebrain bundle and the periventricular system, respectively, and perhaps the whole limbic brain. The major neurotransmitters implicated in depression include norepinephrine, serotonin, and acetylcholine. The catecholamine theory of affective disorders postulates that there is an increase in the functional level of norepinephrine in the brain in mania and a decrease in its level in depression. Psychological aspects include possible turning inward of aggression, altered cognitive style due to repeated failures, and the response to the recognition of the inadequacy of the individual to perform a task. An integrated model postulates multiple factors (environmental, psychological, genetic, and neurophysiological) that may lead to a final common pathway of dysfunction in the reinforcement system of the brain.

In depression, there is often a marked increase in the corticosteroids and an abnormal dexamethasone suppression test. Growth hormone response to hypoglycemia is often decreased in depression.

The *evaluation* of depression should consider the severity and nature of depression, suicide risk, advisability of hospitalization, and management plans. *Medical conditions* that may cause depression, such as endocrinopathies and occult malignancies, should be carefully ruled out.

An important consideration in the evaluation of depression is the patient's suicide potential. The risk factors for suicide include male sex; single, separated, or divorced marital status; older age; certain professions; lack of social support; heavy drinking; recent stressful event; anomie; certain religious groups; and, most important, presence of depres-

sion and past history of suicide attempt. Among the signs of increasing suicide risk are help-seeking behavior, including contacting a physician for medical problems, seeming lifting of depression, giving away possessions, and reference to suicide.

Suicide attempt is much more common than completed suicide. The uncompleted suicide attempters tend to be younger and female as compared to completed suicides. A suicide attempt often results in the context of interpersonal conflict. It is a serious cry for help and should be evaluated systematically.

The *management of grief reactions* includes supportive interpersonal environment and reassurances concerning the symptomatology when indicated. Antianxiety agents might be indicated transiently in some cases. *Chronic depressive disorder* should be considered a characterological style that is not amenable to rapid change but should be a consideration in managing the patient for concurrent medical problems.

Acute depressive syndrome due to major affective disorder should be treated with psychotherapy (doctor–patient relationship) and a specific treatment such as antidepressant medications or electroconvulsive therapy. In bipolar disorders, lithium carbonate is indicated.

The *underlying medical disease* should be treated in depressive syndrome associated with a medical condition.

An important clinical decision in managing a depressed patient is whether or not to hospitalize. Suicidality and inability to function are indications for hospitalization.

Implications

For the Patient

Depression is often a natural response to a medical illness or hospitalization with attendant loss of autonomy and function. Severe depression (depressive syndrome), however, colors every aspect of the patient's life, including his thinking process (slowed and lacking concentration), outlook (hopeless), and behavior (self-defeating or unable to act). Thus, depressed patients often do *not* consult a physician in the presence of serious symptoms or signs of disease and may have an unrealistically pessimistic outlook on existing disease. On the other hand, a relatively mild depression may increase the tendency for help-seeking behavior, especially when accompanied by vague pains and preoccupation with bodily parts. Depression and bereavement are often contributing factors to disease. Depression increases morbidity and mor-

tality in the presence of medical disease and following surgical procedures.

For the Physician

Since depression is common among medical patients, but often not recognized by the physician (Murphy, 1975b), doctors should deliberately evaluate a patient for possible depression. Since depression can be caused by an underlying, undiagnosed medical disease such as occult carcinoma or endocrinopathies, a thorough medical workup should be performed on a patient who is depressed. When a patient appears mentally slowed or especially apathetic or pessimistic about the medical condition, the possibility of concurrent depressive syndrome should be entertained.

Physicians should also be aware that fatigue, vague pains and aches, insomnia, and somatic preoccupation may be symptoms of the depressive syndrome. In these cases, specific questions should be asked to establish or rule out the presence of the syndrome.

For the Community and the Health-Care System

Since mild depression responds to environmental stimuli, hospitals should provide a cheerful physical and environmental situation for the patients. This may also have a preventive effect. More research by the medical profession is needed to increase our understanding of the mechanisms by which depression and bereavement increase morbidity and mortality.

Recommended Reading

Akiskal HS, McKinney WT: Overview of recent research in depression. *Arch Gen Psychiatry* **32**:285–305, 1975. A thorough review of research findings on depression. The authors propose a unified model of depression. Extensive references.

Flach FF, Draghi SC (eds): *The Nature and Treatment of Depression.* New York, John Wiley & Sons, 1975. A comprehensive volume with chapters on various aspects of depression, including the interpersonal, psychodynamic, and environmental, as well as consideration of different age groups; the biological aspects of depression and various treatment modalities are also presented.

Freud S: Mourning and melancholia, in Strachey J (ed): *Standard Edition of the Complete Psychological Works of Sigmund Freud.* London, Hogarth Press, 1975, Vol 14, pp 237–260. Also in *Collected Papers of Sigmund Freud.* London, Hogarth Press, 1925, Vol 4, pp

152–170. (Originally published in 1917). A classic work on the early psychoanalytic understanding of depression, in which Freud postulates the turning inward of aggression.

Jacobsen E: *Depression*. New York, International Universities Press, 1971. A detailed psychoanalytic treatise on depression.

Parkes, CM: *Bereavement: Studies of Grief in Adult Life*. New York, International Universities Press, 1972. A comprehensive discussion and description of the bereavement process. Recommended because so many patients and their families are at risk for bereavement.

References

Akiskal HS, McKinney WT: Overview of recent research in depression. *Arch Gen Psychiatry* **32**:285–305, 1975.

Altschule MD: *Bodily Physiology in Mental and Emotional Disorders*. New York, Grune & Stratton, 1953.

Åsberg M, Bertilsson L, Tuck D, *et al.*: Indolamine metabolites in the cerebrospinal fluid of depressed patients before and during treatment with nortriptyline. *Clin Pharmacol Ther* **14**:277–286, 1973.

Beck A: *Depression: Clinical, Experimental, and Theoretical Aspects*. New York, Harper & Row, 1967.

Bibring E: The mechanism of depression, in Greenacre P (ed): *Affective Disorders*. New York, International Universities Press, 1965, pp 13–48.

Bliss E, Zwanziger J: Brain amines and emotional stress. *J Psychiatr Res* **4**:189–198, 1966.

Bliss E, Wilson V, Zwanziger J: Changes in brain norepinephrine in self-stimulating and "aversive" animals. *J Psychiatr Res* **4**:59–63, 1966.

Bornstein PE, Clayton PJ, Halikas JA, *et al.*: The depression of widowhood after thirteen months. *Br J Psychiatry* **122**:561–566, 1973.

Bowers MF, Heninger GR, Gerbode F: Cerebrospinal fluid 5-hydroxyindolacetic acid and homovanillic acid in psychiatric patients. *Int J Neuropharmacol* **8**:255–262, 1970.

Bowlby J: Grief and mourning in infancy and early childhood. *Psychoanal Study Child* **15**:9–52, 1960.

Boyd JH, Weissman MM: Epidemiology of affective disorders: A reexamination and future directions. *Arch Gen. Psychiatry* **38**:1039–1046, 1981.

Carroll BJ: The hypothalamic–pituitary axis: Functions, control mechanisms, and method of study, in Davies B, Carroll BJ, Mowbray RM (eds): *Depressive Illness: Some Research Studies*. Springfield, Illinois, Charles C Thomas, 1972.

Carroll BJ, Feinberg M, Greden JF, *et al.*: A specific laboratory test for the diagnosis of melancholia: Standardization, validation, and clinical utility. *Arch Gen Psychiatry* **38**:15–22, 1981

Charney DS, Heninger GR, Sternberg DE, *et al.*: Adrenergic receptor sensitivity in depression: Effects of clonidine in depressed patients and healthy subjects. *Arch Gen Psychiatry* **39**:290–294, 1982.

Charney DS, Menkes DB, Heninger GR: Receptor sensitivity and the mechanism of action of antidepressant treatment: Implications for the etiology and therapy of depression. *Arch Gen Psychiatry* **38**:1160–1180, 1981.

Clayton PJ, Halikas JA, Maurine WL: The depression of widowhood. *Br J Psychiatry* **120**:71–78, 1972

Davis JM: Central biogenic amines and theories of depression and mania, in Fann WE, Karacan I, Pokorny AD, et al. (eds): *Phenomenology and Treatment of Depression.* Jamaica, New York, Spectrum Publications, 1977, pp. 17–32.

Diagnostic and Statistical Manual of Mental Disorders (DSM III). Washington, DC, American Psychiatric Association, 1980.

Durkheim E: *Suicide.* New York, The Free Press, 1966. (Originally published in French in 1897.)

Ettigi PG, Brown GM: Psychoneuroendocrinology of affective disorders: An overview. *Am J Psychiatry* **134**:493–501, 1977.

Freud S: Mourning and melancholia, in Strachey J (ed): *Standard Edition of the Complete Psychological Works of Sigmund Freud.* London, Hogarth Press, 1957, Vol. 14, pp 237–260. (Originally published in 1917).

Harlow H, Harlow M: Social deprivation in monkeys. *Sci Am* **207**:137–146, 1962.

Harlow H, Harlow M: Learning to love. *Am Sci* **54**:244–272, 1966a.

Harlow H, Harlow M: Affection in primates. *Discovery* **27**:11–17, 1966b.

Heine BE, Sainsbury P, Chynoweth RC: Hypertension and emotional disturbance. *J Psychiatr Res* **7**:119–130, 1969.

Hirschfeld RMA, Cross CK: Epidemiology of affective disorders: Psychosocial risk factors. *Arch Gen Psychiatry* **39**:35–46, 1982.

Huston PE: Psychotic depressive reaction, in Freedman AM, Kaplan HI, Sadock BJ (eds): *Comprehensive Textbook of Psychiatry, II,* ed 2. Baltimore, Williams & Wilkins, 1975, Vol 1, pp 1043–1055.

Izard CE: *Patterns of Emotions.* New York, Academic Press, 1972.

Izard CE: *Human Emotions.* New York, Plenum Press, 1977.

Jacobs S, Ostfeld AM: An epidemiological review of the mortality of bereavement. *Psychosom Med* **39**:344–357, 1978

Janowsky DS, El-Yousef MK, Davis JM, et al.: A cholinergic–adrenergic hypothesis of mania and depression. *Lancet* **1**:632–635, 1972.

Janowsky DS, El-Yousef MK, Davis JM: Parasympathetic suppression of manic symptoms of physostigmine. *Arch Gen Psychiatry* **28**:542–547, 1973.

Kaufman IC, Rosenblum LA: The reaction to separation in infant monkeys: Anaclitic depression and conservation–withdrawal. *Psychosom Med* **29**:648–675, 1967.

Kennedy JA, Bakst H: The influence of emotions on the outcome of cardiac surgery: A predictive study. *Bull NY Acad Med* **42**:811–845, 1966.

Koslow SH, Maas JW, Bowden CL, et al.: CSF and urinary biogenic amines and metabolites in depression and mania: A controlled, univariate analysis. *Arch Gen Psychiatry* **40**:999–1010, 1983.

Levovitz BZ, Shekelle RB, Ostfeld AM, et al.: Prospective and retrospective psychological studies of coronary heart disease. Psychosom Med **29**:265–272, 1967.

Lindemann E: Symptomatology and management of acute grief. *Am J Psychiatry* **101**:141–148, 1944.

Maas J: Clinical and biochemical heterogeneity of depressive disorders. *Ann Intern Med* **88**:556–563, 1978.

Motto JA: The recognition and management of the suicidal patient, in Flach FF, Draghi SC (eds): *The Nature and Treatment of Depression.* New York, John Wiley & Sons, 1975, pp. 229–254.

Murphy GE: The physician's responsibility for suicide: 1. An error of commission. *Ann Intern Med* **82**:301–304, 1975a.

Murphy GE: The physician's responsibility for suicide: II. Errors of omission. *Ann Intern Med* **82**:305–309, 1975b.

Neuropsychopharmacology, 1: The Monoamine Systems. Nutley, New Jersey, Roche Laboratories, 1976.

Parkes CM: *Bereavement: Studies of Grief in Adult Life.* New York, International Universities Press, 1972.

Pokorny AD: Suicide in depression, in Fann WE, Karacan I, Pokomy AD, *et al.* (eds): *Phenomenology and Treatment of Depression.* Jamaica, New York, Spectrum Publications, 1977, pp 197–216.

Prange A, Wilson I, Lynn CW, *et al.*: L-Tryptophan in mania: Contribution to a permissive hypothesis of affective disorders. *Arch Gen Psychiatry* **30**:56–62, 1974.

Reiser MF, Bakst H: Psychophysiological and psychodynamic problems of the patient with structural heart disease, in Reiser MF (ed): *American Handbook of Psychiatry,* ed 2. New York, Basic Books, 1975, Vol 4, pp. 618–652.

Robertson J, Bowlby J: Responses of young children to separation from their mothers. *Courr Cent Int Enfence* **2**:131–142, 1952.

Robins E, Gassner S, Kayes J, *et al.*: The communication of suicidal intent: A study of 134 consecutive cases of successful (completed) suicide. *Am J Psychiatry* **115**:724–733, 1959a.

Robins E, Murphy G, Wilkinson R, *et al*: Some clinical considerations in the prevention of suicide based on a study of 134 successful suicides. *Am J Public Health* **49**:888–889, 1959b.

Sachar EJ: Endocrine factors in depressive illness, in Flach FF, Draghi SC (eds): *The Nature and Treatment of Depression.* New York, John Wiley & Sons, 1975, pp 397–411.

Schatzberg AF, Rothschild AJ, Stahl JB, *et al.*: The dexamethasone suppression test: Identification of subtypes of depression. *Am J Psychiatry* **140**:88–91, 1983.

Schildkraut JJ, Kety SS: Biogenic amines and emotion. *Science* **156**:21–30, 1967.

Schmale AH: Giving up as a final common pathway to changes in health. *Adv. Psychosom Med* **8**:20–40, 1972.

Schneidman ES: Suicide in Freedman AM, Kaplan HI, Sacock BJ (eds): *Comprehensive Textbook of Psychiatry, II,* ed 2. Baltimore, Williams & Wilkins, 1975, Vol 2.

Seay B, Harlow HF: Maternal separation in the rhesus monkey. *J Nerv Ment Dis* **140**:434–441, 1965.

Seligman M, Maier S: Failure to escape traumatic shock, *J Exp Psychol* **74**:1–9, 1967.

Silver MA, Bohnert M, Beck A, *et al*: Relation of depression to attempted suicide and seriousness of intent. *Arch Gen Psychiatry* **25**:573–576, 1971.

Spitz R: Anaclitic depression: An inquiry into the genesis of psychiatric conditions in early childhood. *Psychoanal Study Child* **2**:313–342, 1942.

Sweeney DR, Maas JW: Specificity of depressive diseases. *Annu Rev Med* **29**:219–229, 1978.

Tsuang MT, Woolson RF, Fleming JA: Long-term outcome of major psychoses. *Arch Gen Psychiatry* **36**:1295–1301, 1979.

Weissman MM: The epidemiology of suicide attempts, 1960–1971. Arch Gen Psychiatry **30**:737–746, 1974.

Welner A, Marten S, Wochnick E, *et al*: Psychiatric disorders among professional women. *Arch Gen. Psychiatry* **36**:169–173, 1979.

Whybrow P, Mendels J: Toward a biology of depression: Some suggestions from neurophysiology. *Am J psychiatry* **125**:45–54, 1969.

Wolpe J: Neurotic depression: Experimental analog, clinical syndromes, and treatment. *Am J Psychother* **25**:362–368, 1971.

CHAPTER 7

Psychosis

1. A 19-year-old male college student was brought to the emergency room. It was noted that he had become progressively distracted and withdrawn and had suffered from severe insomnia for the past two or three months. Initially, this behavioral change had been thought to be due to the breakup with his girlfriend that had occurred several months earlier. When he started to converse with a nonexistent person in his room, however, and accused his roommate of homosexual advances, his friends persuaded him that he needed help for his "nerves."

2. A 26-year-old married woman was brought to the psychiatric clinic by her husband. Over the past several weeks, she had become irritable and argumentative and had stayed awake most of the night arranging and rearranging furniture. She had several arguments with her husband about her conviction that he was having an extramarital affair even though there was no objective evidence that he was. On psychiatric examination, it was found that for the past two months she had been taking large doses of amphetamines to lose weight. She was a pharmacist and had ready access to the amphetamines.

3. Dear Family Fridori and Family Graf or Schmidli!

Here in this smith-house it doesn't go very well. This is indeed no parish-house or even a poor-house but in this place there is noise, anger, grumbling—sunny—heavenly-knells all year round. Many a small and large landowner, wind-bag or poor drunk from Thalweil, Addisweil, from Albis, from Salz, from Seen, from Rorbach, from Rorbas have never again returned to their own homes, etc., etc., etc.

143

Greetings to all who are still alive.
My own relatives no longer exist.

Anna

[From Eugen Bleuler's Dementia Praecox or The Group of Schizophrenias,
translated by J. Zinkin. New York, International Universities Press, 1950.
Reprinted with permission.]

4. A 65-year-old married salesman became agitated and argumentative in
the surgical recovery room following a cholecystectomy. He accused the
nurses of conspiring to rob him of his property—in fact, he was convinced
that the hospital was a front for organized crime. When his wife came to
see him, he accused her of being in cahoots with the "crooks." He stated
that he actually saw the director of the hospital meeting in his room with
well-known organized crime figures. He claimed that he overheard their
conversation plotting to rob him and kill him.

When the psychiatric consultant examined his medical chart, the onset
of the patient's behavioral problems was found to coincide with the admin-
istration of meperidine for his surgical pain. When the pain medicine was
changed, the behavioral problems subsided rapidly.

Psychosis, Madness, and Insanity

Perhaps everyone recognizes that psychosis is a serious illness, some-
times referred to in lay terms as craziness or madness. We know, how-
ever, that we can all feel crazy sometimes, especially under stress or
under the influence of drugs. In fact, Freud (1900) pointed out that even
normal persons in dreams exhibit thinking processes and engage in be-
haviors that would be considered quite psychotic in the awake state.
Some people deliberately seek experiences resembling psychosis by in-
gesting drugs such as mescaline or LSD. Rosenhan (1973), in an often-
cited study, arranged to have eight apparently normal people present
themselves to a number of different mental hospitals with feigned psy-
chotic symptoms including hallucinations. They were indeed admitted
under the diagnosis of schizophrenia, and even when they behaved
quite normally once admitted, they had some difficulty in being dis-
charged from the hospitals. This study, often cited as an example of
inaccuracy in psychiatric diagnosis, proves quite something else if one
understands the nature of psychotic phenomena—that it is quite pos-
sible for normal people to pretend to be psychotic. Psychosis is often
feigned on the stage and in literature. To an extent, then, crazy behaviors
associated with psychosis may be seen to be a part of the human rep-

ertoire of behaviors. What, then, distinguishes psychosis as an illness from unusual behaviors that occur within the normal range? While normal persons can feign psychosis, and can induce a crazy state by using drugs, they usually have control over whether or not they will be, or act, psychotic. A psychotic *patient*, on the other hand, has no control over his illness unless medical help is given. This does not mean that a psychotic patient will always remain psychotic without treatment; as with any illness, there may be spontaneous exacerbations and remissions of the symptoms. The patient in the midst of a psychotic attack cannot, however, get over being psychotic by simply willing to do so.

General Phenomenology of Psychosis

Psychosis is a psychiatric syndrome in which there are profound and prolonged involuntary alterations in perception, affect, thinking, and behavior (Bowers, 1983). This results in a break in the patient's relationship with reality, often causing socially inappropriate or "crazy" behavior. Characteristic perceptual changes in psychosis are hallucinations and illusions. Hallucinations are perceptual experiences without any external sensory input, while illusions refer to distorted perceptions of external stimuli. For example, in vignettes 1 and 4, there is clear evidence of auditory and visual hallucinations, respectively.

Common aberrations in thought in psychosis include delusions. Delusions refer to aberrations in content of thought, e.g., fixed ideas that are not based on reality, such as delusions of persecution (vignette 4) and delusions of infidelity (vignette 2). Delusions of grandeur are also common in psychotic states associated with mania (e.g., vignette 2 in Chapter 6). Delusions are usually "logic-tight"; that is, no amount of logic will convince a delusional patient that his ideas are not true. In terms of thought *process*, psychotic patients often manifest abnormalities in formal thought process, e.g., blocking, difficulties with *associations*—the linking of one idea to another. "Loose associations" are commonly associated with schizophrenia and other psychotic states (vignette 3).

The behavioral changes that occur in psychosis reflect the patient's perceptual and cognitive state; the patient may follow a hallucinated command, or his behavior may be socially inappropriate or bizarre due to delusions or impairment of reality testing, as mentioned earlier. In fact, this impairment of the individual's relationship with reality is often considered to be the hallmark of the syndrome of psychosis. The motoric behavior of the patient may also be grossly abnormal in a psychotic state. Agitation is common, but on occasion, the patient may present with

catatonia, characterized by mutism, unresponsiveness, and rigidity. When the physician changes the patient's posture by, for example, lifting up his arm, the arm remains in the new position ("waxy flexibility"). The vital signs are usually normal in this state. This *catatonic syndrome* has been regarded as typical of a form of schizophrenia, but many other conditions and diseases can give rise to many of the features of catatonia (as in Table 6).

There are phenomenological differences between the acute psychotic state and the chronic phase of psychosis, although they both share the basic features described above. The sensorium in acute psychosis is typically hyperalert (Bowers, 1974). The behavior tends to be more agitated and floridly disruptive, and the patients are exquisitely sensitive to external stimuli, which may be interpreted in a highly personalized way (e.g., the sound of the ambulance is an indication that "the aliens are coming to get me"). In chronic psychosis, on the other hand, the patient's behavior tends to be more subdued despite the presence of delusions and hallucinations. In chronic schizophrenia, the so-called negative symptoms, such as anhedonia and paucity of thought, may predominate. It should be noted, however, that there may be episodes of florid psychosis in chronically psychotic patients.

We should, at this point, review the phenomenology and etiological theories of schizophrenia in some detail, since acquaintance with what is known about schizophrenia is essential in the evaluation and management of patients with psychosis.

Schizophrenia—A Classic Psychosis

Schizophrenia is, perhaps, the illness that most people think of when the term "psychosis" is mentioned. Schizophrenia is indeed a representative chronic psychotic illness, and most psychosis research has been conducted with the goal in mind of understanding and treating schizophrenia.

Brief History

Descriptions of psychotic phenomena date back to ancient times, but systematic investigations concerning psychosis had to wait until the 19th century, when a number of psychiatrists made systematic attempts at describing and classifying the chronic psychosis that is today known as schizophrenia.

Table 6. List of Conditions Often Associated with the Catatonic Syndrome[a]

Psychiatric disorders
  Schizophrenia
  Affective disorders
  Neuroses and related phenomena
Neurological disorders
  Basal ganglia (following bilateral surgical lesions of the globus pallidus)
  Limbic system and temporal lobes
    Akinetic mutism
    Focal temporal abnormalities
  Diencephalon
    Tumors and traumatic hemorrhage in the region of the third ventricle
    Focal lesions of the thalamus
  Other brain lesions
    Frontal lobe tumors
    Focal frontal lobe lesions
    Anterior cerebral artery aneurysm
    Arterial malformation of the posterior circulation
    Diffuse brain trauma
    Diffuse encephalomalacia following closed head injury
    Petit mal status
    Postictal phase of epilepsy
    Wernicke's encephalopathy
    Tuberous sclerosis
    General paresis
    Narcolepsy
    Acute phase of encephalitis lethargica
    Cerebral macular degeneration
Metabolic conditions
  Diabetic ketoacidosis
  Hypercalcemia from parathyroid adenoma
  Pellagra
  Acute intermittent porphyria
  Homocystinuria
  Membranous glomerulonephritis
  Hepatic encephalopathy
Toxic agents
  Organic fluorides
  Illuminating gas
  Psychotomimetic drugs
  Chronic amphetamine intoxication
  Phencyclidine (PCP) intoxication
Pharmacological agents
  Aspirin intoxication
  ACTH
  High-potency antipsychotic agents

[a] After Gelenberg (1976). Reproduced with permission.

Morel (1860), a French psychiatrist, coined the term *demence precoce* in 1856 to describe an adolescent patient who became gradually withdrawn, gloomy, and silent.

The German psychiatrist Emil Kraepelin in 1896 systematically described in his book *Psychiatry, 5th Edition,* under the rubric "Dementia praecox," the signs and symptoms of psychotic illnesses that seemed to have a course characterized by progressive deterioration in mental functioning. Dementia praecox, which means "premature losing of mind" as opposed to senile dementia, included "catatonia," described some years earlier by another German psychiatrist, Kahlbaum, and "hebephrenia," a form of psychosis that usually occurs in adolescents and young adults and in which severe regression, giggling, posturing, and other inappropriate "silly" behaviors are prominent symptoms, described by still another German psychiatrist, Hecker. According to Kraepelin, then, progressive deterioration was the hallmark of dementia praecox. He thus differentiated dementia praecox from manic–depressive illness, which did not result in a progressive deterioration (see Chapter 6 for further discussion of manic–depressive illness).

Kraepelin paid particular attention to systematic data-gathering and describing the symptoms and natural course of psychiatric illnesses. This systematic *descriptive approach* in psychiatry, often referred to as the Kraepelinian approach, forms a major foundation of modern psychiatry, side by side with the *dynamic approach* developed by Sigmund Freud.

Eugen Bleuler, a Swiss psychiatrist, radically changed the concept of dementia praecox by pointing out that not all patients with the symptoms described by Kraepelin actually had an unremitting deteriorating course. He coined the term *schizophrenia,* which means "split mind," in his book *Dementia Praecox or the Group of Schizophrenias,* published in Germany in 1911. Bleuler proposed that in schizophrenia, there are four fundamental symptoms: disturbances in (1) association and (2) affect and the presence of severe (3) autism and (4) ambivalence. These are the "4 A's" that are still used as useful diagnostic indicators of schizophrenia. Bleuler proposed that other symptoms such as hallucinations, delusions, illusions, and motoric changes (e.g., catatonia) were all accessory or secondary symptoms of schizophrenia. Bleuler was influenced by both Kraepelin and Freud, and his work represents a synthesis of both approaches.

Definition

Schizophrenia is a chronic psychotic disorder of unknown etiology. The essential features of schizophrenia, according to the DSM III, are

the presence of the psychotic syndrome during the active phase of the illness, characteristic symptoms involving multiple psychological processes, and deterioration from a previous level of functioning. The DSM III also defines schizophrenia as an illness that has its onset before the age of 45 and a duration of at least 6 weeks.

Clinical Features and Diagnostic Criteria

The DSM III diagnostic criteria for schizophrenia are listed in Table 7.

Schneiderian First Rank Symptoms and Bleuler's 4 A's. Kurt Schneider (1959) described characteristics symptoms of schizophrenia, which he called the "first rank symptoms." They include complete auditory hallucinations such as hearing two voices conversing with each other, voices commenting on one's behavior, hearing one's thoughts spoken aloud, feeling that one's thoughts are taken away by others (thought withdrawal), feeling that others are putting thoughts into one's head (thought insertion), feeling that one's thoughts are spread to others (thought broadcasting), and delusions of control, such as the belief that one's actions are controlled by radio waves coming from spaceships. The presence of even one of the Schneiderian first rank symptoms in a patient warrants a serious consideration of the diagnosis of schizophrenia.

The important features of the Schneiderian first rank symptoms are auditory hallucinations and delusions. These symptoms are related to Bleuler's *autism* (one of the 4 A's) or dereistic thinking, which means that the patient lives in a world of his own with tenuous relationship with external reality, a world determined primarily by inner psychic drives and stimuli. The patient usually withdraws emotionally and physically from social relationships, being preoccupied with unrealistic fantasies and illogical thoughts. In fact, family members or friends often notice that the patient seems to have withdrawn into a world of his or her own.

Another important feature of schizophrenia is what is known as "formal thought disorder," which means that the form or process of thinking, as opposed to the content of thought, is impaired. The most common example of such a thought disorder is *loosening of associations* of ideas, another of Bleuler's 4 A's. When associations are loosened, the connection between one idea and the next is impaired, so that the patient may skip from one topic to a completely unrelated topic without any

Table 7. DSM III Diagnostic Criteria for a Schizophrenic Disorder[a]

A. At least one of the following during a phase of the illness:
   1. Bizarre delusions (content is patently absurd and has no possible basis in fact), such as delusions of being controlled, thought broadcasting, thought insertion, or thought withdrawal.
   2. Somatic, grandiose, religious, nihilistic, or other delusions without persecutory or jealous content.
   3. Delusions with persecutory or jealous content if accompanied by hallucinations of any type.
   4. Auditory hallucinations in which either a voice keeps up a running commentary on the individual's behavior or thoughts or two or more voices converse with each other.
   5. Auditory hallucinations on several occasions with content of more than one or two words, having no apparent relation to depression or elation.
   6. Incoherence, marked loosening of associations, markedly illogical thinking, or marked poverty of content of speech if associated with at least one of the following:
      a. Blunted, flat, or inappropriate affect
      b. Delusions or hallucinations
      c. Catatonic or other grossly disorganized behavior
B. Deterioration from a previous level of functioning in such areas as work, social relations, and self-care.
C. Duration: Continuous signs of the illness for at least six months at some time during the person's life, with some signs of the illness at present. The six-month period must include an active phase during which there were symptoms from A, with or without a prodromal or residual phase, as defined below.
   Prodromal phase: A clear deterioration in functioning before the active phase of the illness not due to a disturbance in mood or to a Substance Use Disorder and involving at least two of the symptoms noted below.
   Residual phase: Persistence, following the active phase of the illness, of at least two of the symptoms noted below, not due to a disturbance in mood or to a Substance Use Disorder: Prodromal or residual symptoms
   1. Social isolation or withdrawal
   2. Marked impairment in role functioning as wage-earner, student, or homemaker
   3. Markedly peculiar behavior (e.g., collecting garbage, talking to self in public, or hoarding food)
   4. Marked impairment in personal hygiene and grooming
   5. Blunted, flat, or inappropriate affect
   6. Digressive, vague, overelaborate, circumstantial, or metaphorical speech
   7. Odd or bizarre ideation, or magical thinking, e.g., superstitiousness, clairvoyance, telepathy, "sixth sense," "others can feel my feelings," overvalued ideas, ideas of reference
   8. Unusual perceptual experiences, e.g., recurrent illusions, sensing the presence of a force or person not actually present
   Examples: Six months of prodromal symptoms with one week of symptoms from A; no prodromal symptoms with six months of symptoms from A; no prodromal symptoms with two weeks of symptoms from A and six months of residual symptoms; six months of symptoms from A, apparently followed by several years of complete remission, with one week of symptoms in A in current episode.
D. The full depressive or manic syndrome (criteria A and B of major depressive or manic episode), if present, developed after any psychotic symptoms, or was brief in duration relative to the duration of the psychotic symptoms in A.
E. Onset of prodromal or active phase of the illness before the age of 45.
F. Not due to any Organic Mental Disorder or Mental Retardation.

[a] Reprinted with permission from the American Psychiatric Association.

seeming awareness that he is talking about unrelated subjects. When a patient's associations are loose, his speech may be incoherent (vignette 3). An extreme form of loose associations is sometimes called "word salad." In addition to loose associations, the schizophrenic patient may show stilted language, neologisms (making up of new words), perseveration (repetition of the same words or ideas), clang associations (words that are linked by the sound rather than the meaning, e.g., "I drank milk, and silk, and ilk, and ink, and dink, and think.") Thought blocking is also sometimes seen, in which the patient is completely without any thoughts in the middle of an ongoing statement or conversation.

Another common symptom of schizophrenia, and one of Bleuler's 4 A's, is the *blunting* or *flattening of affect*. Flat affect denotes a marked reduction in the intensity of affective expression, so that there is practically no evidence of expression; blunting means a less severe reduction in affective expression. With flat affect, the patient usually speaks in a monotone (if he speaks at all), and the facial muscles appear immobile. Inappropriate affect may also be exhibited in schizophrenia; for example, the patient may giggle while talking about a sad event, such as the death of his mother.

Sense of self is often impaired in schizophrenia, such that a sense of individuality is lost. Problems with sense of identity are common. This is sometimes referred to as a loss of *ego boundaries*.

Volition, or self-initiated, goal-directed activity, is often impaired in schizophrenia. This may be associated with severe *ambivalence* regarding decision-making (this is the last of Bleuler's 4 A's). The patient may show lack of interest in activities, lack of enjoyment (anhedonia), and lack of motivation.

Positive and Negative Symptoms. The symptoms of schizophrenia may be classified into "positive" and "negative" ones (Andreasen and Olsen, 1982). The positive symptoms are the florid psychotic symptoms such as hallucinations and delusions that are commonly shared by all psychoses. The negative symptoms, on the other hand, are "deficit" symptoms that are particularly pronounced during the chronic or residual phase of the illness. They include flatness of affect, paucity of thought, anhedonia, and motoric retardation.

On the basis of symptomatology and drug-therapy response, Crow (1980) proposed that there might be two types of schizophrenia: *Type I* is characterized by mostly "positive" symptoms, probably dopamine-sensitive, and therapeutic response to antipsychotic drugs (dopamine-receptor blockers) is good (see the section on Neurotransmitters). *Type II* schizophrenia, on the other hand, is characterized by a predominance

of the "negative symptoms," is dopamine-insensitive, and treatment outcome with antipsychotic drugs is poor. The "negative" symptoms have been associated with cerebral ventricular enlargement on computerized tomography (Andreasen *et al.*, 1982).

Clinical Types. On the basis of the predominant clinical picture, schizophrenia may be subdivided into the types described below. It should be emphasized, however, that over the course of time, the predominant presentation may change. Antipsychotic drugs may also alter the clinical picture as the "positive" symptoms respond better than the "negative" ones.

1. Disorganized (hebephrenic) type: Characterized by marked incoherence and blunted, inappropriate, or silly affect. Posturing and odd behavior are prominent. The illness typically begins in adolescence.
2. Catatonic type: Characterized by marked psychomotor disturbances, which may include catatonia, rigidity, stupor, excitement, negativism, and posturing (see also Table 6).
3. Paranoid type: Characterized by delusions of a persecutory or grandiose nature; delusional jealousy or hallucinations with persecutory or grandiose content may also occur. Generally, the onset of psychosis in this type is later than in other types of schizophrenia (late 20s to 30s). Functional deterioration in this type is usually milder than in other types of schizophrenia.
4. Undifferentiated type: Characterized by a mixed picture, such as delusions, hallucinations, and incoherence or disorganized behavior.
5. Residual type: This diagnosis is given to patients who have had at least one episode of schizophrenia and who are not floridly psychotic although certain symptoms persist, such as blunting of affect, social withdrawal, looseness of associations, and illogical thinking.

Course and Prognosis

Schizophrenia is a chronic illness, but the course is not necessarily progressively downhill as it was considered to be in Kraepelin's time. The advent of antipsychotic drugs has improved the prognosis greatly, so that with continuing therapy, reasonably satisfactory degrees of functioning can be maintained in many patients for long periods of time.

There is, however, usually some deterioration in the functional capacity. The family's expectations of a patient may have to be lowered once schizophrenia has developed.

There are several factors that seem to be associated with the *prognosis* for functional adjustment of patients with schizophrenia. Factors that are associated with a good prognosis include: absence of premorbid personality disturbance, good level of premorbid social functioning, clear-cut precipitating events, abrupt onset, onset in mid-life, presence of confusion in the clinical picture, and a family history of affective disorder (after DSM III). A particularly poor prognostic indicator is early insidious onset with predominant "negative" symptoms.

Mortality

Psychiatric patients in general have a higher mortality rate than the general population. Affective disorders (mania and depression) and schizophrenia are particularly implicated in an increased mortality rate. In a 30 to 40-year follow-up study of patients with various psychiatric and nonpsychiatric disorders in Iowa, Tsuang and Woolson (1978) found that an excess mortality rate existed for both schizophrenia and affective disorders. While the excess mortality was most pronounced during the first 10 years following hospitalization (2–4 times the control population), the increased mortality rate continued throughout the study period in schizophrenia. While the rate of suicide and accidents are increased in schizophrenia these do not completely account for the excess mortality rate.

Epidemiological Aspects

Approximately 100,000 new schizophrenic patients receive care each year in the United States. At any given time, about 0.5–3.0% of the population seems to be afflicted with schizophrenia according to various community surveys (Strauss and Carpenter, 1981).

The lifetime prevalence rate based on studies in Europe and Asia that used a relatively narrow definition of schizophrenia is 0.2–1%.

Sex ratio: Schizophrenia is equally common among males and females.

Socioeconomic status: Schizophrenia is more prevalent in lower socioeconomic populations. Faris and Dunham (1939) found in their classic study in Chicago that the highest first-hospitalization rates for schizo-

phrenia occurred in the poor, inner-city areas. Hollingshead and Redlich (1958) found in New Haven that the prevalence rates of psychotic disorders were higher in the lower socioeconomic classes, while the prevalence rates of neurotic disorders (anxiety disorders) were higher in the upper socioeconomic classes.

These socioeconomic differences in the prevalence of schizophrenia may be explained by two possibilities: (1) the *origin hypothesis* postulates that the disorganized urban ghetto environment and genetic pooling of the low socioeconomic populations give rise to the genetic and environmental conditions that cause schizophrenia. (2) the *drift hypothesis* postulates that the individuals afflicted with schizophrenia drift downward in socioeconomic status and living environment to the more impoverished areas.

Etiological Aspects

Schizophrenia is a disorder of as yet undetermined etiology, but there are considerable relevant data and many theories about aspects of its etiology.

Biological Dimension Genetic factors: Schizophrenia clearly runs in families. The risk of developing schizophrenia in the general population is somewhat less than 1%, while the prevalence for parents of children who are known schizophrenics is 12%. The morbidity risk for schizophrenia for full siblings of schizophrenic patients is 13–14%. The risk for children with one schizophrenic parent is 8–18%. If both parents are schizophrenic, the morbidity risk for their children may be as high as 50% (Rosenthal, 1968). In the case of twins, heterozygous twins have the same risk as other siblings, while homozygous (identical) twins have a concordance rate for schizophrenia of approximately 50% (However, there is much variability in the concordance rate depending on the study, from practically 0 to 86%.) The high rate of concordance for monozygotic twins suggests a strong genetic component in schizophrenia, but the lack of absolute concordance also proves that there are factors other than genetic ones that operate in the development of the schizophrenic syndrome.

Studies of adopted children contributed to an understanding of genetic factors in schizophrenia. In general, there seems to be an increased incidence of prevalence or schizophrenia in biological relatives of chronic schizophrenic patients, but not in their adoptive relatives. However,

acute schizophrenia was not increased in these biological relatives (Gottesman and Shields, 1976; Kety *et al.*, 1968).

Smooth-pursuit eye-tracking impairment has been reported in schizophrenic patients and their nonschizophrenic relatives. This abnormality was reported to be associated with social introversion and has been suggested as a genetic marker of those who are vulnerable to schizophrenia (Holzman *et al.*, 1974; Siever *et al.*, 1982).

There are undoubtedly genetic factors that contribute to a vulnerability to schizophrenia, but the nature of the vulnerability is not yet clearly understood.

Neuroanatomical and neurophysiological considerations: Disorders in arousal (hyperarousal in acute psychosis, hypoarousal in the insidious or chronic phase) and inability to sustain attention are common in schizophrenic patients. Disturbances in the emotional life (mood and affect) also characterize most schizophrenics. From these manifestations, it is reasonable to assume that neurophysiological abnormalities in the reticular activating and limbic systems are involved in schizophrenia (see Chapter 6 for a discussion of these brain areas). Some studies have demonstrated an association between certain psychophysiological measures of brain function and treatment outcome with antipsychotic drugs (Landau *et al.*, 1975; Strauss and Carpenter, 1981). Unwanted and irrelevant memories and thoughts often intrude uncontrollably into the patient's consciousness. Such phenomena may also represent a disturbance of the "filtering" function of the reticular activating system concerning extraneous stimuli. At times of such extraneous sensory inundations, the patient may be behaviorally mute and catatonic. The EEG, however, often shows a pattern of hyperarousal (low-voltage fast activity) during these episodes.

Other phenomena often seen in schizophrenia, such as feelings of unreality about the environment (*derealization*) or concerning the self (*depersonalization*), experiences of thought insertion and withdrawal, hallucinations, and disturbances of arousal and attention, all bear some resemblance to symptoms displayed in seizure disorders (epilepsy) involving the limbic brain ("partial complex seizures" or "limbic" or "temporal lobe" seizures). Chemically induced seizures of the amygdaloid nucleus have been shown to produce chronic "psychotic" behaviors in cats, such as apparent hallucinations, inappropriate fear and attack responses, and hypervigilance (Stevens, 1973).

Ventricular enlargement on computerized tomography (CT scan) has been reported in some schizophrenic patients. There is evidence that such ventricular enlargement may be associated with the "negative" or "deficit" symptoms of schizophrenia rather than the "positive" symp-

toms (Andreasen *et al.*, 1982). On the basis of these findings, Andreasen and her colleagues hypothesize that there might be two types of schizophrenia: a "negative" or "defect" schizophrenia characterized by the "negative" signs, and caused by diffuse atrophic changes in the brain, and a "positive" schizophrenia, caused by more focal brain dysfunction involving neurotransmitters, with a predominance of the "positive" symptoms. There is evidence, however, that both "positive" and "negative" symptoms exist in many patients depending on the phase of the schizophrenic illness.

Positron emission tomography (PET scan) tends to show decreased frontal lobe activity in some schizophrenic patients (Buchsbaum *et al.*, 1982).

Neurological "soft signs" (such as awkwardness in the pronation and supination of the arm) have been reported in some schizophrenic patients, particularly those with premorbid asociality (Quitkin *et al.*, 1976). The presence of these signs is thought to indicate the presence of diffuse and general brain damage, as in "minimal brain dysfunction syndrome." There is also evidence on CT scan that the normal asymmetry in brain structures may be reversed more often in schizophrenic patients than in normals, particularly in the frontal and occipital lobes (Luchins *et al.*, 1979). Such findings seem consistent with theories that postulate disturbances in the right–left brain specialization in schizophrenia, e.g., that disturbance of logical thought in patients with schizophrenia may indicate a dysfunction of the dominant (usually left) hemisphere (Flor-Henry, 1976).

In summary, then, there is evidence suggesting disordered function of the limbic system in schizophrenia, and in some chronic schizophrenic patients with predominant "negative" symptoms, there may be diffuse atrophic changes in the brain and these may be associated with "soft" neurological signs.

Biochemical factors: The idea that biochemical factors are involved in schizophrenia receives strong support from a number of important facts. They include (1) certain drugs can cause clinical psychotic states that are in many ways indistinguishable from some schizophrenic psychotic states (e.g., the amphetamines); (2) different chemical classes of drugs that are effective in ameliorating many of the serious symptoms of schizophrenia share common pharmacological features (e.g., ability to block postsynaptic dopamine receptors; see below); and (3) when genetic factors are involved in transmission of vulnerability to an illness, as in schizophrenia, the mediating mechanisms operate through biochemical pathways.

Drug-induced psychosis. Altered states of consciousness induced by the use of marijuana were reported over a thousand years ago in India,

and Aztec Indians in Mexico used the peyote plant (which contains the psychotomimetic drug mescaline) as a part of their religious ceremonies (Snyder, 1974).

There are several classes of drugs that commonly cause psychosis—the hallucinogens (psychedelic drugs) and CNS stimulants. The hallucinogens include LSD, PCP, and mescaline; D-amphetamine and cocaine are examples of CNS stimulants. Drug-induced psychoses have been observed with high frequency in both medical and psychiatric settings since the 1960s, when self-administration of psychedelic drugs became popular throughout the world.

Some hallucinogens are structurally related to endogenous substances. For example, mescaline is chemically similar to norepinephrine. The *transmethylation theory* of schizophrenia postulates that certain hallucinogens such as bufotenine and DMT (*N,N*-dimethyltryptamine) are endogenously produced in some schizophrenic patients by abnormal methylation of biogenic amines (Osmond and Smythies, 1952; Rosengarten and Friedhoff, 1976). The enzymes required to produce such transmethylated hallucinogens have been found in the human brain, and the administration of methyl donors such as methionine that would enhance such reactions have been shown to exacerbate psychotic symptoms in some known schizophrenic patients. According to the transmethylation hypothesis, the administration of methyl acceptors such as nicotinic acid should reduce schizophrenic symptoms. The results of experiments designed to test this hypothesis are conflicting and indeterminate.

In general, psychoses caused by drugs are acute psychoses with hyperalertness and severe anxiety. In many instances, they resemble the acute stage of schizophrenia (Bowers and Freedman, 1966). Amphetamine psychosis is almost indistinguishable from paranoid schizophrenia, and may be caused by the abuse of "diet pills," as in vignette 2. Drug-induced psychoses typically lack, however, the "negative" symptoms of schizophrenia and the chronic course.

Neurotransmitters. With the advent of antipsychotic drugs, a new biochemical theory emerged—the *dopamine theory of schizophrenia*. All drugs that were effective against schizophrenia (and psychosis) were shown to be blockers of the brain neurotransmitter dopamine (Snyder, 1974), while dopamine agonists like amphetamine and L-dopa often provoke psychosis. Phenothiazines (e.g., chlorpromazine) and butyrophenones (e.g., haloperidol) are examples of antipsychotic drugs that are potent dopamine antagonists. Usually, there is a general correlation between a drug's dopamine-receptor-blocking activity as manifested by extrapyramidal side effects (e.g., parkinsonian tremor and cogwheel rig-

idity caused by the deficiency of functional levels of dopamine in the substantia nigra) and its antipsychotic potency. One clinically useful indication of a drug's dopamine-antagonist activity rests on the fact that dopamine inhibits prolactin secretion by the pituitary; hence, a drug's ability to increase serum prolactin levels has been used as a screening test for potential new antipsychotic agents. However, as we will see later in this chapter, the locations of dopamine receptors that are important in psychoses (mesolimbic system) are not identical with those that are involved in the extrapyramidal symptoms (nigrostriatal tract) or in prolactin secretion (dopamine tract within the hypothalamus). Thus, these side effects should not be inescapable when more specific antipsychotic drugs are invented and developed.

There are some reports of finding direct evidence of increase in brain dopamine and dopamine receptors in the brains of schizophrenic patients on postmortem examination (Mackay et al., 1978). The increase in dopamine levels was not related to the amount of antipsychotic drugs used, but the increase in the number of receptors was seen only in patients who had been maintained on antipsychotic drugs for a long time (Creese and Snyder, 1982). Recently, bimodal distribution of dopamine receptors in postmortem brains of schizophrenics has been reported, a finding that is compatible with the notion that there are two types of schizophrenia (Seeman et al., 1984).

There are at least four types of dopamine receptors in the brain. The D1 but not the D2 receptors stimulate adenylate cyclase activity. The D2 receptors have been shown to be increased in schizophrenic brains by 50–100% (Lee and Seeman, 1977, 1980).

However, schizophrenia cannot be explained simply on the basis of increased functional levels of dopamine in the brain. Antipsychotic drugs are not uniformly effective in all schizophrenics; in fact, there is a subgroup of patients who show little or no response to dopamine-receptor blockers [dopamine-insensitive or type II of Crow (1980)]. In fact, the "negative" symptoms of schizophrenia are the most resistant to the antipsychotic drugs. Studies on dopamine turnover in the brain have so far been equivocal (Haracz, 1982). Also, it is clear that an aberration of the dopamine system is not specific for schizophrenia as opposed to other psychoses, since almost all psychotic states, regardless of cause, seem to respond to the dopamine-receptor-blocking agents.

Neurotransmitters other than dopamine have also been implicated in schizophrenia. For example, decreased serotonergic function has been suggested (Bartholini et al., 1968; Bowers, 1972).

Despite these problems, however, it is reasonable to regard dysfunction in brain dopaminergic neurons as playing an important part

in schizophrenic psychoses. The specific dopaminergic system hypothesized to be involved (hyperfunctional) in schizophrenia is the mesolimbic system, which originates from the melanin-containing cells in midbrain tegmentum (ventral tegmental nucleus of Tsai) and terminates on small interneurons of the limbic striatum in the region of the nucleus accumbens (or accombens), olfactory tubercle, amygdala, and caudate nucleus [see Figure 11 (Chapter 6)].

Janice Stevens (1973) postulated, on the basis of phenomenological, neuroanatomical, and pharmacological data, that there may be dopaminergic and serotonergic push–pull servosystems in the limbic striatum that may act as a "gate" between limbic inputs and perception–behavior outputs. According to her proposal, the interneurons of the limbic striatum, on which the predominantly cholinergic axons from neocortex and limbic cortex converge, may be concerned with the selection or gating of endogenous and exogenous stimuli that compete for access to conscious attention and for access to autonomic endocrine functions and to neural pathways that control behavior. The threshold of these normally hyperpolarized limbic interneurons, according to this theory, may be influenced by the reciprocal actions of the ascending dopaminergic and serotonergic neurons. Schizophrenic psychosis, then, could result from a chronic disorder in the aminergic regulation of this "limbic striatal filter," with some of the symptoms representing "release phenomena."

Other substances. Monoamine oxidase: There are reports of decreased levels of the enzyme monoamine oxidase (MAO) in the blood platelets of schizophrenic patients (Wyatt *et al.*, 1980). First-degree relatives of schizophrenics have also been reported to have lower levels of MAO activity (Berrettini *et al.*, 1980), but lower platelet MAO activity has been reported in other psychiatric disorders as well, including affective disorders and alcoholism. Low MAO activity may represent a genetic marker of nonspecific vulnerability to psychiatric disorders.

Endorphins: Endorphins seem to be involved in the regulation of dopamine and serotonin synthesis. The exact nature of involvement, if any, of the endorphin system in schizophrenia is as yet undetermined (Bowers, 1980).

Prostaglandins: Disorders of prostaglandins have also been reported, but the data so far are contradictory (Horrobin, 1977; Kafka *et al.*, 1979; Bowers, 1980).

Creatine phosphokinase: The clinical significance of the increased levels of serum creatine phosphokinase (CPK) that have been reported in schizophrenia (Meltzer, 1976) is unclear.

Immune mechanisms and viral agents. Increased levels of serum immunoglobulins G and M have been reported in schizophrenia, and the

possibility that schizophrenia is an autoimmune disease has been suggested (Burch *et al.*, 1968; Amkraut *et al.*, 1973). Virus-like agents have reportedly been found in the cerebrospinal fluid of schizophrenics (Tyrrell *et al.*, 1979; Crow *et al.*, 1979). Atypical lymphocytes that show stimulated features have been found in schizophrenics ("P" cells); these P cells do not seem to be related to neuroleptic medication (Hirata-Hibi *et al.*, 1982). Although the evidence of immunological changes is intriguing, no definite immunological mechanisms or viral agents have been identified as specific to schizophrenia.

Personal Dimension. Problems in cognitive and affective development may also contribute to predisposition and vulnerability to schizophrenia. In terms of the personality structure, abnormalities of *ego functions*, particularly cognitive systems, are prominent in schizophrenia (and other psychoses). From a developmental perspective, *regression* to earlier, infantile states of cognition and affect regulation characterize schizophrenic symptomatology. Evidence of regression includes illogical thought process (*primary process thinking*), massive denial of reality, autism, and magical thinking. Some severely regressed schizophrenic patients may even assume the fetal position for long periods of time.

In general, psychological difficulties implicated in theories of etiology and pathogenesis concerning schizophrenia involve three types of theories and their combinations: *deficit theories, conflict theories,* and *maladaptive learning theories.*

Deficit theories postulate that some inborn psychological defects may be precursors of later illness. Conflict theories propose that certain early conflicts, for example, in the oral phase of development, cause a fixation in development, and that the individual may then regress back to that stage of cognition and function when confronted with severely stressful situations in (later) life. Maladaptive learning theories propose that schizophrenic behavior may be based on abnormal interpersonal behaviors and patterned thinking learned over time (Strauss and Carpenter, 1981). Lidz *et al.* (1965) emphasize the role of abnormal family patterns of interaction in shaping behaviors that will later be manifest as a part of schizophrenic psychopathology.

Studies of high-risk children (such as the offspring of schizophrenics) do indicate that premorbid cognitive and behavioral defects are present even in childhood. For example, problems with attention and reaction time to stimuli, problems with activity–passivity, and poor social relationships are common in children who later become schizophrenic (Chapman, 1979; Strauss *et al.*, 1977).

There is considerable evidence to support the fact that major psychological conflicts associated with stressful life situations may precipitate an acute schizophrenic psychosis (Bowers, 1974) regardless of the nature of the etiological factors that predispose to it.

In summary, then, we might state that in patients with schizophrenia, various combinations of psychological deficits, learned maladaptive behaviors, and intrapsychic conflicts along with biological and environmental factors may have contributed to the development of the illness. The precise sequence and nature of the interactions among these factors are not known, and their relative weights may vary in different patients and in patients with different clinical forms and stages of the disorder.

Environmental Dimension. Environmental factors that have been postulated to be associated with the pathogenesis of schizophrenia include abnormal family environment and the effect of socioeconomic milieu.

As discussed previously, the fact that the prevalence of schizophrenia is higher in the urban ghetto environment gave rise to the hypothesis that the stressful environment in these areas might be a causative factor in schizophrenia (Faris and Dunham, 1939). This finding is in part supported by the fact that the prevalence of the illness is higher in nonwhites (who predominate in urban ghetto areas) as compared to whites (Kramer, 1978). Caution is needed in interpreting such epidemiological data, since there is good evidence that schizophrenic patients drift down in socioeconomic status, and also the gene pool in the underprivileged sector may contribute to the vulnerability.

Family studies illuminate the nature of developmental factors that contribute to development of schizophrenia. *Patterns of communication* have been reported to be abnormal in schizophrenic families. The *double bind* described by Bateson *et al.* (1956) is an example of a communication deviance that if consistently characteristic of most transactions between adults and child could lead to difficulty. In it, the child is put in a "no-win" situation by the parents. For example, a mother buys two neckties for her son, one red, the other blue. When the mother sees her son wearing the red tie, she asks him, "What's the matter, don't you like the blue tie?"

If the child is exposed consistently and over time to such no-win situations, psychosis might offer the only or best "way out." Wynne *et al.* (1977) described typical patterns of communication deviance in schizophrenic families, including failure to arrive at a closure, premature closure, and lack of "visualizability." They postulated that these types of communication problems would compromise children's ability to deal

conceptually with life's demands. Wynne *et al.* (1958) also described a psychological "rubber fence" that surrounds certain families of schizophrenics. Such a family isolates itself from others and provides little real contact for the child with the outside world, and the communication pattern of such a family may be "pseudomutual" (a special form of communication whereby agreement seems to occur without actual communication of concepts or facts).

Lidz *et al.* (1965) described two characteristic patterns found in families of patients with schizophrenia, *skewed* and *schismatic*. In the skewed family, one member of the family who has abnormal behavior patterns dominates the interactional patterns and sets an abnormal "norm" of behavior. In the schismatic family, the parents are severely split. Each parent is involved in hostile competition for the attention and affection of the child. Such abnormal family environments, when unremittingly consistent, can be considered to be detrimental to normal psychosocial development and to create personality structures conducive to the development of schizophrenia.

Controlled empirical studies of families of patients with schizophrenia have demonstrated that high levels of expressed emotion in family interactions, particularly high levels of expressed hostility and aggression, correlate with increased probability of psychotic relapse of schizophrenic patients who are in remission (Brown *et al.*, 1972; Vaughn and Leff, 1976).

In summary, schizophrenia seems to be an illness in which biological, psychological, and environmental factors may all contribute to predisposition, vulnerability, and precipitation.

Dysfunction (hyperfunction) of the mesolimbic dopaminergic pathway may be a final common pathway in schizophrenic psychosis. Additionally, there might also be structural changes in the brains of schizophrenics who manifest predominantly "negative" symptoms.

Nature of Psychosis

We mentioned that drug-induced "model" psychosis gave rise to biochemical theories concerning schizophrenia. Many features of psychoses that are clearly nonschizophrenic, such as those induced by amphetamines, are indistinguishable from schizophrenia. Drugs that are effective in schizophrenia, e.g., the dopamine-receptor blockers, are also effective in many psychoses other than schizophrenia. Thus, theories concerning the biochemical disorder underlying psychotic symptoms in schizophrenia apply as well to practically all psychotic conditions. In

fact, antipsychotic drugs such as the phenothiazines are used widely in the treatment of many acute psychotic states including mania and psychotic organic brain syndromes.

As might be expected, the "positive" or florid symptoms of schizophrenia that are sensitive to antipsychotic drugs, such as hallucinations and delusions, are symptoms that are commonly found in psychoses other than schizophrenia. "Negative" symptoms such as asociality and poverty of thought that are often resistant to antipsychotic drugs are the ones that seem to be more specifically encountered in schizophrenia. All this suggests that psychosis as a syndrome may be a *final common pathway* for expression of a number of underlying disorders including the antecedent genetic and psychological traits that characterize individuals who are predisposed and vulnerable to develop schizophrenia.

Bowers (1974) postulated that the central nervous system possesses a latent capacity for a pattern of functioning that is experientially known as human psychotic consciousness. This postulated neurobiologically altered mode may be "switched on" under certain circumstances, the most notable of which is drug ingestion. This altered mode may also be transiently operational during dreaming, and under sensory deprivation conditions, when hallucinations naturally occur.

Many factors may lower the threshold for the state-change to psychosis. Genetic and personality developmental factors would be long-term factors also encountered within psychiatric disorders such as affective disorders, borderline personality disorder, and drug dependence. Changes in physical and social environment may also contribute to a psychotic break, as in sensory deprivation (such as in intensive care units), and at times of severe interpersonal stress. Changes in the internal environment also contribute to psychosis, as in toxic states due to disease or drugs [e.g., endocrine diseases, neurological diseases; see Table 16 (Chapter 12)].

In conclusion, then, psychosis may be considered to be a stress-related *final common pathway* of the brain that functions in response to and expresses a variety of predisposing and contributing biological, psychological, and environmental factors.

Evaluation of Psychosis

Psychosis is not an uncommon phenomenon among hospitalized patients, even in medical and surgical units. Most commonly, such psychoses are due to drug toxicity (such as narcotic analgesics) or drug withdrawal, impaired circulation to the brain, or a combination of or-

ganic and environmental factors such as sensory deprivation and overload of the intensive care unit, drugs, and sleep deprivation.

It is also important to recognize that patients who *present* with psychotic symptoms to a psychiatrist may have an underlying medical or surgical disease rather than schizophrenia or another psychiatric disorder. Common among diseases that present with psychotic symptoms are endocrinopathies, neurological diseases, and metabolic diseases (Jefferson and Marshall, 1981; Leigh and Kramer, 1984).

Evaluation of the psychotic syndrome, then, must be comprehensive and include the biological, psychological, and environmental dimensions of the patient. Comprehensive evaluation of the patient naturally leads to a differential diagnosis and to an understanding of the interrelated predisposing, contributing, and precipitating factors of the psychotic syndrome.

Biological Dimension

The most important considerations in the biological dimension include the presence or absence of toxic states and medical or neurological diseases (current context). Physical and neurological examination, laboratory tests (including toxic screen), and careful medical history are obviously important. For example, evidence of sympathetic hyperactivity with increased blood pressure and the presence of amphetamines in the urine would establish the diagnosis of amphetamine psychosis (vignette 2).

Toxic psychosis is often caused by commonly used anticholinergic agents such as atropine. Signs of sympathetic overactivity and anticholinergic activity (e.g., tachycardia, dry, hot skin, mydriasis) should be present. Anticholinergic psychosis may be temporarily reversed by the intravenous administration of physostigmine (a cholinesterase inhibitor).

EEG and CT scan may yield valuable data in making the diagnosis of viral encephalitis or a space-occupying lesion causing psychosis.

In the recent context, a history of self-administered drug use or abuse (analgesics, alcohol, and hypnotics) or of medications will give clues concerning toxic or withdrawal psychoses, e.g., LSD- or PCP-induced toxic states or propranolol- or digitalis-induced psychosis or alcohol or barbiturate withdrawal.

Family history of schizophrenia or bipolar affective disorder increases the possibility that the patient may have schizophrenia or manic psychosis, respectively.

Personal Dimension

The current mental status of the patient establishes the presence of the psychotic state through such features as hallucinations, delusions, loosening of associations, and illogical thinking. The type of hallucination is often important in distinguishing schizophrenia from organic psychosis. Visual hallucinations of the unformed type are particularly indicative of organic psychosis, while auditory hallucinations are more common in schizophrenia.

Cognitive aspects of the mental-status examination often provide clues concerning the etiology of the psychotic syndrome. Disorientation and confusion are typically seen in psychoses associated with organic brain syndrome rather than schizophrenia. Careful evaluation concerning possible toxic states, neurological conditions, and other medical diseases is called for in the presence of these symptoms.

It is also important to inquire into the history of the psychotic syndrome—onset (insidious or acute), clinical features (e.g., gradual social withdrawal, predominantly paranoid flavor), and change in functioning. The presence of characteristic clinical features of schizophrenia will help establish the diagnosis, as well as possibly give clues to the prognosis (e.g., poor prognosis in the asocial, "negative" schizophrenia).

In the background context, the premorbid personality often provides clues concerning the cause of the psychosis; the withdrawn, introverted premorbid personality is more common with schizophrenia, while a person with cyclothymic personality may develop a psychotic manic syndrome. Persons who are characterized by extreme lability in emotions and behavior and stormy relationships may occasionally develop transient psychotic episodes that seem to "seal off" spontaneously. Such patients may have *borderline personality disorder.*

There are patients who manifest both severe depression or mania *and* features of schizophrenia. These patients are classified as having *schizoaffective disorder.*

Environmental Dimension

The current social and physical environment of the patient may provide important clues concerning the causes of the psychotic syndrome. For example, a teen-ager who is in a drug culture is most likely to have a drug-induced psychosis. A cardiac patient in the coronary care unit is most likely to have an *intensive care unit syndrome* or a toxic psy-

chosis due to analgesics or antiarrhythmic drugs. A person living alone in progressive social isolation is more likely to have schizophrenia.

Recent changes in the environment, such as breaking up with a boyfriend or girlfriend, may have precipitated a situational psychosis or an acute schizophrenic psychosis (with a better prognosis than in "negative" schizophrenia with gradual insidious onset and no precipitating factors).

Early background information concerning family relationships may reveal patterns suggesting a predisposition to psychosis such as "skewed" or "schismatic" parental relationships or consistent exposure to "double bind."

Differential Diagnosis of Psychosis

On the basis of a comprehensive evaluation of the psychotic syndrome, a differential diagnosis should be performed to make appropriate plans for managing the patient.

As in the case of the depressive syndrome, it is important to distinguish between psychosis secondary to a specific cause and what might be called primary (or idiopathic) disorders, e.g., psychoses that include schizophrenia, schizoaffective disorder, and affective disorders (as well as "schizophreniform psychosis" with unknown etiology and "paranoid disorder," in which there are no associated signs of schizophrenia).

The secondary psychoses are diagnosed on the basis of an identifiable etiological factor, such as drugs, medical disease, stimulus deprivation, or major situational stress.

The primary psychoses are diagnosed first by ruling out secondary psychoses, but should also be supported by the symptomatology, clinical course, and background information such as family history of schizophrenia (see Table 7).

Management of Psychosis

A comprehensive evaluation of the psychotic patient should lead to a comprehensive management strategy. Removal of the causative agent in secondary psychosis is, of course, the most important consideration. However, if the patient is acutely psychotic and agitated, immediate management of the psychotic behavior may be necessary before the causative agent can be identified and eliminated.

The treatment of psychosis should be done in collaboration with a

psychiatrist, who can provide expertise in psychotherapy, environmental management, and pharmacotherapy. Once schizophrenia or any other chronic primary psychosis has been diagnosed, referral to a psychiatrist may be advisable, since protracted and ongoing specialized treatment will generally be necessary.

Biological Dimension

As previously discussed, antipsychotic drugs are effective in almost all kinds of psychoses and can be effectively used for symptomatic treatment of both primary and secondary psychoses. In general, the dose of the antipsychotic drug may have to be much higher to treat schizophrenia as compared to secondary psychoses.

Typically, the antipsychotic drugs are phenothiazines (e.g., chlorpromazine, perphenazine, fluphenazine) or butyrophenones (e.g., haloperidol). The drugs should be titrated to control the psychotic symptoms (particularly the "positive" symptoms such as hallucinations and delusions). In acutely psychotic, disruptive patients, parenteral administration may be necessary, since they may be unable to take oral medications. Chlorpromazine, 25 mg intramuscularly may be given every 4 hr, or for less sedation, haloperidol, 5 mg intramuscularly every 4 hr may be tried until the disruptive symptoms are under control. Schizophrenic patients may require maintenance doses for protracted periods of time to prevent relapse. It should be remembered, however, that embarking on a long-term course of antipsychotic drugs for a patient is a serious business.

Tardive dyskinesia, which has been associated with long-term use of antipsychotics, is a movement disorder consisting of involuntary writhing and jerking movements in the orobuccal region that spread to the rest of the body. This often irreversible condition is probably caused by dopamine-receptor supersensitivity induced by long-term dopaminergic blockade.

The choice of antipsychotic drugs depends to a large extent on the side effects of the drugs. The common side effects that are important include: sedation, orthostatic hypotension, extrapyramidal effects (pseudoparkinsonism), photosensitivity, and occasional jaundice. Agranulocytosis is a rare but serious side effect. Different drugs have different patterns and degrees of these side effects, as discussed in Chapter 20.

Personal Dimension

Supportive psychotherapy in combination with drug therapy as outlined above is the treatment of choice in schizophrenia or any chronic

psychosis (Leigh, 1983). Psychotherapy is geared to help maintain the patient's contact with reality and to provide a regular relationship with an accepting figure who will monitor the patient's symptoms and level of social function and the effect of medications. Additionally, it may offer psychosocial rehabilitation and support for further emotional growth once the acute phase is passed and a good therapeutic relationship has been established.

Reality-oriented counseling to solve immediate problems of living, such as the living situation (e.g., home vs. halfway house), is frequently necessary. The frequency and locus of psychotherapeutic sessions should be individualized. Usually, during the acute phase, hospitalization is necessary with daily contact. On discharge, patients usually require at least weekly sessions until they are stabilized and reasonably rehabilitated. Then the frequency may be reduced to once every few months with the understanding that the patient may contact the physician at any time when necessary.

Even in cases of secondary psychosis, psychotherapy is often needed in addition to the removal of the causative agent to help the patient integrate the frightening effects of the psychotic experience.

Long-term insight-oriented psychotherapy may be helpful after the acute episodes. It is highly specialized treatment and should be used only by experts in carefully selected cases, usually from among patients refractory to combined short-term hospital, drug, and supportive psychotherapy management (Fromm-Reichmann, 1948). The degree of regression and autism and the "negative" symptoms often preclude intensive psychotherapy during the acute phase of schizophrenic psychosis, but consistent presence of the therapist during this phase may set the stage for more active treatment later.

Environmental Dimension

Acutely psychotic patients are typically in a state of fear and hypervigilance. They experience and react to internal and external stimuli in a highly exaggerated fashion. Thus, it is important to provide, as much as possible, an environment of low stimuli for such patients before adequate evaluation can be performed (Bowers, 1983). A relatively dimly lit, quiet room with adequate space is an optimal setting for interviewing such a patient. The room should be free of potentially dangerous visible objects, such as scalpels and scissors, and the door should be visible to the patient with ready access and left open to reassure the patient.

On the other hand, it is equally important for the evaluating physician to feel secure. If indicated because of violent behavior, security officers may need to be called to restrain the patient before any evaluation can take place.

In secondary psychosis due to pathogenic influences in the immediate environment, as in the intensive care unit, transfer to another unit may be considered. In psychosis associated with organic brain syndrome (see Chapter 8), a multibed room with a calendar and night lights may be therapeutic in reducing the disorientation and confusion that may contribute to misinterpretations of environmental stimuli. Frequent visits from family members, familiar objects, and photographs of close relatives help the patient keep a hold on reality.

Hospitalization is perhaps the most important treatment in the environmental dimension for acutely psychotic patients. The indications for hospitalization include grossly disruptive behavior or violent, homicidal, suicidal, or self-destructive behavior or potential.

Family evaluation and therapy is particularly important in younger, "first-break" schizophrenics. In some cases, abnormal interactional patterns of the family may be identified and corrective measures may be instituted, which may reduce the pathogenic influence on the patient. More important, education of family members concerning the chronic nature of schizophrenia and its treatment modalities will enhance their understanding of the patient, which most patients sorely need, and ensure both the family's and the patient's cooperation and collaboration.

Summary

Psychosis is a syndrome characterized by involuntary alterations in perception, thinking, and behavior. Some important symptoms include hallucinations, delusions, loosening of associations, and agitation or catatonia.

Many drugs, toxic states, and medical and neurological diseases are associated with psychosis.

Schizophrenia, which affects about 1% of the population during their lifetime, is but one type of chronic psychotic syndrome with remissions and exacerbations. Autism, association problems, affective flattening, and severe ambivalence causing problems with intentional behavior (Bleuler's 4 A's) have been described as characteristic symptomatology. Auditory hallucinations, delusions, and experiences of thought insertion, thought withdrawal, thought broadcasting, and thought control are also considered to be characteristic. Genetic, developmental, psy-

chological, and environmental factors have been identified in its path-
ogenesis. The "positive" (or florid) symptoms of schizophrenia seem to
be more responsive to antipsychotic drugs than the "negative" (or def-
icit) symptoms. Disturbances in the limbic brain, particularly in the *meso-
limbic dopaminergic system*, seem to be associated with the psychotic
symptoms in schizophrenia.

Psychosis may be conceptualized to be a *final common pathway* of
altered consciousness the neurobiological alteration of which may be in
the mesolimbic dopaminergic system. Heredity, early family interac-
tions, and psychological conflicts may provide varying degrees of pre-
disposition, and severe developmental stressors including interpersonal
relationships and endocrine changes (as in adolescence) may be precip-
itating factors. Drugs or endogenous toxins may precipitate psychosis
even in persons with little constitutional predisposition to psychosis.

The *evaluation* of psychosis should consider the symptomatology,
onset, and associated biological and environmental factors such as the
possibility of drugs or toxins and pathogenic environments. Since
acutely psychotic patients are fearful and hypervigilant, a quiet, secure,
and tranquilizing environment is necessary for optimal evaluation. Be-
fore primary psychiatric disorder (or idiopathic psychosis) is diagnosed,
the possibility of secondary psychosis should be considered. In second-
ary psychosis, treatment of the underlying cause is crucial, although
symptomatic treatment with antipsychotic drugs may also be necessary
initially before the primary cause can be alleviated.

Follow-up psychotherapy to reintegrate the psychotic experience
with the patient's ongoing life may also be needed in managing the
patient with a secondary psychosis.

In schizophrenia or other chronic primary psychosis, antipsychotic
drugs in combination with supportive psychotherapy are indicated on
a long-term basis. Education of the family is necessary, as well as their
collaboration with the therapist whenever possible. An important con-
sideration in the environmental dimension concerning the psychotic pa-
tient is *hospitalization*, which is often necessary during an acute episode
to control disruptive, suicidal, homicidal, or otherwise self-destructive
behavior.

Implications

For the Patient

Psychosis is a frightening experience for the patient. In secondary
psychosis, it is important to be certain that the patient understands the

fact that the psychotic symptoms are a part of the medical condition, drug side effect, or other cause and that he/she is not "going crazy." In schizophrenia and other primary psychosis, the patients are usually isolated from others, mistreated, and misunderstood. Thus, the availability of the physician who understands the patient's experiences sets the stage for a long-term trusting and collaborative relationship. Education of the patient concerning the nature of his illness, proposed treatment, and the effects and possible side effects of medications will help allay irrational fears and prevent a rupture in the therapeutic relationship should any side effects occur.

For the Physician

There is a tendency for general physicians to consider all psychotic behavior to be exclusively in the domain of the psychiatrist. They should recognize that many medical and neurological diseases, drugs and their withdrawal states, and various metabolic toxic states as well as sensory deprivation can cause psychosis, especially in susceptible individuals. A comprehensive evaluation is necessary in psychosis as in depression or anxiety syndromes. Consultation with a psychiatrist concerning the evaluation, differential diagnosis, and management of the psychotic patient is usually advisable, but in the case of secondary psychosis, collaborative management with the primary physician is essential. The physician should be aware of the principles of management of the acutely psychotic patient as might be encountered in many primary-care settings as well as in the emergency room.

For the Community and the Health-Care System

The chronically psychotic patient has been historically stigmatized and isolated from the community. While this trend has been reversed in more recent years through "deinstitutionalization" of chronic patients from state hospitals, there are inadequate facilities for the patients who require long-term residential or protected treatment setting. Education of society in general is needed to provide humane treatment settings for those who require protection and to destigmatize those who have been well rehabilitated through combined effective drug therapy and psychosocial intervention and thus are able to be useful members of the community. It is imperative that society generate and implement plans to correct the problems presented by the growing population of chron-

ically psychotic "street people." At the level of social policy, plans should now be in progress to cope with the increasing numbers of schizophrenic patients that can be anticipated from the apparently increasing incidence rate (Kramer, 1978). Medical schools and hospital training programs should educate future physicians and health-care personnel in comprehensive evaluation and initial management of the psychotic syndrome.

Recommended Reading

Bowers MB. Jr: Retreat from Sanity—The Structure of Emerging Psychosis New York, Human Sciences Press. 1974. This book describes the experiences of a number of patients who develop psychoses. A very readable book with vivid descriptions of the phenomena of psychosis with thought-provoking discussions.

Leigh H (ed): *Psychiatry in the Practice of Medicine*. Menlo Park, California, Addison-Wesley Publishing Co, 1983. Chapter 7, Evaluation and management of psychosis, by Malcolm Bowers, Jr, and Chapter 8, Schizophrenia, by Hoyle Leigh contain practical information concerning the clinical aspects of psychosis and schizophrenia. These chapters complement the basic information provided in this book.

Leigh H, Kramer SI: The psychiatric manifestations of endocrine disease. *Adv Intern Med* **29**:413–445, 1984. An up-to-date review of the psychiatric manifestations of endocrine and metabolic diseases. These manifestations include psychosis, anxiety, depression, and personality change, among others.

Lidz T, Fleck S, Cornelison AR: *Schizophrenia and the Family*. New York, International Universities Press, 1965. This book describes the classic Yale study on the families of schizophrenia that led to the identification of "skewed" and "schismatic" families.

Stevens JR: An anatomy of schizophrenia? *Arch Gen Psychiatry* **29**:177–189, 1973. A good review of literature concerning the neurobiology and phenomenology of schizophrenia leading up to the author's conclusions that schizophrenia involves a disorder of a hypothesized dopaminergic and serotonergic "push–pull" servosystem in the limbic striatum.

Strauss JS, Carpenter WT Jr: Schizophrenia. New York, Plenum Press, 1981. A readable and concise book summarizing information concerning various aspects of schizophrenia, including epidemiology, etiology, course, and treatment.

References

Amkraut AP, Solomon GF, Allansmith M, *et al.*: Immuno-globulins and improvement in acute schizophrenic reactions. *Arch Gen Psychiatry* **28**:673–677, 1973.

Andreasen NC, Olsen, S: Negative vs. positive schizophrenia. *Arch Gen Psychiatry* **39**:789–794, 1982.

Andreasen NC, Olsen SA, Dennert JW, *et al.*: Ventricular enlargement in schizophrenia: Relationship to positive and negative symptoms. *Am J Psychiatry* **139**:297–302, 1982.

Bartholini G, Da Prada M, Pletscher A: Decrease of cerebral 5-hydroxytryptamine by 3,4-dihydroxyphenylalanine after inhibition of extracerebral decarboxylase. *J Pharm Pharmacol* **20**:228–229, 1968.

Bateson G, Jackson D, Haley J, et al.: Towards a theory of schizophrenia. Behav Sci 1:251–264, 1956.

Berrettini WH, Benfield TC, Schmidt AO, et al.: Platelet monoamine oxidase in families of chronic schizophrenics. Schizophrenia Bull 6:235–237, 1980.

Bowers MB Jr: Acute psychosis induced by psychotomimetic drug abuse: Neurochemical findings. Arch Gen Psychiatry 27:440–442, 1972.

Bowers MB Jr: Retreat from Sanity—The Structure of Emerging Psychosis New York, Human Sciences Press, 1974.

Bowers MB Jr: Biochemical processes in schizophrenia: An update. NIMH Special Report, Schizophrenia, 1980, pp 27–34.

Bowers MB Jr: Evaluation and management of psychosis, in Leigh H (ed): Psychiatry in the Practice of Medicine. Menlo Park, California, Addison-Wesley Publishing Co, 1983, pp 103–112.

Bowers MB Jr, Freedman DX: Psychedelic experiences in acute psychosis. Arch Gen Psychiatry 125:240–248, 1966.

Brown GW, Birley JLT, Wing JK: Influence of family life on the course of schizophrenic disorders: A replication. Br J Psychiatry 121:241–258, 1972.

Buchsbaum MS, Ingvar DH, Kessler R, et al.: Cerebral glucography with positron tomography: Use in normal subjects and in patients with schizophrenia. Arch Gen Psychiatry 39:251–259, 1982.

Burch PRJ, Rowell NR, Burwell RG: Schizophrenia: Auto-immune or auto-aggressive? Br Med J 2:50, 1968.

Chapman LJ: Recent advances in the study of schizophrenic cognition. Schizophrenia Bull 5:568–580, 1979.

Creese I, Snyder SH: Increased brain dopamine and dopamine receptors in schizophrenia. Arch Gen Psychiatry 39:991–997, 1982.

Crow TJ: Molecular pathology of schizophrenia: More than one disease process? Br Med J 280:66–68, 1980.

Crow TJ, Ferrier IN, Johnstone EC, et al.: Characteristics of patients with schizophrenia or neurological disorder and virus-like agent in cerebrospinal fluid. Lancet 1:842–844, 1979.

Faris REL, Dunham HW: Mental Disorders in Urban Areas. Chicago, University of Chicago Press, 1939.

Flor-Henry P: Lateralized temporal–limbic dysfunction and psychopathology. Ann NY Acad Sci 280:777–795, 1976.

Freud S: The Interpretation of Dreams, Strachey J (ed). New York, Avon Books, 1965 (originally published, 1900).

Fromm-Reichmann F: Notes on the development of treatment of schizophrenia by psychoanalytic psychotherapy. Psychiatry 11:263–273, 1948.

Gelenberg AJ: The catatonic syndrome. Lancet 1:1339–1341, 1976.

Gottesman II, Shields J: A critical review of recent adoption, twin, and family studies of schizophrenia: Behavioral genetics perspectives. Schizophrenia Bull 2:360–401, 1976.

Haracz JL: The dopamine hypothesis: An overview of studies with schizophrenic patients. Schizophrenia Bull 8:438–469, 1982.

Hirata-Hibi M, Higashi S, Tachibana T, et al.: Stimulated lymphocytes in schizophrenia. Arch Gen Psychiatry 39:82–87, 1982.

Hollingshead AB, Redlich FC: Social Class and Mental Illness. New York, Wiley, 1958.

Holzman PS, Proctor LR, Levy DL, et al.: Eye tracking dysfunctions in schizophrenic patients and their relatives. Arch Gen Psychiatry 31:143–151, 1974.

Horrobin DF: Schizophrenia as a prostaglandin deficiency disease. Lancet 1:936–937, 1977.

Jefferson JW, Marshall JR: *Neuropsychiatric Features of Medical Disorders*. New York, Plenum Press, 1981.

Kafka MS, Van Kammen DP, Bunney WE: Reduced cyclic AMP production in the blood platelets from schizophrenic patients. *Am J Psychiatry* **136**:685–687, 1979.

Kety SS, Rosenthal D, Wender PH, *et al.*: The types and prevalence of mental illness in the biological and adoptive families of adopted schizophrenics, in Rosenthal D, Ketty SS (eds): *The Transmission of Schizophrenia*. Oxford, Pergamon Press, 1968, pp 345–362.

Kramer M: Population changes and schizophrenia, 1970–1985, in Wynne LC, Cromwell RL, Matthysse S (eds): *The Nature of Schizophrenia: New Approaches to Research and Treatment*. New York, Wiley, 1978, pp 545–571.

Landau SG, Buchsbaum MS, Carpenter WT, *et al.*: Schizophrenia and stimulus intensity control. *Arch Gen Psychiatry* **32**:1239–1245, 1975.

Lee T, Seeman P: Dopamine receptors in normal and schizophrenic human brains. *Soc Neurosci Abstr* **3**:443, 1977.

Lee T, Seeman P: Elevation of brain neuroleptic/dopamine receptors in schizophrenia. *Am J Psychiatry* **137**:191–197, 1980.

Leigh H: Schizophrenia, in Leigh H (ed): *Psychiatry in the Practice of Medicine*. Menlo Park, California, Addison-Wesley Publishing Co, 1983, pp 113–121.

Leigh H, Kramer SI: The psychiatric manifestations of endocrine disease. *Adv Intern Med* **29**:413–445, 1984.

Lidz T, Fleck S, Cornelison AR: *Schizophrenia and the Family*. New York, International Universities Press, 1965.

Luchins DJ, Weinberger DR, Wyatt RJ: Schizophrenia. *Arch Gen Psychiatry* **36**:1309–1311, 1979.

Mackay AVP, Doble A, Bird E, et al.: 3H-Spiperone binding in normal and schizophrenic postmortem human brain. *Life Sci* **23**:527–532, 1978.

Meltzer HI: Neuromuscular dysfunction in schizophrenia. *Schizophrenia Bull* **2**:106–146, 1976.

Morel BA: *Traite des Maladies Mentales*. Paris, Victor Masson, 1860.

Osmond H, Smythies J: Schizophrenia: A new approach. *J Ment Sci* **98**:309–315, 1952.

Quitkin F, Rifkin A, Klein DF: Neurologic soft signs in schizophrenia and character disorders. *Arch Gen Psychiatry* **33**:845–853, 1976.

Rosengarten H, Friedhoff AJ: A review of recent studies of the biosynthesis and excretion of hallucinogens formed by methylation of neurotransmitters or related substances. *Schizophrenia Bull* **2**:90–105, 1976.

Rosenhan DL: On being sane in insane places. *Science* **197**:250–252, 1973.

Rosenthal D: The heredity–environment issue in schizophrenia: Summary of the constant and present status of our knowledge, in Rosenthal D, Kety SS (eds): *The Transmission of Schizophrenia*. London, Pergamon Press, 1968, p 413.

Schneider K: *Clinical Psychopathology*, New York, Grune & Stratton, 1959.

Seeman P, Ulpian C, Bergeron C, *et al.*: Bimodal distribution of dopamine receptor densities in brains of schizophrenics. *Science* **225**:728–731, 1984.

Siever LJ, Haier RJ, Coursey RD, *et al.*: Smooth pursuit eye tracking impairment. *Arch Gen Psychiatry* **39**:1001–1005, 1982.

Snyder, SH: *Madness and the Brain*. New York, McGraw-Hill, 1974.

Stevens JR: An anatomy of schizophrenia? *Arch Gen Psychiatry* **29**:177–189, 1973.

Strauss JS, Carpenter WT Jr: *Schizophrenia*. New York, Plenum Press, 1981.

Strauss JS, Kokes RF, Klorman R, *et al.*: Premorbid adjustment in schizophrenia: Concepts, measures, and implications. *Schizophrenia Bull* **3**(2):182–244, 1977.

Tsuang MT, Woolson RF: Excess mortality in schizophrenia and affective disorders: Do suicides and accidental deaths solely account for this excess? *Arch Gen Psychiatry* **35**:1181–1185, 1978.

Tyrrell DAJ, Parry RP, Crow TJ, *et al.*: Possible virus in schizophrenia and some neurological disorders. *Lancet* **1**:839–841, 1979.

Vaughn CE, Leff JP: The influence of family and social factors on the course of psychiatric illness: A comparison of schizophrenic and depressed neurotic patients. *Br J Psychiatry* **129**:125–137, 1976.

Wyatt RJ, Potkin SG, Bridge TP *et al.*: Monoamine oxidase in schizophrenia: An overview. *Schizophrenia Bull* **6**:199–207, 1980.

Wynne L, Rickoff I, Day J, *et al.*: Pseudomutuality in the family relations of schizophrenics. *Psychiatry* **21**:205–220, 1958.

Wynne LC, Singer MT, Barko J, *et al.*: Schizophrenics and their families: recent research on parental communication, In Tanner JM (ed): *Developments in Psychiatric Research.* London, Hodder & Stoughton, 1977, pp 254–286.

Confusion, Delirium, and Dementia: Organic Brain Syndromes and the Elderly Patient

1. A 42-year-old man had an uneventful cholecystectomy in the hospital. He was given meperidine, 75 mg, intramuscularly for postoperative pain. Several hours following the injection, he became confused and assaultive and accused the nursing staff of poisoning him. He was also observed to converse loudly with an invisible figure in the room. There was no past or family history of psychiatric disorder concerning this patient.

2. A 70-year-old man was brought to the emergency room in a coma. The patient lived alone in an apartment. He had been hospitalized two weeks ago for congestive heart failure, for which a digitalis preparation was pre-scribed. He had been discharged one week ago, with one month's supply of Digoxin tablets, one to be taken every day. A nearly empty bottle of Digoxin was found at his bedside when he was discovered comatose by his neighbor.

3. A 17-year-old woman was admitted to the hospital in acute renal failure. At the time of admission, she seemed to be quite drowsy. When the resident physician attempted to take her history, she was confused and disoriented. She kept referring to the doctor as "waiter," and seemed to think she was in a restaurant.

4. A 78-year-old man was admitted to a nursing home. The admission be-came necessary when the patient's son, with whom he had been living for

the past five years since the death of his wife, was transferred to another state. At the time of admission, the son told the nursing home staff that he had noticed his father to be forgetful in the past two years, but that he had no behavioral problems whatsoever. He had been able to do work in the garden and to go to the grocery store, and he seemed to be, in general, content. He had agreed to go to the nursing home rather than attempt to move with the son's family to an unfamiliar state. On the third day of admission, however, the patient became confused and agitated, and his behavior became grossly inappropriate. For example, he urinated and defecated in his room, and tried to grab and kiss a nurse. Diazepam, 5 mg, was given intramuscularly, and the psychiatric consultant was called.

5. A 67-year-old widow was brought to the physician by her daughter. The patient had been living in a housing project for the elderly since the death of her husband 7 years ago. Her daughter, who visited her every week, had noticed over the past four weeks that her mother was becoming apathetic, forgetful, and withdrawn. For example, she was unable to tell her daughter what she had done during the previous day. She also seemed to be inattentive, often not responding to her daughter's questions. The daughter arranged for a psychiatric consultation for her mother.

See also the vignettes in Chapters 12 and 13.

In the previous chapters on anxiety, affect, and psychosis, we examined the final common pathway syndromes presumptively associated with the limbic system, hypothalamus, and other areas of the brain stem. In this chapter, we will discuss the organic brain syndromes of delirium and dementia, which are final common pathway syndromes associated with global brain dysfunction or general cerebrocortical dysfunction or both.

Definitions and Phenomenology

Organic Brain Syndrome

Organic brain syndrome refers to a constellation of behavioral and psychological symptoms and signs that reflect reversible or irreversible brain dysfunction, particularly in the areas of orientation, attention, concentration, memory, logical thinking, abstraction, judgment, and affect (DSM III, 1980, p 101). In fact, Engel and Romano (1959) defined organic brain syndrome as a syndrome of cerebral insufficiency.

Delirium

Delirium is an organic brain syndrome in which clouding of consciousness (or reduction in awareness of the environment) is a prominent feature. The patient is disoriented (see Chapter 12), with memory deficits (particularly recent memory), and major difficulties with attention and concentration are seen. Perceptual disturbances are also common, such as illusions, hallucinations, and misinterpretations of stimuli. Speech may be at times incoherent, and disturbances of sleep–wakefulness cycle are common, such as insomnia or daytime drowsiness. There may be agitation or psychomotor retardation (see vignettes 1 and 3). Delirium usually develops over a relatively short period (hours to days), and one of its outstanding features is that level of consciousness tends to fluctuate markedly during the course of a day; periods of lucidity often alternate with confusion and disorientation.

Dementia

Dementia is an organic brain syndrome in which there is an acquired loss of intellectual abilities to the extent that it interferes with social or occupational functioning. Also prominent are memory loss and impairment of abstract thinking and judgment. Personality changes and changes in behavioral patterns are also common. Neurological signs of brain dysfunction may be present, such as aphasia (language dysfunction), apraxia (inability to carry out motor activities despite intact comprehension and motor apparatus), and agnosia (inability to recognize objects in the presence of intact sensory apparatus). Dementia generally has an insidious onset, but the mode of onset depends largely on the underlying disease. For example, the onset of dementia secondary to a head trauma may be rapid. In pure dementia, there is no clouding of consciousness. However, delirium and dementia often do coexist.

Organic brain syndromes include, in addition to delirium and dementia, amnestic syndrome (loss of memory is the prominent feature), organic hallucinosis (hallucinations are the prominent feature), and organic personality change (prominent personality change in the absence of dementia).

Epidemiology of Organic Brain Syndromes

Organic brain syndromes are perhaps the most common psychiatric conditions with which a general physician deals in his or her daily prac-

tice. In the general hospital, metabolic and electrolyte changes that may accompany acute and chronic illness, surgery, and administration of drugs all predispose and contribute to delirium.

Approximately 5–15% of patients on medical or surgical inpatient units are thought to manifest evidence of delirium (Lipowski, 1980; Titchener *et al.*, 1956). The incidence of delirium in surgical intensive care units has been reported to be 18–30% and in coronary care units, 2–20% (Lipowski, 1980). Approximately 30% of the 20- to 70-year-old population is expected to experience an episode of delirium within their lifetimes (Lipowski, 1967).

Dementia is prevalent among the elderly. Approximately 4–5% of the Northern European population aged 65 years and older were reported to have severe dementia and 11–12% to have mild to moderate dementia (Katzman, 1976; Terry, 1976a). In one study, 17% of an elderly community population was reported to have evidence of dementia (Fisch *et al.*, 1968).

In the general hospital, many elderly patients with mild dementia may develop superimposed delirium due to metabolic changes associated with disease.

Pathophysiology of Organic Brain Syndromes

By definition, organic brain syndrome denotes (potentially) demonstrable brain dysfunction.

In the case of delirium, the dysfunction is predominantly caused by a metabolic derangement of the neurons due to a change in the extracellular environment, i.e., presence of toxic substances such as drugs, hypoxia, abnormal metabolites, or electrolyte imbalance. Since these chemical or electrolyte abnormalities usually affect the whole brain, the resulting symptoms include global changes such as clouding of consciousness. The EEG often shows a general slowing as compared to premorbid tracings. In severe delirium, delta waves may be prominent (see Chapter 10).

Dementia is generally considered to result from widespread destruction of neurons, particularly in the cerebral cortex. Thus, cortical atrophy, widening of the sulci, and enlarged ventricles are common findings on computerized axial tomographic (CAT or CT) scans. The specific lesions depend on the underlying disease (see below), e.g., ischemic lesions in multiinfarct dementia, neurofibrillary tangles, senile plaques, and granulovacuolar bodies in Alzheimer's disease, and mass in brain tumor.

The term *subcortical dementia* has been used to denote dementia associated with lesions of the subcortical structures, such as the basal ganglia, with relative sparing of the cortical neurons. In subcortical dementia, verbal and perceptuomotor abilities may be largely preserved, while there is impairment of memory, abstraction, personality change, and a marked slowing of thought processes. Parkinson's disease, Huntington's chorea, Wilson's disease, and progressive supranuclear palsy are examples of diseases that may cause cortical or subcortical dementia (Albert *et al.*, 1974).

Prevalence of Dementia in the Elderly

Dementia is primarily a disorder of the elderly and, in the past, was considered to be normal in an elderly person. While dementia is the major psychiatric problem in the elderly, it is now known that severe memory loss and confusion are pathological, even in the aged population. About 10% of persons over the age of 65 are estimated to have clinically significant intellectual impairment, based on data from the United Kingdom (Beck *et al.*, 1982). About half of the intellectually impaired population have dementia. In the United States, approximately 1 million people may be afflicted with severe dementia and another 3 million with mild to moderate dementia (Schneck *et al.*, 1982). Dementia is likely to become an increasingly greater problem in the future, since the number of persons over 65 years of age is estimated to increase by 1600 a day or about 600,000 per year in the United States (Beck *et al.*, 1982).

In conclusion, dementia, or intellectual impairment severe enough to interfere with social and occupational function, is a common but abnormal condition in the elderly, affecting 10–20% of that population.

Alzheimer's Disease and Multiinfarct Dementia

In the past, dementia in the elderly was considered to be caused by cerebral arteriosclerosis. Alzheimer's disease (see below) was considered to be a dementing disease of the presenium (below the age of 65). It was shown in the 1960s, however, that there were approximately the same amounts of arteriosclerosis in the brains of both demented and nondemented elderly persons (Corsellis and Evans, 1965). In a study of the brains of 50 demented and 28 nondemented elderly patients, 50% of the dementia brains showed the histological features of Alzheimer's disease without evidence of ischemic lesions. Twelve percent of the de-

mentia brains showed primarily infarct lesions, and another 5% were also associated with cerebrovascular changes. Some 8–18% of the dementia brains were associated with both Alzheimer's changes and infarcts. Ten percent could not be classified, and 5% were secondary to other causes. Histologically and clinically, there is no difference between Alzheimer's disease occurring before or after the age of 65.

Now, the DSM III classifies dementia in the elderly into (1) primary degenerative dementia (Alzheimer's disease), senile or presenile onset, and (2) Multiinfarct dementia. Alzheimer's disease and multiinfarct dementia together comprise approximately 75% of all dementias in the elderly.

Alzheimer's Disease

Primary degenerative dementia, or Alzheimer's disease, is the most common dementing disease of the elderly. The prevalence rate is approximately 6% (0.38–18.2%) (Schneck *et al.*, 1982). Although some report a higher prevalence rate for women than for men, there is controversy concerning the sex ratio for Alzheimer's disease.

Pathology. Grossly, the Alzheimer brain tends to weigh less than the normal brain and often shows generalized cortical atrophy, which may be particularly prominent in the parietal, temporal, and frontal lobes. Ventricular dilatation, even if slight, is always found (Tomlinson *et al.*, 1968, 1970). Significant correlations between the ventricular size measured on the CT scan and the degree of cognitive impairment in early mild to moderate cases of Alzheimer's disease have been reported (DeLeon *et al.*, 1980; Merskey *et al.*, 1980).

While cortical atrophy may seem to indicate diffuse neuronal loss, aging itself causes a loss of cortical neurons, and there seems to be little difference in the neuronal density between the brains of normal aged persons and those of Alzheimer's disease patients (Terry *et al.*, 1977). There seem to be specific areas of the brain in which marked neuronal loss occurs in Alzheimer's disease: the hippocampus, the medial temporal lobe, and the subiculum (Schneck *et al.*, 1982).

The major microscopic abnormalities in Alzheimer's disease are *neurofibrillary tangles, senile plaques,* and *granulovacuolar bodies.*

The neurofibrillary tangles are visualized by reduced silver stain to extend from the cell body into the dendrites, progressively occluding cytoplasmic space previously devoted to protein synthesis and transport (Beck *et al.*, 1982). They are particularly abundant in the cerebral cortex,

especially in the hippocampus [see Figure 4 (Chapter 4)]. Electron microscopy reveals that the neurofibrillary tangles consist of paired helical filaments, twisted linear structures with a half-period of 800 Å and a maximum width of 240 Å (Terry *et al.*, 1964; Terry, 1976b; Wisniewski and Soifer, 1979).

Neurofibrillary tangles are also seen in more than 50% of the brains of normal nonpsychotic, nondemented persons between the ages of 50 and 59 years, in more than 80% of patients aged 60–69, and in 100% of patients over 70 years of age according to one study (Matsuyama and Nakamura, 1978). They have also been found in a variety of brain diseases, including adult Down's syndrome and postencephalitic Parkinson's disease.

It is interesting that neurofibrillary tangles are not found naturally in any other species than man. Injection of aluminum into the brains of animals, however, causes neurofibrillary tangles that are similar but not identical in structure to the neurofibrillary tangles found in man.

Senile plaques consist of multiple, small necrotic foci with amyloid-rich centers surrounded by radial palisades of degenerating axons and dendrites. They are found mostly in the cerebral cortex, particularly in the temporal areas, although they may also be found in the white matter, basal ganglia, brain stem, and cerebellum. The degree of intellectual deterioration in Alzheimer's disease seems to correlate with the number of senile plaques per unit area of the brain (Schneck *et al.*, 1982). Senile plaques also occur in normal aging brains. In Kuru and Creutzfeldt–Jakob disease, both of which are dementing slow-virus diseases, senile plaques are characteristically present in the patients' brains.

The granulovacuolar body or degeneration is characterized by foamy inclusions at soma–dendrite junctions. These inclusions are present in large numbers in the hippocampus of Alzheimer patients. Granulovacuolar bodies are also found in normal elderly brains, but patients with Alzheimer's disease seem to have 2–100 times greater incidence of granulovacuolar degeneration than age-matched controls.

Neurotransmitters in Alzheimer's Disease. Alterations in a number of neurotransmitter levels have been reported in Alzheimer patients. Of particular importance is a reduction in the cholinergic activity in the brain.

Decreased activities of the enzyme choline acetyltransferase (ChAT), the biosynthetic enzyme for acetylcholine, have been reported in autopsied Alzheimer cerebral cortices (Rossor, 1982). ChAT is confined to the cholinergic neurons and is stable after death; thus, this enzyme is a good indicator of the integrity of cholinergic neurons. Acetyl-

cholinesterase activity has also been shown to be decreased in Alzheimer's disease. Biopsy studies of the temporal lobe have directly confirmed ChAT deficit and reduced acetylcholine synthesis in Alzheimer patients (Spillane *et al.*, 1977; Bowen *et al.*, 1979; Sims *et al.*, 1980).

Cholinergic neurons are very widespread in the CNS, including the cerebral cortex, basal ganglia, thalamus, cerebellum, and spinal cord. In Alzheimer's disease, the most marked reductions in the cholinergic activity seem to occur in the temporal cortex, the hippocampus, and the amygdala. The degree of this reduction, which may reach 95% of control values, has been positively correlated with the density of senile plaques and neurofibrillary tangles and the degree of dementia (Beck *et al.*, 1982).

The *cholinergic hypothesis* of Alzheimer's disease and geriatric memory dysfunction (Bartus *et al.*, 1982) postulates that a cholinergic dysfunction underlies the memory loss. Support for this hypothesis comes from several findings: Anticholinergic drugs such as atropine can produce transient memory dysfunction (as well as psychosis), which can be reversed with choline or physostigmine (acetylcholinesterase inhibitor). The administration of physostigmine, or the acetylcholine agonist arecholine, to normal humans and experimental animals can transiently improve some aspects of memory. The administration of choline, or its dietary precursor lecithin, may also transiently improve memory in experimental situations (Bartus *et al.*, 1982; Beck *et al.*, 1982). None of these choline enhancers has yet been demonstrated to be consistently beneficial for the memory impairment in Alzheimer's patients.

Decreased amounts of dopamine, norepinephrine, and somatostatin have been found in the brains of Alzheimer's disease patients (Beck *et al.*, 1982). A reduction of 15–85% in the number of noradrenergic cell bodies in the locus ceruleus has also been reported in Alzheimer's disease.

Etiological Considerations. The etiology of Alzheimer's disease is as yet unknown. Etiological theories include genetic, viral, autoimmune, and toxic theories.

Evidence of *genetic* contribution to Alzheimer's disease comes from two observations: (1) all Down's syndrome (trisomy 21) patients who survive to adulthood eventually develop Alzheimer's disease (and Alzheimer's brain pathology) (Heston, 1979; Sinex and Merril, 1982); (2) Alzheimer's disease seems to run in families (Sinex and Myers, 1982). The genetic mode of transmission of Alzheimer's disease has not been established. Dominant mode with reduced penetrance as well as polygenic models have been proposed for presenile Alzheimer's, while a

model of polygenic heterogeneity seems to be widely accepted for late-onset Alzheimer's disease (Schneck *et al.*, 1982).

A *viral* etiology for Alzheimer's disease may be suspected from the findings that (1) many viral encephalitides cause late-onset dementia (e.g., subacute sclerosing panencephalitis, which is presumed to be a sequela of measles virus infection, and dementia associated with post-encephalitic Parkinson's disease) and (2) Creutzfeldt–Jakob disease and Kuru, both of which are caused by slow-viruses, have clinical and histological features similar to those of Alzheimer's disease. When the brains of chimpanzees were injected with tissues from the diseased brains of two patients with familial Alzheimer's disease, a chronic, progressive neurological illness ensued in these animals, perhaps demonstrating the transmission of Alzheimer's disease (Crapper and De Boni, 1979).

Autoimmune mechanisms have been postulated for Alzheimer's disease on the basis of antibrain antibodies (Tkach and Hokama, 1970) in Alzheimer patients and in aging rats. Also, the immunological abnormalities associated with Down's syndrome (reduced T-cell function) may also be implicated in Alzheimer's disease. There is, however, no conclusive evidence concerning an immunological etiology of Alzheimer's disease.

An intriguing etiological hypothesis for Alzheimer's disease is that *aluminum* may serve as a toxic agent in the lesions. Aluminum levels of 10–30 times normal values were found in brain tissue of patients with Alzheimer's disease (Crapper *et al.*, 1976). Although an attempt to replicate these findings was unsuccessful, aluminum was demonstrated in the nuclear region of neurofibrillary tangles containing neuronal cells from Alzheimer's patients through the use of scanning electron microscopy and X-ray spectrometry (Perl and Brody, 1980). As noted previously (see the section on Pathology), injection of aluminum into animal brains causes neurofibrillary tangles, but these tangles are structurally different from the neurofibrillary tangles that occur in human beings. However, identical immunostaining for Alzheimer's neurofibrillary tangles and aluminum-induced neurofibrillary tangles has been reported (Gambetti *et al.*, 1979). Since aluminum is ubiquitous in the environment, and there is no evidence that persons exposed to high levels of aluminum have a higher incidence of Alzheimer's disease (dialysis dementia, in which aluminum was implicated, has a different picture than Alzheimer's), it is generally believed that aluminum may be secondarily sequestered in diseased cells rather than being the causative agent of the dementia (Beck *et al.*, 1982).

Multiinfarct Dementia

Multiinfarct dementia is more common in men than in women, and most common between the ages of 40 and 60.

Multiple and extensive localized areas of softening characterize the brain with multiinfarct dementia. Cerebral and extracerebral vessels usually show hypertensive, atherosclerotic, or inflammatory abnormalities. Unlike Alzheimer's disease, multiinfarct dementia is a dementia secondary to vascular changes, and the etiology is that of the underlying vascular disease.

Clinical Course of the Dementias in the Elderly

The clinical course of dementia of the Alzheimer type is characterized by an insidious onset, with progression from Phase I, forgetfulness phase, through Phase II, confusional phase, to Phase III, dementia phase, with progressive confusion, memory loss, and behavioral problems including inappropriate behavior, loss of social judgment, and, at times, psychotic behavior. Originally, Alzheimer's dementia was considered to be a disease of the presenium (under age 65), but now there is believed to be no evidence that there is any difference, either clinically or pathologically, between Alzheimer's disease occurring in the presenium and that occurring at a more advanced age.

In multiinfarct dementia, the onset is usually rapid, often following a series of strokes, and the course often fluctuates, with episodes of confusion followed by relative remissions, and the personality tends to be better preserved than in dementia of the Alzheimer type. Multiinfarct dementia is often associated with hypertension and a series of cerebrovascular accidents.

Dementia is known to decrease life expectancy markedly. In one study, the average survival period was 2.6 years following diagnosis for demented men and 2.3 years for demented women, as compared to 8.7 years and 10.9 years for age-matched, nondemented men and women, respectively (Kay *et al.*, 1964).

Memory Dysfunction in the Elderly

While dementia as a syndrome is not the norm in the elderly population, there is generally a decline in memory and intellectual function with advancing age (Corkin *et al.*, 1982). Cognitive decline with ad-

vancing age has also been demonstrated in other mammalian species, including mice, rats, and monkeys (Bartus *et al.*, 1982). Such decline in intellectual functioning may be associated with age-related loss of neurons as well as other degenerative changes in the brain.

As we discussed above, nearly all pathological changes that occur in Alzheimer's disease are also present in normal aging brain. Thus, it appears that the difference between the decline of intellectual function in the normal elderly and that of dementia is a matter of degree, perhaps correlated to the degree of pathological changes in the brain. On the other hand, it is possible that the senile plaques and neurofibrillary tangles, even if they are found in elderly persons without dementia, nonetheless represent pathological conditions.

Evaluation of Organic Brain Syndromes

Biological Dimension

Organic brain syndrome, by definition, implies that there is a biological etiology to the syndrome that must be investigated. Careful medical history, physical examination, and laboratory tests are indicated in any patient with organic brain syndrome to identify the biological causative agent. Careful history for both licit and illicit drug use is an important aspect of history-taking.

If delirium is suspected, particular attention should be paid to conditions and diseases that cause metabolic changes in the brain—conditions that cause an alteration in the extracellular environment. These include, among others, endogenous and exogenous toxic states, fever, electrolyte imbalance, hypoxia, and drug-withdrawal states. Laboratory tests are of utomost importance in evaluating delirium, since they are likely to provide information concerning the exact metabolic abnormality (e.g., uremia, hypokalemia). The confusion in vignette 3 was probably caused by uremia. The EEG is useful in documenting cerebral dysfunction when significant slowing is present. However, the EEG slowing may be only in relation to premorbid frequency, without reaching abnormal proportions.

When dementia is suspected, emphasis should be on identifying medical and neurological diseases that may cause structural or chronic metabolic changes in the brain. While dementia is usually a result of irreversible and widespread neuronal death, the progression of dementia may be halted and some of the lost functions may even be restored if the underlying medical condition is treated or controlled. Among such

Table 8. Reversible Causes of Dementia[a]

Depression ("pseudodementia")
Intoxication
  Therapeutic drugs
  Alcohol
  Other substances (e.g., heavy metals, CO)
Metabolic–endocrine derangements
  Renal failure
  Hyponatremia
  Volume depletion
  Hypoglycemia
  Hepatic failure
  Hypothyroidism
  Hyperthyroidism
  Hypercalcemia
  Cushing's syndrome
  Hypopituitarism
Brain disorders
  Stroke
  Subdural hematoma
  Infection (e.g., meningitis, neurosyphilis, abscess)
  Tumors (primary or metastatic)
  Normal-pressure hydrocephalus
Cardiopulmonary disorders (e.g., congestive heart failure, arrhythmias, chronic obstructive pulmonary disease)
Generalized infections (e.g., tuberculosis, endocarditis)
Deficiency states (e.g., vitamin B_{12}, folate, niacin)
Miscellaneous causes
  Sensory deprivation (e.g., blindness, deafness)
  Hospitalization (e.g., from isolation or anesthesia)
  Fecal impaction
  Anemia
  Remote effects of cancer

[a] From Beck *et al.* (1982). Reprinted with permission.

treatable causes of dementia are endocrinopathies (e.g., hypo- and hyperthyroidism, Addison's disease, Cushing's disease, parathyroid disease), metabolic diseases and nutritional deficiencies (e.g., diabetes mellitus, thiamine deficiency, vitamin B_{12} deficiency, pellagra), infections (e.g., brain abscess, neurosyphilis), neoplasms (e.g., meningioma), and vascular disorders (e.g., atherosclerosis, systemic lupus erythematosus, cerebrovascular accidents). Pseudodementia of depression is also often misdiagnosed as irreversible dementia (see below).

    See Table 8 for a list of reversible causes of dementia (Beck *et al.*, 1982).

Unfortunately, primary progressive degenerative dementias, such as Alzheimer's disease and Pick's disease, account for more than 50% of dementias in persons over 65 years of age.

The most common dementing conditions of the elderly, Alzheimer's disease and multiinfarct dementia, account for some 75% of the dementias in the elderly.

Personal Dimension

Symptoms and signs in the personal dimension constitute the cardinal features of organic brain syndromes. Fluctuating levels of consciousness, perceptual changes, and impairment of attention and memory are characteristic features of delirium. Forgetfulness, decrease in intellectual ability, and personality change (exaggeration of premorbid personality traits such as querulousness, suspiciousness, and impulsivity) are prominent in dementia. Careful history-taking in the personal dimension will also provide information concerning the onset—rather rapid and fluctuating in delirium, and insidious and progressive in dementia.

Mental-status examination, described in detail in Chapter 12, documents the specific deficits in organic brain syndrome. Abnormalities in *appearance* may be present; a delirious or demented patient often appears cachectic because of the underlying disease. The *sensorium* is characteristically clouded in delirium. *Orientation* is impaired markedly in delirium, and progressively in dementia. *Speech and motor activity* are often impaired in delirium and dementia. *Cognitive processes, including memory, attention, concentration, logical thinking, abstraction, calculation, and judgment, are impaired in organic brain syndromes*. Of particular importance is the memory impairment, which is an early sign of dementia, and is especially pronounced concerning recent events. For example, a patient who took a prescribed pill at 9 P.M. may not remember at 10 P.M. that he had already taken the medicine, so he may take an additional dose at 10. At 11 P.M. he may have again forgotten taking the pill at 10, and so on. Accidental overdoses of this kind are quite common among elderly patients with mild to moderate dementia (see vignette 2).

Perceptual abnormalities are common, particularly illusions and misperceptions, as well as frank hallucinations. In organic brain syndromes, *visual hallucinations* are quite common, in distinction to schizophrenia, in which auditory hallucinations are more characteristic.

The affective disturbance in organic brain syndromes is *lability*—the patient may cry one minute and then burst out laughing in a matter

of seconds. There is also impairment in the modulation of affect, so that the patient may strike out in a fit of anger at the slightest provocation. When confronted with a cognitive deficit (e.g., not being able to do a simple calculation), the patient may show irritability, anger, or panic. This phenomenon has been called "catastrophic reaction" by Goldstein (1952). The sudden confrontation with an intellectual deficit may cause a collapse of the defense mechanism of denial (see Chapter 5), resulting in overwhelming anxiety. In interviewing patients with suspected dementia, the physician must use tact and gentleness to avoid a catastrophic reaction.

Environmental Dimension

Information to be gained in the environmental dimension is essential in understanding organic brain syndromes. To begin with, because of the confusion and memory deficit so common among patients with delirium and dementia, the physician may be unable to obtain a reliable medical history from the patient. A spouse, relative, or friend may be indispensable not only in obtaining history, but also in providing objective information concerning the patient's performance—for example, was he able to shop, cook, get around town, and otherwise live independently? An assessment of the living conditions of the patient suspected of having organic brain syndrome is crucial—for example, does the apartment have an elevator and are there persons nearby who might be able to supervise the administration of the patient's prescribed drugs?

Environmental factors may precipitate or exacerbate an organic brain syndrome. Delirium may occur in states of sensory deprivation or overload, as in the intensive care unit psychosis (see Chapter 18). Other environmental factors that may contribute to organic brain syndromes include environmental toxins (e.g., heavy metals), extremes of temperature or noise, and infectious agents.

Many patients with organic brain syndrome maintain routine daily function as long as they are in a familiar environment. This is because their adaptational ability is not taxed through the need to learn new things (remote memory and routinized activities are not as impaired as recent memory and newly learned tasks). Once such a patient is removed from the familiar surroundings and placed in a strange and often confusing environment such as that in a hospital, sudden decompensation of the patient's defense mechanisms and personality may occur, with an exacerbation of the preexisting deficits as in vignette 4. Thus, a careful history concerning any recent changes in physical and social

environment and in activities and routines may provide important information.

Differential Diagnosis of Organic Brain Syndromes

The syndromic diagnosis of delirium or dementia is only the first step in a differential diagnostic process that should lead to a definitive diagnosis of an underlying disease as outlined in the preceding section.

In this section, we will discuss the differential diagnosis of the organic brain syndromes from other psychiatric and behavioral syndromes.

Delirium should be differentiated from schizophrenia, acute mania, and other forms of "nonorganic" psychosis. While hallucinations, agitation, and disordered thinking and speech are common in both categories, clouding of sensorium is unlikely in schizophrenia, mania, and "nonorganic" psychoses. Visual hallucinations are more indicative of delirium than of schizophrenia. Laboratory abnormalities (e.g., electrolyte imbalance) as well as EEG slowing are helpful in establishing the diagnosis of delirium.

Dementia should be differentiated from "normal process of aging," schizophrenia, and depression. While there may be some deterioration in memory with aging, normal aging does not produce the degree of intellectual deterioration as in dementia to the extent that it interferes with social and occupational functioning. While chronic schizophrenia may be accompanied with some intellectual deterioration, it is rarely as progressive as in dementia. Schizophrenia cannot be diagnosed *de novo* in a patient over the age of 45 (that is, if the patient is over age 45, as most patients who are suspected of dementia are, the syndrome cannot be due to schizophrenia unless the patient had schizophrenic episodes in the past). The identification of the underlying disease establishes the diagnosis of dementia.

Depression is an important syndrome to be differentiated from dementia. As discussed in Chapter 6, cognitive difficulties including inability to concentrate, indecisiveness, and slowed thinking processes are common in patients with the depressive syndrome. When these cognitive difficulties are pronounced in a patient with depression, this syndrome is sometimes referred to as "pseudodementia" of depression. The significance of this differentiation is that pseudodementia of depression abates when the depression is treated successfully. Patients with pseudodementia usually have other signs of depression. In general, patients with pseudodementia tend to have histories of previous depres-

sion, the onset of the cognitive difficulties is relatively more recent and abrupt, and the symptoms tend to be more fluctuating than in dementia patients. "I don't know" is a more frequent answer given by a pseudodementia patient, while "near-miss" answers are more likely given by the truly demented patient. EEG and CT scan abnormalities tend to support the diagnosis of true dementia, but in cases of serious doubt, a trial of antidepressant therapy may be necessary for a definitive differential (Beck *et al.*, 1982).

The patient described in vignette 5 was shown to have symptoms and signs of the depressive syndrome on careful examination. She had sleep disturbance (early morning awakening), anorexia, guilty ruminations, and suicidal ideation in addition to the apathy and forgetfulness. She would often reply "I don't know" to simple questions. An EEG and CT scan were normal. Under the presumptive diagnosis of pseudodementia of depression, she was given a course of desipramine, a tricyclic antidepressant (see Chapter 20). Within eight weeks of treatment, she showed a remarkable recovery from her cognitive and depressive symptoms.

Management of Organic Brain Syndromes

Biological Dimension

Accurate diagnosis of the underlying biological condition is essential in the successful management of organic brain syndromes. In the case of delirium, this is particularly important, since there is no such thing as "primary or idiopathic delirium." Delirium is always secondary to a metabolic derangement in the brain. A corollary of this is that the treatment of delirium must always be directed to the underlying biological condition. Treatment or management of the diagnosed medical or surgical disease, toxic state, drug-withdrawal state, or laboratory abnormality, then, is the definitive treatment of delirium.

There is no effective drug treatment for delirium *per se*, with the exception of drug-withdrawal states. In fact, discontinuation of nonessential drugs should be considered, since delirium is often caused by the toxic or idiosyncratic effect of a drug. In delirium caused by withdrawal of CNS depressant drugs (e.g., delirium tremens), other CNS depressant drugs such as benzodiazepines are effective. Small doses of antipsychotic drugs may be tried for severe agitation in delirious patients if the agitation renders the patient unmanageable. This is no substitute,

however, for identification and treatment of the underlying etiological condition.

In dementia, identification and treatment or control of the underlying disease should be attempted whenever possible. One should recognize that there are a number of treatable and reversible diseases that cause dementia. Etiological treatment is not possible, however, for the primary degenerative diseases such as Alzheimer's disease.

Drug Therapy for Elderly Patients and Patients with Irreversible Dementia

General Principles: Americans aged 65 and older receive 22% of all drug prescriptions (Rabin, 1972). Nearly two thirds of the elderly use drugs on a regular basis, using more than 13 prescriptions per year. Less than 5% of the aged population abstain from all drugs (Salzman, 1982).

Since most patients with irreversible dementia are elderly, special consideration should be given to the age-related changes in drug metabolism. Elderly patients, in general, have diminished hepatic and renal function; thus, the blood level of a dose of drug may be higher than in a younger person because of the prolonged half-life. Drugs should be started at a very small dose and increased very gradually if necessary. On the other hand, concomitant medications [the elderly tend to take more than one drug—an average of 5–12 medications a day (Salzman, 1982)] may delay the absorption of some drugs (antacids and milk of magnesia often do this) or cause undesirable interactions.

Since memory deficit is a prominent feature of patients with dementia, compliance with a verbally given drug regimen cannot be assured. Whenever possible, medications should be dispensed by someone other than the patient—spouse, relative, friend, or a visiting nurse. As much as possible, drugs should be dispensed in individual containers that specify the dates and times when the drugs are to be taken.

See Table 9 for a list of age-related biological changes and their clinical and pharmacological consequences.

Psychotropic Drugs: At least one third of elderly patients hospitalized for medical or surgical illness in a general hospital received at least one psychotropic drug according to one report (Salzman and van der Kolk, 1980). About half of patients receiving a psychotropic drug report that the drug is essential for their daily functioning (Kalchthaler *et al.*, 1977).

Caution is necessary if elderly patients are to be treated effectively with psychotropic drugs without subjecting them to unnecessary polypharmacy, psychological dependence on drugs, and the side effects of potent drugs. Thompson *et al.* (1983) proposed a routine screening procedure prior to the institution of a psychotropic drug regimen for the elderly, which is presented in Table 10. In general, psychotropic

Table 9. Age-Related Biological Changes and Their Clinical and Pharmacological Consequences[a]

Factor	Change	Consequence
Absorption	1. May be delayed due to antacids, milk of magnesia, or anticholinergic drugs	1. These changes are not specific for the elderly, but are more likely to occur in the elderly patient who takes these drugs. 2. Delayed passive absorption of chlordiazepoxide
Distribution	1. Increase in fat/muscle ratio, which leads to increased apparent volume of distribution of lipid-soluble drugs	1. All psychotropic drugs except lithium are lipid-soluble. Clinical consequences of increased volume of distribution are uncertain in elderly patients; may prolong clearance.
Plasma albumin levels	1. Decrease	1. Hypoalbuminemia has been associated with increased psychotropic drug toxicity in the elderly.
First-pass effect	1. Decreases with age and congestive heart failure	1. Theoretically would increase the fraction of unmetabolized psychotropic drugs with high or intermediate extraction such as imipramine, desipramine, and nortriptyline.
Hepatic metabolism	2. Decreased by propranolol and cimetidine 1. Decreased demethylation	1. May be responsible for increased plasma levels and prolonged excretion half-lives of imipramine, amitriptyline, chlordiazepoxide, and diazepam, depending on exact site of methyl removal. Decreases thioridazine metabolism.

2. Decreased hydroxylation	2. Impaired or delayed production of hydroxy metabolite of tricyclic antidepressants, leading to higher blood levels of above drugs and nortriptyline.
	3. Prolongation of time to reach steady-state levels of above drugs.
Excretion	
1. Gradual age-related decrease in renal blood flow; reduction in drug clearance; decline in glomerular filtration rate, reabsorptive capacity, and excretory capacity of tubules	1. Decreased lithium clearance; prolongation of time to reach steady state of lithium.
Receptor-site sensitivity	
1. Conflicting reports about age-related CNS toxicity of lithium	1. Increased confusion with lithium in the elderly.
2. Increased CNS sensitivity to benzodiazepines	2. Age-related increase in sedation, disinhibition, and confusion with chlordiazepoxide, nitrazepam, and flurazepam.
3. Decrease in nigrostriatal dopamine	3. Increase in extrapyramidal side effects with neuroleptics.
4. Decrease in CNS cholinergic functioning	4. Increased sensitivity to anticholinergic properties of psychotropic drugs; increase in toxic confusional states with tricyclics.

Table 10. Routine Screening Procedures Recommended for Elderly Patients in Whom Use of Psychotropic Drugs Is Being Considered[a]

History
  Is a medical illness causing the "psychiatric" symptoms?
  Is a drug the patient is currently taking causing the psychiatric symptoms?
  Has the patient had these or other psychiatric symptoms in the past? If so, what was the diagnosis and what medication, if any, was therapeutically effective? What side effects, if any developed?
Physical examination
  Is there evidence of neurological, renal, hepatic, or other medical disease that would further increase the elderly patient's risk for side effects?
Mental status
  Is there a psychiatric illness of recent onset?
  Is there evidence of dementia or delirium?
Laboratory studies
  Is there evidence of decreased hepatic synthesizing function (i.e., decreased serum albumin) or decreased renal function (i.e., decreased creatinine clearance)?
Drug interactions
  What adverse drug interactions might develop if the psychotropic drug was added to medications the patient is currently taking?

[a] From Thompson *et al.* (1983). Reprinted with permission.

drugs should be started in smaller doses (30–50% of normal adult starting dose) and increased very gradually. The dosage should be titrated against therapeutic effect and side effects. The physician should periodically attempt to taper off the medication. Psychotropic drugs should not be continued indefinitely in the absence of any clear-cut improvement in target symptoms. If in doubt, discontinue the medication (Thompson *et al.*, 1983).

Drugs for Anxiety, Agitation, and Psychosis: Benzodiazepines are useful for mild to moderate anxiety states in the elderly. The longer-acting benzodiazepines have the advantage of being able to be administered only once a day, but the disadvantage of potential cumulative effects. There is evidence that the elderly, compared to younger patients, are more sensitive to benzodiazepines, due partly to increased target-organ sensitivity and partly to impairment of the drug metabolism (Thompson *et al.*, 1983). Antianxiety drugs should be used only for short-term relief of anxiety, and not for long-term coping with stresses of life, for which psychotherapy or counseling is more appropriate (Thompson *et al.*, 1983). In using benzodiazepines, one should be aware that they are CNS depressants, that they potentiate the effect of alcohol, and that they are habit-forming.

Table 11. Selected Benzodiazepines: Pharmacological Factors of Special Relevance for the Elderly[a]

Drug	FDA-approved use	Half-life	Usual initial dose for the elderly
Flurazepam	Hypnotic	50–100 hours (major metabolite)	15 mg at bedtime
Temazepam	Hypnotic	5–15 hours	15 mg at bedtime
Oxazepam	Anxiolytic	5–20 hours	10 mg three times a day
Diazepam	Anxiolytic	20–100 hours (major metabolite)	2 mg per day or twice a day
Lorazepam	Anxiolytic	10–20 hours	0.5–2 mg per day

[a] From Thompson *et al.* (1983). Reprinted with permission.

See Table 11 for a list of benzodiazepines and their relevant attributes in the treatment of the elderly.

Antipsychotic drugs, in small doses, are effective in controlling severe agitation and confusion. These drugs, of course, are also used in treating coexisting psychosis. Antipsychotic drugs should be used only as a temporary measure and in the smallest effective doses in treating agitation and confusion because of the risk of tardive dyskinesia, a chronic and often irreversible movement disorder.

See Table 12 for a list of antipsychotic drugs with special relevance to the elderly patient.

Table 12. Selected Antipsychotics: Pharmacological Factors of Special Relevance for the Elderly[a]

Agent	Relative potency[b]	Predominant side effects	Usual initial daily dose for the elderly
Chlorpromazine	100	Sedating, anticholinergic	10–25 mg two or three times a day
Thioridazine	95–100	Sedating, anticholinergic	10–25 mg two or three times a day
Thiothixene	5	Extrapyramidal	2–3 mg
Haloperidol	2	Extrapyramidal	0.5–2 mg
Fluphenazine	2	Extrapyramidal	0.5–2 mg

[a] From Thompson *et al.* (1983). Reprinted with permission.
[b] Chlorpromazine was arbitrarily assigned a potency of 100, for the sake of comparison with other agents. Thus, 100 mg of Chlorpromazine approximately equals 2 mg of Fluphenazine.

Hypnotic Drugs: The elderly constitute about 11% of the whole population, but they are reported to receive almost 40% of all sedative–hypnotic prescriptions in the United States (Solomon *et al.*, 1979). There is a sleep-pattern change as well as a reduction in the total sleep time with age [see Figure 21 (Chapter 10)]. There is often a disturbance in sleep continuity in elderly persons. Napping is also common, which may reduce the amount of nighttime sleep.

In general, hypnotic drugs are not indicated for elderly patients, except on a temporary basis for relatively severe difficulty in falling asleep. Persistent sleep disturbances and early morning awakening should alert the physician to the possibility of a medical disease or the depressive syndrome.

When a hypnotic drug is indicated, benzodiazepines such as flurazepam and temazepam may be used (see Tables 11 and 18).

Barbiturates should be avoided in elderly patients, since these drugs are particularly prone to cause paradoxical excitement, suppression of rapid eye movement (REM) sleep (Chapter 10), and rebound nightmares and insomnia (Kales and Kales, 1974).

Antidepressant Drugs: Depression is an important syndrome that often mimics (pseudodementia) or coexists with dementia in the elderly. Clinically significant depression is considered to be present at any given time in at least 10% of the elderly population (Thompson *et al.*, 1983). Recognition and treatment of the depressive syndrome, then, is an important aspect of managing the elderly patient with dementia. (See Chapter 6 for further discussion of the depressive syndrome.)

Antidepressant drugs are effective when used in combination with psychotherapy and social support. Tricyclic antidepressants are most commonly used in elderly patients. Special attention should be paid to the anticholinergic and hypotensive effects of antidepressant drugs. Tricyclic antidepressants also have quinidine like antiarrhythmic action, which may interact with other antiarrhythmic drugs, or cause heart block in patients with preexisting bundle-branch block.

See Table 13 for a list of antidepressant drugs with special relevance to elderly patients (Thompson *et al.*, 1983).

Drugs to Improve Memory: There is as yet no demonstrably and consistently effective drug for the memory problems in elderly or demented patients. The search for drugs that may prevent or reverse intellectual deterioration is continuing along various lines. Among others, they include (1) agents that augment cholinergic function in the brain, (2) CNS stimulants, (3) drugs that affect dopaminergic, GABAergic, and pepti-

Table 13. Selected Antidepressants: Pharmacological Factors of Special Relevance for the Elderly[a]

Agent	Category	Relative anticholinergic effects	Relative sedative effects	Usual initial daily dose for the elderly
Amitriptyline	Tricyclic tertiary amine	6+	5+	10 mg three times a day and possibly 20 mg at bedtime
Doxepin	Tricyclic tertiary amine	3+	6+	25–50 mg
Imipramine	Tricyclic tertiary amine	4+	3+	30–40 mg
Nortriptyline	Tricyclic secondary amine	3+	2+	25–50 mg
Desipramine	Tricyclic secondary amine	1+	2+	25–50 mg
Maprotiline	Tetracyclic	3+	3+	25 mg per day or twice a day
Amoxapine	Dibenzoxazepine	3+	3+	25 mg three times a day
Trazodone	Triazolopyridine	1+	3+	500–100 mg

[a] From Thompson *et al.* (1983). Reprinted with permission.

dergic neurotransmission, (4) metabolic enhancers, (5) cerebral vaso-dilators, (6) drugs that affect autooxidation (e.g., Coenzyme Q), and (7) drugs that affect immune and autoimmune mechanisms.

Some promising results have been obtained with the administration of dietary lecithin, a choline precursor, and dihydroergotoxine mesylate (Hydergine), a stimulant. In aged rats, it has been shown that piracetam, a stimulant, and choline act synergistically in improving memory retention (Roberts, 1982). Although the results have not been striking, dihydroergotoxine mesylate may be used in the dose range of 3–6 mg orally per day for mild to moderate memory deficit in the elderly patient (Salzman, 1982).

Personal Dimension

Patients with acute delirium are typically confused, bewildered, and often frightened. A reassuring and nondemanding approach by health-care personnel will tend to reduce the patient's anxiety. Because of the impaired memory, concentration, and attention span, formal psycho-

therapy has no place in the treatment of a delirious patient. Once delirium has cleared, patients often need psychotherapeutic help to integrate the frightening experience (which is often described as being dreamlike) into their ongoing life. Letting patients tell the clinician, when they wish, about the frightening thoughts, hallucinations, and other experiences, and letting them know that these are common phenomena caused by transient toxic influences on the brain, can help patients overcome their fears (e.g., of recurrence, of being "crazy") and continue with their normal lives.

Patients with progressive dementia may require supportive psychotherapy to help them cope with daily stresses and directive counseling in managing their lives. This kind of support can be given by the patient's significant other, such as spouse or friend, as well as by healthcare personnel. The purpose of psychotherapy or counseling for the dementia patient is to supplement the declining problem-solving ability and memory while providing a sense of being cared for. Scheduling and routinizing daily activities to reduce demands for new learning is also an important part of treatment in the personal dimension.

Environmental Dimension

When a pathogenic environment is identified, removal of the patient from the environment (e.g., transfer of the patient out of the intensive care unit in cases of ICU psychosis) or stabilization of the environment may be effective in manging the patient with organic brain syndrome. Night lights, a calendar, and familiar objects (such as photographs of family) from home can stabilize and familiarize a strange environment. The staff should orient the patient repeatedly, since the patient may not remember the date or the place even if the nurse has told the patient what they are only a few hours previously.

Hospitalization should be considered for patients who have acute delirium, either alone or superimposed on dementia, since delirium is usually reversible if the cause is identified and treated vigorously. Hospitalization also provides a protective environment for the severely agitated or confused patient.

Many patients with dementia require the help of another person in supervising their medication regimens and, eventually, for daily living and personal hygiene. Visiting nurse or homemaker service may be necessary.

Usually, nursing home placement is eventually necessary for patients with such progressive dementias as Alzheimer's disease. Families of patients who must be admitted to nursing homes often require counseling and reassurance, since they tend to feel guilty and anxious. It is useful for the families to know that in end-stage dementia, the suffering of the significant other is usually greater than that of the patient.

Summary

Organic brain syndromes are characterized by cognitive dysfunction due to a metabolic or structural brain dysfunction. Major areas of deficit include sensorium, orientation, attention, memory, concentration, logical thinking, abstraction, and judgment. Lability of affect is also present, and perceptual abnormalities such as illusions and hallucinations may occur.

In delirium, clouding of sensorium is characteristic, the onset is usually rapid, and the course is fluctuating and usually reversible. Delirium is caused by a metabolic derangement of the neurons secondary to alterations in the neuronal environment, e.g., toxins, electrolyte imbalance, or drug withdrawal. Delirium is always secondary to an underlying organic condition, and laboratory findings are helpful in identifying and monitoring the metabolic alteration. Treatment of delirium should always be geared to the underlying metabolic cause.

Dementia refers to a syndrome of acquired loss of intellectual abilities to the extent that social and occupational function is impaired. There is often progressive loss of memory and impairment of abstract thinking and judgment. Personality change is also common. Dementia tends to occur more commonly in the elderly. The onset and course of dementia depend on the underlying cause. There are many reversible or treatable causes of dementia, including vitamin deficiency syndromes (e.g., thiamine, B_{12}, niacin), endocrinopathies, and metabolic disorders. Alzheimer's disease and multiinfarct dementia account for approximately 75% of dementia in the elderly. Alzheimer's disease is the most common cause of senile dementia, and is characterized microscopically by large numbers of neurofibrillary tangles, senile plaques, and granulovacuolar degeneration in the brain. There may be a selective involvement of cholinergic neurons in Alzheimer's disease. Aluminum, found in the neurofibrillary tangles, has also been implicated. Genetic factors, viruses, and immunological factors have also been implicated in the pathogenesis of Alzheimer's disease. All Down's syndrome patients who survive long

enough develop Alzheimer changes in the brain. CT scan of Alzheimer's disease patients usually reveals evidence of cortical atrophy and enlarged ventricles.

Pseudodementia of depression is frequently misdiagnosed as Alzheimer's disease. Coexisting symptoms and signs of depression, past history of depression, and relatively rapid and fluctuating symptomatology should alert the physician concerning the possibility of pseudodementia, which is quite reversible with successful treatment of depression.

While there seems to be a mild generalized reduction in memory function and intellectual ability with aging, dementia is not a normal concomitant of aging. Some 80–90% of the elderly population do not develop dementia.

The evaluation of organic brain syndromes should be comprehensive, with particular attention to the clinical manifestations by careful mental-status examination, to the underlying biological condition through physical examination and laboratory tests, and to the precipitating environmental factors.

Treatment of organic brain syndromes should always be directed to the underlying cause. Antipsychotic drugs may be used in small doses to control severe agitation or psychotic symptoms, but never as a substitute for definitive treatment of the underlying disease. Night lights, familiar objects, and repeated orienting stimuli help reduce disorientation and confusion. In irreversible dementia, supportive and protective environment, counseling, and symptomatic drug treatment may be indicated.

Drug therapy for elderly patients requires special considerations. Drug metabolism is usually delayed in older persons due to decreases in hepatic and renal function. Thus, drug blood levels and half-lives are often drastically increased in the elderly. Since elderly patients usually use more than one drug on an ongoing basis, the physician must consider the interactions of the drugs the patient is taking. Elderly patients may be particularly sensitive to benzodiazepines, and paradoxical agitation may occur with CNS depressant drugs. In general, drug therapy should be instituted with caution, for specific target symptoms or goals, and drugs should be started at a very low dose and increased very gradually.

For patients with major memory problems, drugs should be dispensed by someone other than the patient to avoid confusion and accidental overdose. Drugs should be dispensed in individual containers with clear markings for the time and date of ingestion.

Implications

For the Patient

Delirium is a common experience for acutely ill patients. The experience of acute confusion, disorientation, agitation, illusions, and hallucinations is frightening to patients. Explanations concerning the transient and toxic nature of these symptoms may be particularly reassuring.

Suspected dementia is frightening to the patient because of the implications, and naturally generates severe anxiety. Patients should be educated that there are many reversible causes of memory loss. When irreversible progressive dementia has been diagnosed, supportive and protective measures should be undertaken (e.g., writing down daily schedule, supervised medication regimen).

For the Physician

Since organic brain syndrome is one of the most common psychiatric conditions on nonpsychiatric services in the general hospital, the physician should be alert to the possibility of its presence whenever he or she encounters a patient who seems confused or agitated or whose behavior or personality seems to have changed somehow. Careful mental-status examination will document the presence or absence of organic brain syndrome. In performing the mental-status examination (see Chapter 12), the physician should be gentle and supportive and avoid "catastrophic reaction."

If delirium is present, the physician should promptly identify the underlying metabolic abnormality through appropriate examinations and laboratory tests. Effective and speedy treatment of the underlying cause is important, since delirium, if prolonged, may cause irreversible neuronal death, resulting in dementia.

The physician should take careful drug history and drug screens on patients with delirium, since drug intoxication and withdrawal are common causes of delirium.

The physician should be aware that significant memory loss and confusion are abnormal even in the elderly, and should be alert to the possibility that the patient may have dementia that may be secondary to potentially reversible causes, which should be investigated vigorously.

In managing a patient with severe memory deficits, the physician should not rely on verbal instructions. Step-by-step, written instruc-

tions, preferably given to a care-taking person as well as the patient, will ensure compliance with the regimen. The physician should be aware of the special considerations in the drug treatment of the elderly described in this chapter.

For the Community and the Health-Care System

Illicit drug use, a social problem, is a common cause of delirium in the younger population. Society should be educated concerning the dangers of delirium that often results from either intoxication or withdrawal from drugs such as phencyclidine, amphetamines, and barbiturates.

Dementia is a disorder of epidemic proportions in the elderly population. Dementias related to vascular problems (e.g., multiinfarct dementia) may be prevented by better control of hypertension and atherosclerosis through early detection, treatment, and judicious diet.

Alzheimer's disease patients are eventually institutionalized. Since the elderly population is increasing, there is a need to provide more and better facilities for those who are afflicted with this disease as well as other conditions requiring protective environments.

Medical schools should educate future physicians concerning the special considerations in evaluating and managing elderly patients, as well as facilitate research into the mechanisms of age-related changes in intellectual function.

Recommended Reading

Bartus RT, Dean RL III, Beer B, et al.: The cholinergic hypothesis of geriatric memory dysfunction. Science 217:408–417, 1982. A comprehensive and thoughtful review of the hypothesis that memory impairment in the elderly, particularly in Alzheimer patients, is due to the dysfunction of specific cholinergic neurons in the brain.

Beck JC, Benson DF, Scheibel AB, et al.: Dementia in the elderly: The silent epidemic. Ann Intern Med 97:231–241, 1982. A transcript of a special conference at UCLA on the topic. Quite readable. Has good discussions concerning reversible causes of dementia.

Engel GL, Romano J: Delirium, a syndrome of cerebral insufficiency. J Chronic Dis 9:260–277, 1959. This classic paper defined delirium as a syndrome of cerebral insufficiency and showed that slowing of the EEG is a sign of this syndrome.

Schneck MK, Reisberg B, Ferris SH: An overview of current concepts of Alzheimer's disease. Am J Psychiatry 139:165–173, 1982. This is a comprehensive review of historical and up-to-date concepts on Alzheimer's disease.

Sinex EM, Merrill CR (ed): Alzheimer's Disease, Down's Syndrome, and Aging. Ann NY Acad Sci 396, 1982. A collection of papers describing recent research concerning Alzheimer's disease, aging, and Down's syndrome. Includes many papers on the genetic aspects, metabolic aspects, immunological studies, and therapeutic considerations.

Thompson TL II, Moran MG, Nies AS: Psychotropic drug use in the elderly: 2-part article. *N Engl J Med* **303**:134–138, 194–199, 1983. An up-to-date, comprehensive, and readable discussion concerning the special considerations in the use of psychotropic drugs for the elderly. Highly recommended.

References

Albert ML, Feldman RG, Willis AL: The "subcortical dementia" of progressive supranuclear palsy. *J Neurol Neurosurg Psychiatry* **37**:121–130, 1974.

Bartus RT, Dean RL, Beer B, *et al.*: The cholinergic hypothesis of geriatric memory dysfunction. *Science* **217**:408–417, 1982.

Beck JC, Benson DF, Scheibel AB, *et al.*: Dementia in the elderly: The silent epidemic. *Ann Intern Med* **97**:231–241, 1982.

Bowen DM, Spillane JA, Curzon B, *et al.*: Accelerated ageing or selective neuronal loss as an important cause of dementia. *Lancet* **1**:11–14, 1979.

Corkin S, Davis K, Growdon J, *et al.* (eds): *Aging*, Vol. 19: *Alzheimer's disease: A Report of Progress in Research*, New York, Raven Press, 1982.

Corsellis JAN, Evans PH: The relation of stenosis of the extracranial cerebral arteries to mental disorder and cerebral degeneration in old age, in *Proceedings of the Fifth International Congress of Neuropathology*. The Hague, The Netherlands, Mouton & Co, 1965, p 546.

Crapper DR, De Boni U: Etiological factors in dementia, in *Abstracts: Satellite Meeting on Aging of the Brain and Dementia, International Society for Neurochemistry*. Abano Terme, Italy, FIDIA Research Labs, 1979.

Crapper DR, Krishnan SS, Quittkat S: Aluminum, neurofibrillary degeneration and Alzheimer's disease. *Brain* **99**:67–80, 1976.

DeLeon MJ, Ferris SH, George AE, *et al.*: Computed tomography evaluation of brain–behavior relationship in senile dementia of the Alzheimer's type. *Neurobiol Aging* **1**:69–79, 1980.

Engel GL, Romano J: Delirium, a syndrome of cerebral insufficiency, *J Chronic Dis* **9**:260–77, 1959.

Fisch M, Goldfarb AI, Shahinian SP, *et al.*: Chronic brain syndrome in the community aged. *Arch Gen Psychiatry* **18**:739–745, 1968.

Gambetti P, Velasco ME, Dahl P, *et al.*: Neurofibrillary tangles in Alzheimer's disease: An immunohistochemical study, in *Abstracts: Satellite Meeting on Aging of the Brain and Dementia, International Society for Neurochemistry*. Abano Terme, Italy, FIDIA Research Labs, 1979.

Goldstein K: The effects of brain damage on the personality. *Psychiatry* **15**:245–260, 1952.

Heston LL: Alzheimer's disease and senile dementia: Genetic relationship to Down's syndrome and hematologic cancer, in Katzman R (ed): *Congenital and Acquired Cognitive Disorders*, New York, Raven Press, 1979, p 167.

Kalchthaler T, Coccaro E, Lichtige S: Incidence of polypharmacy in a long term facility. *J Am Geriatr Soc* **25**:308–313, 1977.

Kales A, Kales JD: Sleep disorders: Recent findings in the diagnosis and treatment of disturbed sleep. *N Engl J Med* **280**:487–499, 1974.

Katzman R: The prevalence and malignancy of Alzheimer's disease: A major killer (editorial). *Arch Neurol* **33**:217–218, 1976.

Kay DWK, Norris V, Post F: Prognosis in psychiatric disorders of the elderly: An attempt to define indications of early death and early recovery. *J Ment Sci* **102**:129–140, 1956.

Kay DWK, Beamish P, Roth M: Old age mental disorders in Newcastle upon Tyne. *Br J Psychiatry* **110**:146–148, 1964.

Lipowski ZJ: Delirium, clouding of consciousness and confusion. *J Nerv Ment Dis* **145**:227–255, 1967.

Lipowski ZJ: *Delirium: Acute Brain Failure in Man*. Springfield, Illinois, Charles C Thomas, 1980.

Matsuyama H, Nakamura S: Senile change in the brain in the Japanese: Incidence of Alzheimer's neurofibrillary change and senile plaques, in Katzman R, Terry RD, Bick KL (eds): *Alzheimer's Disease: Senile Dementia and Related Disorders*. New York, Raven Press, 1978, p 287.

Merskey H, Ball MJ, Blume WT, *et al.*: Relationship between psychological measurements and cerebral organic changes in Alzheimer's disease. *J Can Sci Neurol* **7**:45–49, 1980.

Perl DP, Brody AR: Alzheimer's disease: X-ray spectrometric evidence of aluminum accumulation in neurofibrillary tangle-bearing neurons. *Science* **208**:297–299, 1980.

Rabin DL: *Use of Medicine: A Review of Prescribed and Nonprescribed Medicine Use*. Washington, DC, US Public Health Service, 1972.

Roberts E: Potential therapies in aging and senile dementia, in Sinex EM, Merril CR (eds): Alzheimer's disease, Down's syndrome, and aging. *Ann NY Acad Sci* **396**:165–178, 1982.

Rossor MN: Dementia. *Lancet*, Nov. 27, 1982, pp 1200–1204.

Salzman C: A primer on geriatric psychopharmacology. *Am J Psychiatry* **139**:67–74, 1982.

Salzman C, van der Kolk BA: Psychotropic drug prescriptions to elderly patients in a general hospital. *J Am Geriatr Soc* **28**:18–22, 1980.

Schneck MK, Reisberg B, Ferris SH: An overview of current concepts of Alzheimer's disease. *Am J Psychiatry* **139**:165–173, 1982.

Sims NR, Bowen DM, Smith CCT, *et al.*: Glucose metabolism and acetylcholine synthesis in relation to neuronal activity in Alzheimer's disease. *Lancet* **1**:333–335, 1980.

Sinex EM, Merril CR (eds): Alzheimer's disease. Down's syndrome, and aging. *Ann N Y Acad Sci* **396**, 1982.

Sinex EM, Myers RH: Alzheimer's disease, Down's syndrome, and aging: The genetic approach, in Sinex EM, Merril CR (eds): Alzheimer's disease, Down's syndrome, and aging. *Ann NY Acad Sci* **393**:3–13, 1982.

Solomon F, White CC, Parron DL, *et al.*: Sleeping pills, insomnia and medical practice. *N Engl J Med* **300**:803–808, 1979.

Spillane JA, White P, Goodhart MJ, *et al.*: Selective vulnerability of neurons in organic dementia. *Nature (London)* **1**:11–14, 1977.

Terry RD: Dementia: A brief review. *Arch Neurol* **33**:1–4, 1976a.

Terry RD: Neurofibrillary tangles of paired helical filaments. *J Neurol Sci* **27**:173–181, 1976b.

Terry RD, Gonatas NK, Weiss M: Ultrastructural studies in Alzheimer's presenile dementia. *Am J Pathol* **44**:269–297, 1964.

Terry RD, Fitzgerald C, Peck A, *et al.*: Cortical cell counts in senile dementia (abstract). *J Neuropathol Exp Neurol* **36**:633, 1977.

Thompson TL II, Moran MG, Nies AS: Psychotropic drug use in the elderly. *N Engl J Med* **308**:134–138, 194–199, 1983.

Titchener JL, Zwerling I, Gottschalk L, *et al.*: Psychosis in surgical patients. *Surg Gynecol Obstet* **101**:59–65, 1956.

Tkach JR, Hokama Y: Autoimmunity in chronic brain syndrome: A preliminary report. *Arch Gen Psychiatry* **23**:61–64, 1970.

Tomlinson BE, Blessed G, Roth M: Observations on the brains of nondemented old people. *J Neurol Sci* **7**:331–356, 1968.

Tomlinson BE, Blessed G, Roth M: Observations on the brains of demented old people. *J Neurol Sci* **11**:205–242, 1970.

Wisniewski HM, Soifer D: Neurofibrillary pathology: Current status and research perspectives. *Mech Ageing Dev* **9**:119–142, 1979.

CHAPTER 9
Pain

1. A 59-year-old man came to the emergency room complaining of crushing pains in the chest. He was immediately admitted to the coronary care unit with a diagnosis of myocardial infarction (heart attack). Morphine was given by injection to relieve the pain.

2. A 23-year-old married woman was admitted to the obstetrics ward today as she developed labor pains. As he was waiting for her to deliver the baby, the husband also experienced severe crampy pains in his abdomen, which disappeared after his wife's delivery.

3. A 20-year-old single woman is still in severe pain after two weeks in the hospital. This is her seventh admission to the hospital in three years because of severe pains in the abdomen. During the first admission, an operation was done to rule out acute appendicitis. Her appendix was normal. She had two additional operations on the abdomen, because the cause of the pain was suspected to be adhesions from the first operation. All tests so far during this hospitalization have been normal, but she continues to have severe crampy pains and asks for frequent pain medications. When she asks for pain medication, she is given a saline injection (placebos) at the order of the physician, who believes that she is addicted to narcotic pain medications. The injection seems to help her for a couple of hours, but then she cries for another injection. According to the nurses, she seems to be always bitter and cynical.

4. A 27-year-old man who is a surgical patient in the hospital is complaining of severe shooting and stabbing pains in his right leg and foot. The patient had an amputation of the right leg and foot four weeks ago, after an industrial accident that crushed his right foot. This is an example of "phantom limb" pain—pain felt as coming from a limb that is no longer there.

209

These are only a few examples of patients with pain frequently seen in the hospital. Many patients with pain obviously do not present themselves to the physician, and almost all of us have from time to time had pain in some part of the body that we were able to ignore or that could be relieved with aspirin. It should be obvious from the foregoing cases that pain is not a simple phenomenon; it is determined and influenced by many factors. What, then, are the mechanisms and functions of pain, and what are the factors that affect this familiar but sometimes puzzling entity?

Definitions and Functions of Pain

Pain is an abstract concept that refers to a personal, private sensation of hurt, often as a result of a harmful stimulus that signals current or impending tissue damage, and an accompanying pattern of responses that operate to protect the organism from harm (Sternbach, 1968).

At a concrete level, pain is a perceptual experience like hearing or vision, but, unlike vision or hearing, pain experience is determined or modified to a greater extent by a multiplicity of factors such as the sufferer's psychological set, including expectations, suggestions, previous experiences, and sociocultural environment.

The function of pain is protective; it is usually a signal that tissue damage is occurring, and it alerts the person to take appropriate action or get away from it if possible. In this sense, pain as a signal of impending threat is analogous to anxiety. Anxiety occurs in anticipation of the threat, while pain occurs when the threatening situation is actually causing damage to the organism. Unlike anxiety, pain is usually perceived as emanating from a part of the body—it is *localizable*.

Like anxiety, pain may signify the presence of a *psychological state* without actual tissue damage. Pain *metaphors* describing emotional states are common, such as the "pain of loneliness" and "heartaches." In fact, descriptions of pain occurring from tissue damage are often also quite metaphorical, such as "splitting headaches," "stabbing pain," and "heartburn." These linguistic uses clearly indicate close association between the experience of pain and the *symbolic* meanings in the mental life of human beings. Thus, it is not surprising that some persons perceive certain psychological states as physical pain, attributable to a body organ.

Pain often generates anxiety; in many situations, anxiety is generated as a signal that a potentially painful process is in the making. *Anxiety*

may also generate pain; pain is the most common heterothetic symptom resulting in doctor–patient contact (see Chapter 1).

Anxiety and pain are probably associated through a learning process (see Chapter 4). In childhood, physical punishment (pain) often occurs in a situation in which the child has anxieties about the possible loss of parental love; fear of the loss of bodily parts in injury is also accompanied by the sensation of pain. For many persons, therefore, pain is often associated with anxiety and anxiety with pain.

Pain is also a frequent symptom of depression, being present in 60–100% of depressed patients (VonKnorring, 1975; Ward *et al.*, 1979). There is evidence that chronic pain syndrome without evidence of underlying tissue damage may be a variant of depressive disorder (Blumer and Heilbronn, 1982). The evidence for this hypothesis is derived from the fact that chronic pain syndrome patients often show aspects of the depressive syndrome (although the depressive affect itself may be absent) and have family histories of affective disorder and that the chronic pain often responds to antidepressant drug treatment. On the other hand, depression may often develop as a consequence of severe and chronic pain due to tissue damage.

Qualities of Pain

Many different adjectives have been used to describe the qualitative aspects of pain. These include, among others, throbbing, pounding, shooting, stabbing, tender, aching, splitting, stinging, and grueling. Melzack and Torgerson (1971) classified 102 such descriptors into three major classes:

1. Words describing the *sensory* qualities of the experience, such as temporal, spatial, pressure, and thermal properties—e.g., pricking, scalding.
2. Words describing the *affective* qualities, such as tension and fear, and autonomic properties—e.g., sickening, exhausting, frightening, wretched.
3. Words *evaluating* the subjective overall intensity of pain—e.g., miserable, unbearable.

The quality of pain reported by patients is most important in helping the physician in diagnosis. For example, the crushing pain reported in vignette 1 is typical of a coronary disease, while a dull, aching pain in the same location (chest) is more likely to be from the chest wall, not the heart. Elaborate metaporical quality in the description of pain, such

as "feeling as if someone is sitting on my stomach, cutting bowels out with a knife," often indicates that the pain is associated with elaborate fantasies of the patient.

Neurophysiological experiments show that pain sensation emanating from tissue damage can be classified into three major types:

1. Pricking pain: the type of pain caused by a needle pricking the skin or by incision of the skin. Strong irritation of a large area of the skin can also cause this sensation.
2. Burning pain: the type of pain felt when the skin is burned. This is often excruciating.
3. Aching pain: a low-intensity pain usually felt deep inside the body, not on the surface.

Pricking pain is conducted through small myelinated type A delta nerve fibers, while burning and aching pains are conducted by even smaller unmyelinated type C nerve fibers.

Nature of Pain

Phenomenologically, pain is a *subjective experience* and falls into the realm of "private" data; that is, the experience of pain *cannot be shared* by others, but can only be reported. This is an important point, since physicians may tend spuriously to objectify pain, as though it were identical in degree to the pathology causing it.

Although certain events, such as injury, are usually associated with pain, under some circumstances, whether pain is experienced or not, and if so, how much, is dependent on factors other than tissue damage; for example, Beecher (1959b) reported that two thirds of the badly wounded men in a World War II battle did not complain of pain or ask for medications for it. This may be a manifestation of "stress anesthesia" that is mediated by release of endorphins under severe stress.

Beecher proposed that there are two *components* to a pain experience—a primary *sensory* component and a reactive *psychological* component. The primary component is the pain sensation itself, which includes the perception, discrimination, and recognition of the noxious stimulus. The secondary component is the suffering aspect of pain, which is an emotional aspect including anxiety. The reactive component is not always commensurate with the primary sensation. Physiological changes associated with pain, such as change in heart rate, blood pressure, and skin conductance, are thought to be related to the reactive component of pain.

Neurophysiology of Pain

The "pain receptors" are considered to be free nerve endings that are stimulated by tissue damage and stimuli that can cause tissue damage. The exact mechanism by which tissue damage stimulates the pain receptors is not known. There seem to be in the free nerve endings receptors that respond only to very strong mechanical stimuli and strong thermal stimuli evoking pain sensation. Also, various chemicals, such as lactic acid formed in the muscle due to lack of oxygen; polypeptides, such as bradykinin formed as a tissue breakdown product; amines (serotonin and histamine); and prostaglandins are known to cause intense pain.

The nerve impulses arising from the pain receptors (nocioceptors) travel through two types of nerve fibers in the sensory nerve: the type A delta fibers and the type C fibers. The A delta fibers have a conduction velocity of 3–20 m/sec, while the C fibers transmit at 0.5–2 m/sec. As mentioned earlier, the pricking type of pain is transmitted by the A delta fibers and the burning, aching pain by the type C fibers. Thus, a sudden painful stimulus can result in two perceptions, an initial pricking sensation followed by an aching or burning sensation a second or so later. The cell bodies of the pain fibers are in the dorsal root ganglia, and most pain fibers enter the spinal cord through the dorsal roots, then ascend or descend one or two segments in Lissauer's tract, terminating in the neurons in the gray matter of the dorsal horns (see Figures 14 and 15). Recent evidence has shown that significant numbers of unmyelinated C fibers enter the spinal cord via the ventral roots and then project to the dorsal horn.

In the dorsal horns, the signals pass through one or more short-fibered neurons, the last of which give rise to long fibers that cross to the contralateral side and ascend in the spinal cord as the *spinothalamic and spinoreticular tracts*.

In the brain, the ascending pain pathway separates into two separate pathways, the "pricking pain pathway" and the "burning pain pathway" (Guyton, 1976). The pricking pain pathway terminates in the caudalmost part of the ventrobasal complex of the thalamus. There are neural connections between this complex and the somatosensory cortex and other areas of the thalamus.

The burning pain pathway terminates in the reticular formation of the brain stem and the intralaminar nucleus of the thalamus. The reticular formation and the intralaminar nuclei are parts of the reticular activating system (RAS) [see Figure 7 (Chapter 4)], the function of which is to regulate the level of arousal of the entire central nervous system. The pain signals activate this system, sending activating signals to the
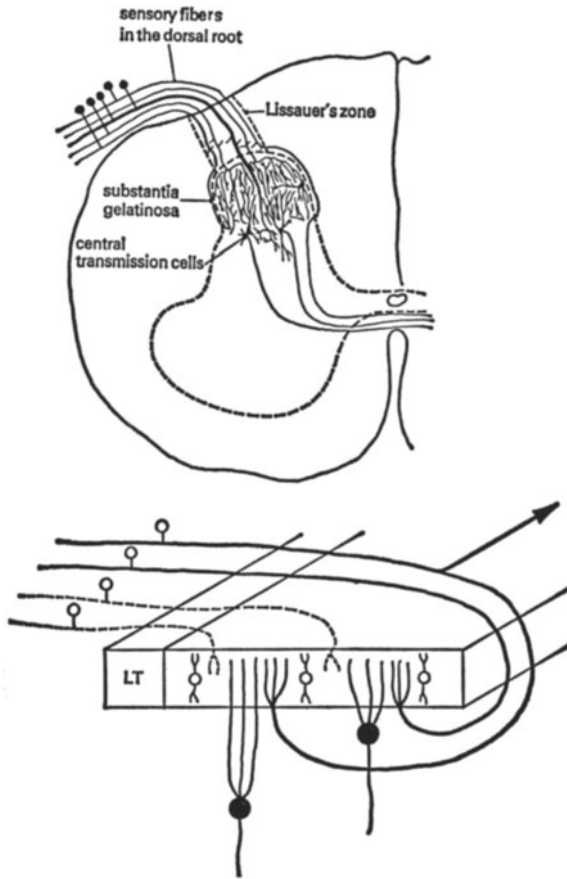
Figure 14. Top: Schematic drawing of the substantia gelatinosa in relation to somatosensory fibers and dorsal horn cells that project their axons across the cord to the anterolateral pathway. (After a figure originally published in AA Pearson, *Arch Neurol/Psychiatry* **68**:515, 1952. Copyright 1952 by the American Medical Association.) Bottom: Main components of the cutaneous afferent system in the upper dorsal horn. The large-diameter cutaneous peripheral fibers are represented by thick lines running from the dorsal root and terminating in the region of the substantia gelatinosa; one of these, as shown, sends a branch toward the brain in the dorsal column. The finer peripheral fibers are represented by dashed lines running directly into the substantia gelatinosa. The large cells, on which cutaneous afferent nerves terminate, are shown as large black spheres with their axons projecting deeper into the dorsal horn. The open circles represent the cells of the substantia gelatinosa. The axons (not shown) of these cells connect them to one another and also run in Lissauer's tract (LT) to distant parts of the substantia gelatinosa. (Adapted from a figure originally published in PD Wall, *Prog Brain Res* **12**:92, 1964. Courtesy of Oxford University Press.) (From Melzack, 1973. Copyright 1973 by Ronald Melzack and reproduced with his permission.)
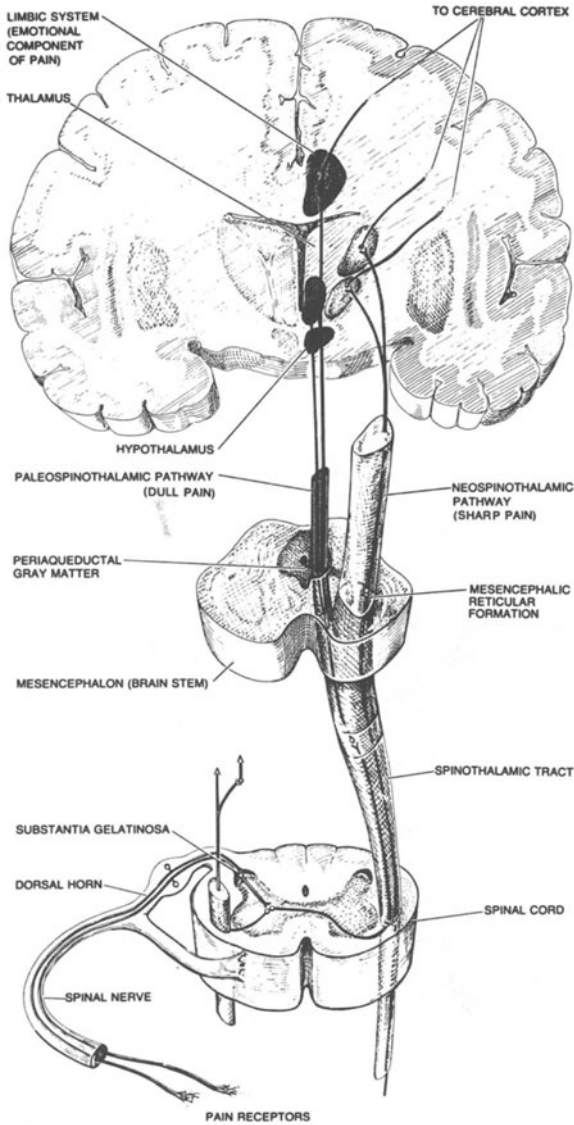
Figure 15. Two types of pain pathways carrying information from the periphery of the nervous system to the brain: the laterally located neospinothalamic pathway, which transmits sharp, localized pain, and the medially located paleospinothalamic pathway (shaded), which transmits less localized burning pain. Burning pain is best relieved by opiates, and opiate receptors have been found to be concentrated in the substantia gelatinosa and in the central thalamus. (From Snyder, 1977. Copyright 1977 by Scientific American, Inc. Reproduced with permission.)

entire cortex, entire brain stem, thalamus, and hypothalamus, which controls the autonomic and neuroendocrine systems. Thus, through the RAS, pain causes a central nervous system state of arousal, promoting defensive reactions to get rid of the noxious stimulus (see Chapter 5).

Pain sensations from various parts of the body are represented in the thalamic nuclei in a somatotopical fashion somewhat like that in the sensory cortex. Although sensory modalities other than pain require intact sensory cortex to be perceived, the perception of pain seems to require the functional integrity of the nervous system *only up to the thalamic level*. The states of other parts of the nervous system, however, have a major impact on the degree of perception and on the interpretation of the perception and the organism's response to it. The cerebral cortex may affect the state of the neurons in the spinal column through efferent nerves, which might, in turn, affect the sensation itself.

The localization of pain seems to depend to a large extent on the simultaneous stimulation of tactile receptors. The signals transmitted by type C fibers are localizable only very grossly, since this pathway terminates very diffusely in the brain. As mentioned earlier, pain is a subjective experience that cannot be measured objectively. It is possible, however, to measure the least amount of stimulus on the skin necessary to elicit the report of pain from an individual (*pain threshold*). This threshold can be measured by projecting a beam of radiant heat onto the skin. The skin temperature at which pain is first perceived is remarkably uniform in different people, regardless of their ethnic or cultural background, sex, age, and other differences. Most people begin to feel the sensation of pain when the skin temperature reaches almost exactly 45°C.

This uniformity of the threshold for pain, however, is valid only in precisely controlled laboratory situations, where the environmental factors are kept constant. The perception of pain is markedly modified in natural conditions by the person's psychological state, such as anticipation, attention, or suggestion. The pain threshold is lowered in injured skin reddened by vasodilatation. Sensitization of pain receptors also occurs when a very strong stimulus is initially applied (Perl, 1976). "Adaptation" to stimuli also occurs, so that there is a decrease in pain sensation after repeated stimuli.

Historical Neurophysiological Theories of Pain Perception

Historically, there are three theories concerning the neurophysiological basis of pain perception: (1) the specificity theory, (2) the pattern theory, and (3) the gate control theory.

The *specificity theory* is the oldest theory; it postulates a specific pain receptor with specific pathways leading to a particular nucleus in the thalamus. In other words, according to this theory, the free nerve endings responsible for pain sensation have no function other than to detect pain, and there is no mechanism for detection of pain other than stimulation of these free nerve endings (receptors). The thalamus is considered to be the primary organ for the integration of pain sensation, according to this theory. In its classic form, this theory cannot account for pain phenomena in which there is no stimulation of the specific pain receptors, as in phantom pain (vignette 4) (see Figure 16).

The *pattern theory* postulates that particular types of input, whether or not they came from specific receptors, would set in motion a particular firing pattern in *reverberatory circuits* in the spinal cord internuncial neurons. This self-propagating nerve impulse in the dorsal horns would then send to the brain volleys of impulses that are perceived as pain. This theory can explain phantom limb pain, since the initial damage to the limb, or the amputation procedure itself, could initiate abnormal firing patterns in the reverberatory circuits in the dorsal horns. There is not good evidence, however, that reverberatory circuits initiating and perpetuating pain exist in the spinal cord.

The *gate control theory* was proposed by Melzack and Wall (1965). This is an attempted integration of the theories concerning pain and postulates the following: A neural mechanism in the dorsal horns of the spinal cord acts like a gate that can increase or decrease the flow of nerve impulses from peripheral fibers to the central nervous system (Melzack, 1973). Once signals from receptors (which are specific for pain, as in the specificity theory) reach the spinal cord, their transmission from the afferent fibers to the cells that give ascending output into the brain (transmission, or T, cells) is modulated by the gating mechanism. The gating mechanism was thought to be located in the *substantia gelatinosa*, a group of small neurons located at the tip of the dorsal horn. The substantia gelatinosa receives impulses from many small and large fibers entering the spinal cord as well as from descending fibers from the brain, such as the reticulospinal tract. According to the gate control theory, large-diameter fibers, such as those responsible for position and touch, have an inhibitory effect on the gate control mechanism, reducing the flow of impulses transmitted to the brain. The small-diameter fibers, such as the pain fibers (both A delta and C), have a facilitatory effect. The descending fibers from the brain have an inhibitory effect; the brain stem reticular formation is also considered to exert a powerful inhibitory control over information projected by the spinal cord transmission cells.
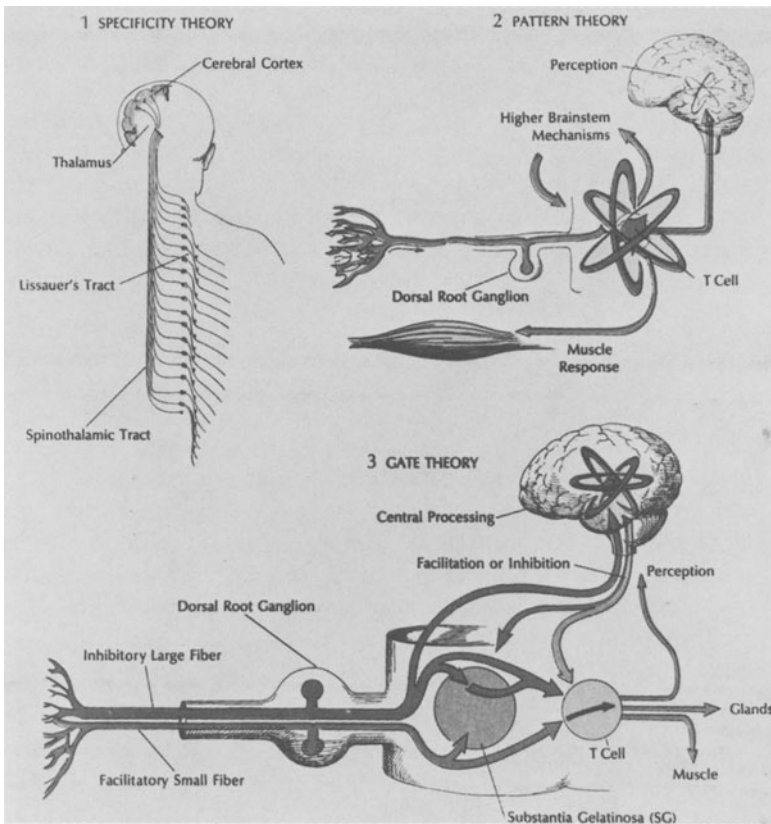
Figure 16. Schematic representation of three theories of pain transmission and perception. The earliest was the **specificity theory** (1), which held that pain stimuli enter the spinal cord through spinal nerves and synapse ipsilaterally, then rise several levels in Lissauer's tract. They then cross the cord and ascend to the thalamus, where they synapse again and rise to the cerebral cortex, where pain is perceived. The pattern theory (2) postulates stimuli entering from nerves through dorsal root ganglia into the spinal cord. The so-called T cell is in the lateral horn of the spinal cord. It sets up a response, part of which results in an impulse to higher brain stem mechanisms. These, in turn, modulate the response by action on the T cell, which fires and sends impulses to the brain, causing perception, and to striated muscle, facilitating response. The gate theory (3) depends on the concept of two "parallel" fibers, both with cell bodies in the dorsal root ganglia. The large fiber has basically an inhibitory effect on pain perception, the small fiber basically a facilitatory effect. The large fiber acts on and stimulates the substantia gelatinosa (SG). Such stimulation will prevent firing of the T cell, which is necessary for pain perception. The small fiber can overcome or modify the large fiber's influence on the SG, or it can directly stimulate the T cell to fire, or it can do both. The large fiber may also act directly on the brain's central processing mechanisms, although the pathways of this action have not been defined. Impulses may be either inhibitory or facilitatory. If the latter, the result will be firing of the T cell, producing pain perception and endocrine and muscle responses. (Reprinted with permission from Pearson, 1976. Drawings by Ms. Carol Donner.)

A specialized system of large-diameter, fast-conducting fibers (probably in the dorsal column of the spinal cord and its projections), which is called the "central control trigger," is postulated to carry information concerning the pain (e.g., location). This system is considered to activate selective brain processes, such as memories of prior experiences, before the perception of pain and then to exert control selectively over the sensory input by modulating the gate control system through descending pathways, or by modulating the receptivity of the cortical neurons to the stimuli coming up more slowly through the pain pathways, or in both ways.

According to this theory, phantom limb pain can be explained on the basis of release from inhibition; the loss of large-diameter fibers from the amputated organ "opens the gate control mechanisms," so that the T cell fires at a very low threshold level or even spontaneously. This theory can also explain the pain-relieving effect of counterirritation (such as liniment and acupuncture), which stimulates the inhibitory large-diameter fibers.

There is controversy concerning the existence of such a gating mechanism and the exact nature of the T cells. More recent findings indicate that there is a specific system of nocioception in which specific receptors that are activated only in case of tissue damage (e.g., high-intensity pressure) give rise to impulses in corresponding specific afferent nerves (high-threshold neurons as opposed to low-threshold neurons such as those subserving touch sensation), leading to activation of specific nocioceptive areas of the central nervous system (Kerr and Wilson, 1978). It is also clear, however, that this specific nocioceptive system is influenced and modified by many factors other than tissue damage, especially through descending fibers from the brain (see below).

Role of Endorphins in Pain Mechanisms

When specific receptors binding morphine were discovered in the human brain in the early 1970s, people wondered why human beings might have developed receptors to an alkaloid made by the poppy plant. Obviously, a more likely explanation was that these receptors were for some morphinelike substance made by our own brains. Recently, a number of substances that possess morphinelike properties have been identified in the brains and pituitary glands of many animals and humans. These properties include, among others, analgesia, respiratory depression, euphoria, and addictiveness. The chemical structures of these substances have been studied, and all have been found to be peptides. The

generic name for the peptides with opiatelike properties is *endorphins*. The name *enkephalins* is given to certain specific pentapeptides belonging to the class of endorphins. Some large-molecule endorphins, such as β-endorphin, a potent opioid peptide containing 31 amino acids, contain in their structures the shorter 5-amino-acid sequences of the enkephalins.

All the endorphins, including enkephalins, are antagonized by narcotic antagonists such as naloxone. Thus, their actions seem to be mediated by binding to the opiate receptors discovered earlier. In addition to properties similar to those of morphine, some endorphins have other important behavioral and physiological effects when injected into the brain or the cerebrospinal fluid. For example, β-endorphin and metenkephalin (which forms a fragment of the structure of β-endorphin) cause catatonic behavior in rats, with a decrease in body temperature. These effects are reversible with naloxone (Bloom *et al.*, 1976).

The opiate receptors, the presumed site of action of both the endorphins and narcotic analgesics, are especially heavily distributed in the limbic brain, in the central thalamus, and in the substantia gelatinosa of the spinal cord. As previously discussed, these areas of the central nervous system are especially concerned with the perception of pain. The importance of the limbic brain in the perception of the emotional component of pain should be obvious from our discussion in Chapter 4. It is possible that the euphoriant effect of narcotics is closely related to the concentration of opiate receptors in the limbic system. The opiate receptors are also abundant in all areas the stimulation of which causes analgesia.

The endorphins and the smaller enkephalins (which have a weaker and shorter duration of action) may be neurotransmitters or neuromodulators that modify the perception of pain. The mechanism of action seems to be through inhibition of neurons related to pain perception by means of a rather unusual mechanism of blocking the sodium influx elicited by excitatory neurotransmitters (Snyder, 1977).

Many forms of analgesia may occur through the release of endogenous endorphins, as demonstrable by their reversal by naloxone. These endorphin-dependent analgesias include: brain-stimulation analgesia, acupuncture and some forms of placebo anesthesia, and nitrous oxide anesthesia. Although endorphins may play a role in the inhibition of pain perception during stress, hypnoanesthesia has not been reversed by naloxone (Goldstein, 1976; Marx, 1977).

It seems, then, that endorphins play an important role in the *self-regulation* of the perception and tolerance of pain by the brain through their action on *pain at the spinal cord all the way up to the emotional reactions*

to the pain sensation mediated by the limbic system. In chronic pain, the endogenous endorphin levels may be elevated; on the other hand, insufficient endorphin release due to genetic or other reasons might predispose a person to feel excessive pain. *Some patients may require greater amounts of narcotic analgesics* for control of minor pain because of insufficient or depleted endorphin-release mechanisms in the central nervous system.

Descending Influences from the Brain

A number of descending systems from the brain influence the state of the spinal cord. Brain stem stimulation can cause a change in the excitability of spinal cord cells (Wall, 1976). Stimulation of the sensori-motor cortex inhibits low-threshold neurons, but the high-threshold units are unaffected (Coulter *et al.*, 1974). A cold block of the spinal cord rostral to the recording site has a tonic inhibitory effect on nocioceptive neurons (Handwerker *et al.*, 1975).

Electrical stimulation of certain areas of the brain produces potent analgesia, called stimulus-produced analgesia (SPA). SPA can also be produced by stimulation of peripheral nerves, although not as consistently as by brain stimulation. The areas of the brain that produce analgesia on stimulation include the periaqueductal gray, the dorsal raphe nucleus, the nucleus raphe magnus of the medulla, the gray matter surrounding the third ventricle, and the septal area (Kerr and Wilson, 1978) (see Figure 15). The analgesia produced by brain stimulation specifically inhibits the activation of the nocioceptive neurons in the dorsal horn without affecting the low-threshold neurons (Beall *et al.*, 1976; Oliveras *et al.*, 1974). SPA produced by brain stimulation is effective against visceral as well as somatic pain.

Injection of very small amounts of morphine into some of the brain areas that can cause SPA also produces a marked elevation in pain threshold, and the analgesia produced by electrical stimulation of the brain areas is reversed by the administration of or pretreatment with naloxone, an opiate antagonist (Kerr and Wilson, 1978).

Animal and human data indicate that there are at least four classes of descending pathways for centrally induced analgesia: neural–opiate, neural–nonopiate, hormonal–opiate, and hormonal–nonopiate (Watkins and Mayer, 1982). In animals, certain environmental stresses such as centrifugal rotation and injection of intraperitoneal hypertonic saline cause potent nonopiate analgesia that is not reversed by naloxone.

If the stressor is electrical shock to the feet, either opiate- or non-opiate-mediated analgesia occurs, depending on the location of the shock; front-paw shock activates the opiate system, while hind-paw shock activates a nonopiate analgesic system. When animals are exposed to the nonelectrified grid following repeated sessions of electric shock to the front paws, potent analgesia develops, which is reversed by naloxone. Thus, the nonelectrified grid may serve as a conditioned stimulus to produce the activation of the opiate analgesic system.

Analgesias induced by front-paw shock, by morphine, and by classical conditioning are quite similar; none is attenuated by the ablation of pituitary or adrenal glands, all are reversed or attenuated by naloxone, and all are subject to the development of morphine tolerance. Thus, they belong to the neural–opiate class of descending analgesia. Hind-paw shock activates the neural–nonopiate system. Shock-produced analgesia seems to induce both neural–opiate and neural–nonopiate analgesia. The dorsolateral funiculus of the spinal cord seems to be a final common pathway of all neurally mediated analgesia, since they are all reversed by lesioning this structure (Watkins and Mayer, 1982).

Acupuncture analgesia, analgesia induced by prolonged shock of all four paws, and immobilization-induced analgesia are also reversed by naloxone, but are distinguishable from neural analgesia in that they are abolished or attenuated by the removal of the pituitary or adrenal glands. Thus, they are examples of hormonal–opiate analgesia. Analgesia induced by cold water swims, on the other hand, requires the integrity of the pituitary gland but is not reversed by naloxone, being an example of hormonal–nonopiate analgesia.

Central Neuropharmacology of Pain

Biogenic amines play an important role in pain perception. Tetrabenazine, a substance that depletes brain monoamines, markedly reduces the analgesia produced by stimulation of the periaqueductal gray in rats (Akil and Liebeskind, 1975). This effect can be reversed by the administration of serotonin or L-dopa.

Dopamine and *serotonin* seem to decrease pain mechanisms in the brain. Stimulation of dopamine receptors in the brain by apomorphine produces a marked increase in brain-stimulation-induced analgesia, while the blockade of dopamine receptors with pimozide impairs the analgesia. The administration of *p*-chlorophenylalanine, an inhibitor of serotonin synthesis, results in decreased brain-stimulation analgesia.

This effect is reversed by the administration of the serotonin precursor 5-hydroxytryptophan.

Brain *norepinephrine* seems to have an effect on pain mechanisms opposite to that of dopamine and serotonin. Disulfiram, a substance that blocks norepinephrine synthesis from dopamine, causes a significant increase in brain-stimulation-induced analgesia.

The effect of morphine and endorphins may be modulated by brain monoamine levels. For example, reserpine antagonizes experimental morphine anesthesia (Kerr and Wilson, 1978).

Substance P is a peptide that has striking depolarizing effects on neurons. It is present in large concentrations in the dorsal roots and substantia gelatinosa, as well as in the hypothalamus and substantia nigra. Many nocioceptive neurons in the spinal cord respond to substance P.

Glutamate, another substance found in high concentrations in the dorsal roots and ganglia, may be a neurotransmitter concerned with nocioception.

Somatostatin (growth-hormone-release-inhibiting hormone) is also a peptide recently found to be in some small-sized neurons in the dorsal root ganglia. There also appears to be a dense plexus of somatostatin-containing fibers in the substantia gelatinosa. Somatostatin has potent depressant activity on neuronal firing and may be an inhibitory transmitter for the perception of pain.

Psychosocial Factors That Influence Pain Experience

Although the pain-sensation threshold seems to be more or less the same for most people in laboratory situations, as described earlier, the *response* to pain, perceptual intensity, and meaning of pain in natural situations are influenced by a number of psychosocial factors. Even in laboratory situations, persons of differing backgrounds show marked differences in pain *tolerance*, the level of pain at which the subject refuses to tolerate any more pain.

Cultural expectations are known to exert a powerful influence on the experience of pain. In some cultures, for example, childbirth is perceived to be quite painful, while in others, especially in the South American cultures that practice *couvade* (Kroeber, 1923), it is accompanied by no visible distress to the woman. In the latter cultures, the woman often works in the field until the time she is about to deliver, and after the delivery of the baby with no signs of pain, she returns to the field to complete her work. Her husband, however, takes to bed while his wife

is delivering and moans and groans as though he were in severe pain. Even in our culture, symptomatic variants of "couvade syndrome" are occasionally seen (vignette 2). In some parts of India, a religious ritual is still practiced in which a chosen man hangs on two steel hooks inserted into the back, which are suspended by a rope, and blesses the children and crops. During the ritual, the man reportedly feels no pain, but rather a "state of exaltation" (Kosambi, 1967) (see Figure 17).

One study in the United States showed that whites tolerated more pain than Orientals, with blacks occupying an intermediate position (Woodrow *et al.*, 1972). In a study of pain involving Old American, Irish, Italian, and Jewish housewives (Sternbach and Tursky, 1964), those of Italian origin were found to have a lower pain tolerance to electric shocks, and the Old Americans showed a more rapid physiological adaptation to repeated shock. Other studies, however, failed to demonstrate such ethnic differences in pain tolerance (Merskey and Spear, 1964; Winsberg and Greenlick, 1967). Ethnic differences in the attitude toward pain have already been discussed (Chapter 1).

What is the mechanism by which cultural and psychosocial factors influence pain experience? Although the exact mechanisms are not known, the descending influences and the recent discovery of the role of endorphins in pain-inhibition mechanisms provide a conceptual framework in which the state of the central nervous system, such as attention, anxiety, depression, and past experiences, and values ingrained in the mind and brain can influence the perception of pain.

Age may also affect pain experience. Tolerance to cutaneous pain is reported to increase with age, while tolerance to deep pain decreases with age (Woodrow *et al.*, 1972).

The *personality* of the individual has an important effect on his reaction to pain; conversely, the experience of prolonged or severe pain may also have an effect on the personality. For example, extroversion is associated with greater pain tolerance and, at the same time, with a tendency to exaggerate the pain experience, as compared to introversion (Lynn and Eysenck, 1961; Eysenck, 1961).

Persistent pain is common in psychiatric disorders, especially those characterized by anxiety, such as neuroses (Merskey, 1965a,b). Chronic pain is also thought to *increase* anxiety and "neuroticism" of patients (Bond, 1971), whether the pain is "organic" or "psychiatric" (Woodforde and Merskey, 1972). Patients with chronic pain also tend to develop a feeling of being out of control of their lives and a sense of suspicion and anger toward others, whom they attempt to "manipulate and control" (Timmermans and Sternbach, 1974). *Depression* is also a common consequence of chronic pain (Robinson *et al.*, 1972). These findings indicate
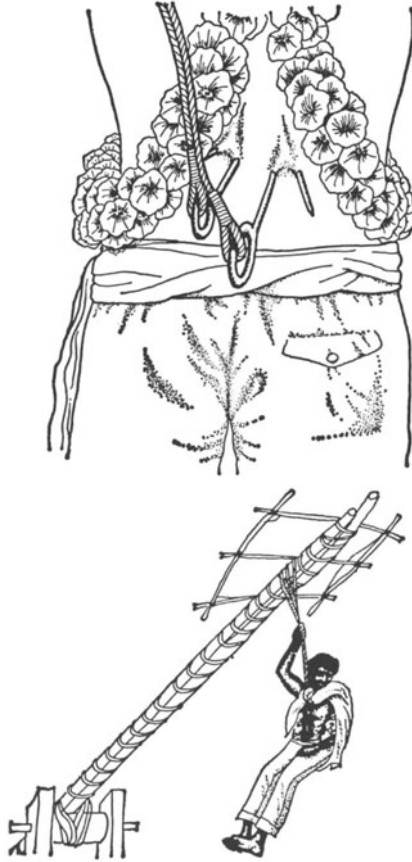
Figure 17. Annual hook-swinging ceremony practiced in remote Indian villages. Top: Two steel hooks are thrust into the small of the back of the "celebrant," who is decked with garlands. The celebrant is later taken to a special cart that has upright timbers and a crossbeam. Bottom: The celebrant is shown hanging onto the ropes as the cart is moved from one village to the next. After he blesses each child and farm field in a village, he swings free, suspended only by the hooks. The crowds cheer at each swing. During the ceremony, the celebrant is in a state of exaltation and shows no sign of pain. (Originally published in Kosambi, 1967.) (From Melzack, 1973. Copyright 1973 by Ronald Melzack and reproduced with his permission.)

that evidence of depression, anxiety, or "neurotic tendencies" in a patient with pain does not justify an automatic diagnosis of "psychogenic" pain.

Among the psychological states that affect the experience of pain are *depression* and *expectancy*. Physical sensations can be accentuated in a state of depressive withdrawal, when the individual's attention is directed toward himself and his bodily sensations. In this instance, any minor discomfort can become magnified and be experienced as serious pain. Depression also tends to result in a vicious cycle of pain escalation—pain causes depression, and then, because of the depressed state, the intensity of pain is accentuated. In cases of pain with signs of depression, antidepressant therapy might be useful to halt this vicious cycle as well as, in some cases, to alleviate the precipitating cause of the pain (see Chapter 6).

Pain has been shown to be associated with *aggression*. In patients with persistent pain, there are often heightened feelings of hostility that are not overtly expressed (Sternbach, 1968). Pain is also implicated in triggering of aggressive behavior in animals. It may be an *unconditioned stimulus for aggressive behavior* (Ulrich *et al.*, 1965). Aggression induced by pain can be conditioned, for example, at the presentation of a tone (Vernon and Ulrich, 1966) (see Chapter 4 for a discussion of conditioning). These studies imply that pain may be exacerbated or exaggerated when patients are angry and that pain may, in turn, provoke angry feelings and aggressive behavior. A non-anxiety- and non-aggressive-provoking environment, then, would seem to contribute to *prevention* of these undesirable problems of exacerbation of pain or aggressive behavior.

Expectations concerning pain play a major role in the person's experience of pain. In experimental pain situations, subjects who were told to expect severe pain had better pain tolerance than those who were not so prepared (Kanfer and Goldfoot, 1966), and when subjects had an opportunity to obtain information concerning forthcoming electric shocks and, in fact, requested such information, they tended to have decreased anxiety concerning the anticipated shock (Jones *et al.*, 1966).

Another dimension related to expectations concerning pain is the placebo effect, which we will discuss next.

Placebo Effect

Although a placebo is a substance that is considered to be pharmacologically inert, it is by no means "nothing." According to Beecher

(1955), it is a powerful therapeutic tool, on the average about one half to two thirds as powerful as morphine in the usual dose (10 mg/70 kg body weight) in relieving severe pain. Although the placebo effect is most often described in pain relief, it occurs in many other situations, such as depression and anxiety. It can produce relief from any or all symptoms for which it is given (Sternbach, 1968).

A placebo, like any other pharmacological agent, can have *side effects*, and the side effects may be "toxic" in appearance at times.

The placebo effect is not always consistent. In one study, about 50% of patients receiving both morphine and placebos for postoperative pain were relieved of the pain when the dispensing of the medication was not prolonged (Lasagna *et al.*, 1954). Of the patients who had *more than one dose of placebo, only 14%* consistently obtained relief from the placebos, 55% had inconsistent responses, and 31% consistently never received relief.

It is now recognized that approximately *one third of the general population are placebo reponders* in clinical situations, whether the pain is from surgery, angina pectoris, cancer, or headache (Beecher, 1959a, 1960). There are no generally accepted tests to differentiate placebo responders from nonresponders. Although placebo reactors were reported to be more anxious, dependent, self-centered, emotionally labile, and preoccupied with internal bodily processes than nonreactors by the Rorschach test (Lasagna *et al.*, 1954), there were no superficial behavioral characteristics—that is, the reactors were not "whiners," but rather had less "self-critical inhibition" of expressing dependency needs. The placebo reactors might be able to receive considerable pain relief through the comfort from nursing care as well as from the confidence in the efficacy of the medications. In fact, the study found that the reactors had a less painful postoperative course and received fewer medications than the nonreactors.

Placebo responses are not simply alterations in the mental state of affective response to pain. Placebo administration can also produce physiological respones (Sternbach, 1968).

The *mechanism* for the placebo effect is probably quite complex, including psychological and psychodynamic factors. The basic neurophysiological mechanism probably includes the *endorphin* pain inhibition system as well as other systems. In one recent study, placebo analgesia was reversed by the administration of naloxone (Levine *et al.*, 1978). Psychologically, gratification of dependency needs in the form of a medication may play a role. On the other hand, Herrnstein (1962) formulated a simple classic conditioning model of the placebo response. In rats, scopolamine depressed lever-pressing behavior, while saline alone did

not. When saline was followed by scopolamine in a conditioning paradigm, saline resulted in depression of the lever-pressing behavior. Herrnstein postulated that a similar type of *conditioned placebo response* might also occur in humans, although increased in complexity.

This model suggests that relief of pain was associated in childhood with certain persons and behaviors as well as affects. Love, comfort, and caring, as well as reduction of anxiety, are related to relief from pain, as are such behaviors as "mother kissing the hurt and making it better." Taking a pill is also often associated with the relief of pain. When the patient is exposed to a situation similar to those in which relief was obtained in the past, such as taking a pill or being cared for by a motherlike person (nurse), the pain may disappear (as in conditioned response).

Sternbach (1968) hypothesizes that the production of an "approach–avoidance conflict" concerning reaction to pain might contribute to the inconsistencies in the response to placebos.

In early childhood, complaining of pain usually brings about comfort and relief. In the course of growing up, however, a child learns that complaining about pain is seen negatively by others, such as, "Don't be a crybaby." This may result in a classic approach–avoidance conflict. In clinical situations, there is both pain and the sick-role expectation of being in a passive and dependent position. This is conducive to *regression* (discussed in Chapter 5)—and evocation of the approach–avoidance conflict.

Thus, patients may experience a conflict between the wish to complain of pain, to experience pain relief and comfort, and the fear that this would be seen as immature and "like a sissy" or otherwise negatively. Some of these patients may become angry and particularly resistant to pain relief even with active drugs.

Although the placebo effect has been considered to be similar to hypnosis, it appears that there is in susceptible persons a specific hypnotic analgesic effect over and above the placebo effect (McGlashan *et al.*, 1969).

Use of Placebos in Medical Practice

It should be clear from the preceding discussions that favorable nonspecific effects brought about by the patient's coming into contact with the health-care system might very well be considered to be related to the placebo effect. This general type of response is an inherent part

of medical practice and may be related to what Parsons called "unconscious psychotherapy" (see Chapter 3).

Placebos in a narrower sense, such as saline injections with specific symptoms as targets, may also be administered in a medical-treatment setting, but they are more often than not misused.

The most common *misuse* of placebos is *as a diagnostic tool*. A surgeon had asked the psychiatrist to see a patient who was suspected of having pain as a hysterical conversion symptom. When the surgeon was told by the nurses that a saline injection had brought on relief of the patient's pain, he turned to the psychiatrist and exclaimed, "Q.E.D.! Now you don't even have to see her; you can just transfer her to the psychiatry ward." This was an incorrect conclusion. Even patients with severe pain caused by demonstrable tissue damage (e.g., such as that associated with metastatic cancer to bone) frequently respond to placebos. *Placebos should never be used to make a differential diagnosis between an "organic" and a "functional" pain, since it is impossible to make such differentiation with placebos* (Shapiro, 1960).

As previously mentioned, placebos may also have *side effects*. In addition to expected "pharmacological" side effects such as nausea, blushing, and tachycardia, there is an important social interpersonal side effect that can occur with the use of placebos in a medical setting. An atmosphere of *"trickery"* and deception often develops when placebos are used to treat a patient with persistent pain. When a saline injection is ordered for a patient, the nurse's attitude and feelings are often affected; that is, a feeling of "tricking" the patient, and perhaps anger at the patient for being put in such a position, may ensue. Such an attitudinal change is often sensed by the patient, who then feels deceived and badly treated (vignette 3). Patients with guarded, suspicious personality styles may become quite angry and upset in this situation and may even leave the hospital prematurely ("against medical advice").

Psychological Meanings of Pain

We have already mentioned that pain is often associated with anxiety and anxiety-provoking situations in childhood. Such associations are, of course, unique to individual patients, and the specific meanings of a particular pain will also be unique to the patient. Punishment, threat, loss, and even reward are "meanings" that pain may acquire. Pavlov (1927, 1928) found that dogs can be conditioned to *associate pain with pleasure* (food). Dogs normally have violent negative reactions to electric shock applied to a paw. Shock regularly presented to a hungry

dog before feeding changed the reaction—the animal would salivate and start to wag its tail and turn toward the food dish immediately after the shock. In these experiments, the electric-shock experience acquired the meaning of a signal of a reward to follow (food). Such learning might account for the behavior of some patients who seem to be deliberately seeking painful experiences and suffering, for example, patients who are "addicted to surgery" (vignette 3). The "secondary gain" of being sick might be a powerfully motivating "reward experience" for some patients. Sexual excitation may be associated with painful experiences, for example, genital stimulation with pain, an association that may have first been experienced during spanking.

Pain is a *regressive stimulus*. In the presence of severe pain, the sufferer's thoughts and actions tend to become like those of a child. Sternbach (1968) writes:

> It is not only that we cry with the pain; what we say, aloud and to ourselves, is childlike. We ask what we have done to deserve such pain, and think back to make a connection between some action of ours and the onset of the pain. We implore others to help us, to take away the hurt. We promise that once the pain is removed we will be different—we will be kinder to others, do good works. We beg for forgiveness, we say we are sorry. We ask God for help, we ask Him to save us.

Many memories of childhood, associated with pain, with punishment, and with relief of pain, may be reactivated in the presence of pain. Sometimes conflictual feelings about other issues may be activated by the experience of pain. For example, some patients cannot tolerate even small amounts of pain because they are afraid of becoming like a child, the regressive meaning of having pain.

Pain may have *different meanings according to personality type*. For a patient who has a long-suffering, self-sacrificing personality style, pain may be the symbol of expiation of his guilt feelings and justification for receiving care. For a patient with a dramatizing, emotional personality, pain may mean that he is no longer attractive. For a patient with an orderly, controlling personality, pain may mean a loss of control (see Chapter 17).

"Psychogenic" Pain

The terms *functional* or *psychogenic* pain refers to pain experienced without demonstrable peripheral tissue damage; by implication, it may be thought to be "caused" by psychological or psychodynamic factors.

Still, pain, no matter what its cause, acquires psychological meaning and may be accompanied by signs of psychological distress. Furthermore, pain may signal early tissue damage or dysfunction that is not yet demonstrable. For these reasons, extreme caution must be exercised in the use of these terms. Nevertheless, there are some persons who suffer long-standing and severe chronic pain without demonstrable tissue damage, in whom the existence of pain can be explained on the basis of psychological factors (like the hallucinations experienced in dreams) and whose improvement depends largely on successful psychological management. Additionally, there are certain people who experience pain with unusual intensity and frequency, in whom the presence or absence of associated tissue damage is only weakly correlated with the quality and intensity of pain and in whom even removal of the lesion may fail to bring relief from pain. Engel (1959) called such patients "pain prone" and identified a marker of personality characteristically encountered in them. He proposed that the physician consider the following questions in evaluating patients with severe pain problems: (1) Are there pathological processes affecting nerve endings and leading to disordered patterns in nerve pathways that would be expected to produce pain? (2) If such processes are present, can the character of the pain experience reported by the patient be fully, partially, or not at all accounted for by the distinctive characteristics of the peripheral pathological process? (3) How are psychological processes operating to determine the ultimate character of the pain experience for the patient and the manner in which it is communicated to the physician? A number of factors and findings may suggest that the pain is psychogenic or psychogenically exacerbated. They include the following:

1. Psychogenic pain tends to be described in a dramatic and metaphorical fashion (such as "a man sitting on my chest").
2. The location of the pain tends to correspond to the patient's subjective body image rather than to anatomical distribution of peripheral nerves and central nervous system pain pathways.
3. Such pain tends to occur at emotionally charged times (e.g., in anticipation of a loss or at the anniversary of such an event).
4. There is a "complaining" quality to the patient's description of the pain, which is usually exacerbated during an interview when an emotionally charged subject is discussed.
5. Psychogenic pain almost never wakes a patient from sleep.

The past histories of pain-prone patients often include similar episodes of obscure pain in the past in which the pain was used as a means of getting attention and love or as a means of atonement for feelings of

guilt. The personality style, obviously, is likely to be the long-suffering, self-sacrificing type (see Chapter 17). It is not unusual to find that these patients had been close to someone who suffered from chronic or severe pain and that the patient's pain might even have started shortly after the loss of such a person. In such an instance, identification with the lost person would be an important psychological defense mechanism involved in the development of the symptom (see Chapter 6).

In some cases of psychogenic pain *secondary gain* might play an important role. This term refers to gratification or advantages that accrue to the person by virtue of the illness but did not contribute to its causation. Secondary gains may then reinforce the symptom and make it hard to give up. They include attention- and love-getting, the opportunity to be "unusually" angry and aggressive, and financial gain such as disability compensation payments. In cases in which pain occurs as a "depressive equivalent" or in pain associated with the depressive syndrome (see Chapter 6), signs of depression, such as suicidal ideas and guilt feelings, may be found as well as such physiological signs of depression as sleep disturbance, weight loss, anorexia, constipation, and loss of *sexual interest*.

Blumer and Heilbronn (1982) proposed that the chronic pain syndrome without identifiable tissue pathology should be considered to be a variant of depressive disorder. On the basis of extensive studies of "psychogenic" pain patients, they identified the following characteristics:

Demographic data: Female/male ratio approximately 2:1; highest age of onset between 45 and 50; mostly lower-middle-class blue collar workers.

Clinical features: Chronic pain that is continuous, hypochondriacal preoccupation, strong desire for a surgical solution, strong guilt feelings, strong denial of difficulties in interpersonal relationships, idealization of spouse (despite frequent history of physical abuse by spouse), insomnia, usually normal appetite, anergia, anhedonia, little emotional expression.

Premorbid characteristics: Hard-working, "workaholic," "solid citizen," frequent history of abuse by spouse.

Family history: High incidence of affective disorders in first-degree relatives, high incidence of disability or deformity in a next of kin.

Psychodynamic considerations: The pain syndrome patients are considered to have strong needs to be accepted and to be dependent. These infantile dependency needs are denied, and, instead, the future patient compensates for inner insecurity through hard work and relentless activity. A shift occurs following a significant loss or disappointment, with

or without painful injury or illness. The solid citizen is transformed into an invalid who can justifiably be dependent and taken care of. This, however, creates a painful dilemma, since the dependency needs continue to be denied, and value is placed on being well and working. Thus, the pain is seen consciously to be the "only problem" and the desire is for it to be eliminated by surgery or other magical means.

We should caution again that while the presence of these indicators should alert the physician that there may be psychogenic components to the patient's pain experience, these findings in and of themselves *by no means* indicate *absence* of organic pathology. Coexistence of psychogenic and tissue factors in pain is not at all unusual. *One of the most common ways in which psychogenic pain is expressed, in fact, is as an elaboration of pain arising in damaged tissue.*

Management of Pain

The management of pain obviously requires a comprehensive approach. Relief from pain is important not only as direct relief from suffering itself, but also because of the untoward effects of physiological concomitants of pain that might be harmful to the patient (such as the increase in cardiac work in myocardial infarction) and the fact that anxiety associated with pain may in time aggravate its intensity as well as the intensity of psychophysiological reactions to it.

For most acute pain, effective *pharmacological agents* such as narcotic analgesics are readily available to the physician and should be used in effective doses. There is some evidence, however, that narcotics are *underutilized* in treating acute pain situations for fear of addiction (Marks and Sachar, 1973). Even terminally ill patients with severe pain are often undertreated with narcotic analgesics. It should be pointed out that the actual *risk of causing narcotic addiction in a hospitalized patient with pain is quite negligible* [less than 1% (Marks and Sachar, 1973)]. Underusage of narcotic analgesics may reinforce the patient's preoccupation with the medication and his drug-seeking behavior, such as calling for the medication before the scheduled time to prevent the development of more severe pain.

Sometimes there may even be an interesting paradoxical pattern in the use of powerful analgesics. The more pain the patient feels, the more he complains, the less likely he is to receive potent pain medications (Pilowsky, 1969). It is no wonder, then, that aggravated aggressive behavior is often found in such patients, especially in view of the fact that pain itself may generate aggressive feelings.

Relief of anxiety is also important in managing pain. This calls not only for a reassuring attitude on the physician's part but also for *informing patients about treatment plans*, especially about procedures that themselves might be painful. Preparing the patient for the pain will help. As pointed out earlier, experimental subjects anticipating severe pain showed a higher tolerance for it and reduced perception of pain when motivated to endure it (Sternbach, 1968).

As mentioned earlier, pain is a powerful regressive stimulus. An unambivalent, caring attitude on the part of the health-care personnel, particularly nurses, can prevent or neutralize anxious and defensive reactions in patients who are embarrassed by regressive needs and behavior.

A comprehensive approach is also particularly necessary in managing patients with *psychogenic or psychologically aggravated pain*. In addition to symptomatic and etiological treatment of underlying disease processes in tissue, the psychological meaning of the pain should be evaluated and a plan for psychological treatment made. This may involve using *antidepressants* (see Chapters 6 and 20), as well as providing interpersonal contact, social support, and psychotherapy when indicated.

Another important reason for recognizing and treating psychogenic pain factors is to prevent unnecessary surgical and other drastic treatments.

Summary

Pain is one of the most common experiences leading to help-seeking behavior. Pain, like anxiety, subserves a protective function. *Anxiety* often accentuates, and occasionally is perceived as, pain, and pain is, as a rule, accompanied by anxiety.

The quality of pain is important in the diagnosis of the underlying disease. Tissue damage or very strong stimuli result in stimulation of the pain receptors (free nerve endings). The pricking type of pain and burning and aching pain are conducted by separate types of nerve fibers, small type A delta fibers and even smaller type C fibers, respectively. *Pricking pain* is ultimately projected to the brain in the thalamus and the somatosensory cortex, while the aching and burning pain pathways are projected diffusely in the *reticular activating system* of the brain and thus influence not only the state of arousal, but also emotional and neuroendocrine responses. The nerve impulses that conduct pain are modified at the spinal-cord level by various influences, including those com-

ing from the brain. Information concerning the nature and location of pain may be transmitted to the brain before the perception of pain, allowing the brain to modify the perception itself (at both brain and spinal-cord levels). Opiate and nonopiate instrinsic pain-control mechanisms are present. The distribution of opiate receptors in the limbic system, thalamus, and spinal cord implies that control of pain perception is closely related to emotional states and memory functions subserved by the limbic system. These mechanisms may provide a basis for known modifying influences on pain perception, such as the psychological state of the individual, his past experiences, and his cultural background.

Although the pain sensation threshold is very similar in most people, tolerance for and reaction to pain may be strongly influenced by psychological and social factors.

The *placebo effect* is found in approximately one third of the general population. The placebo response is a complex phenomenon that includes suggestion, anticipation, and conditioned responses. Endorphins are probably involved in the placebo response. Placebos are powerful and can *never* differentiate pain arising from tissue damage from "psychogenic" pain. Many nonspecific beneficial effects of the health-care system can be attributed to the placebo effect.

Psychogenic pain, pain in the absence of observable tissue damage, is a complex phenomenon. It might be due to increased sensitivity to otherwise mild or negligible pain as a concomitant or variant of depression or a conversion mechanism (e.g., symbolization of a psychological meaning). Since all pain sensation can be associated with a psychological meaning, the discovery of a psychological meaning or the presence of a "secondary gain" should not exclude the possibility that there might be an underlying "organic" disease in a patient complaining of pain. Pain-seeking behavior may be a conditioned response or may also be a motivated behavior with complex psychological meaning.

The *management* of pain requires a comprehensive approach in the biological, psychological, and social dimensions. In addition to the treatment of tissue damage, the cultural and psychological dimensions of the pain should be understood and treated appropriately.

Implications

For the Patient

Pain is a *subjective* experience that cannot be shared objectively. When a patient presents with pain, he is suffering not only from the sensation but also from anxiety that accompanies the pain. His reaction

to pain is influenced by a diversity of factors, such as cultural expectations, ethnic background, and personality. Thus, the probability that a certain level of pain will reach the limit of tolerance or anxiety for a given patient to result in help-seeking behavior is determined not only by the actual intensity of the stimulus but also by the social and psychological factors.

For the Physician

The existence or the absence of pain can never be proven in a given patient by a physician. Current understanding of the neurophysiology of pain clearly indicates that the *brain* has an important role in the perception of pain and that sensation of pain can occur without peripheral tissue damage. Whenever possible, *alleviation* of suffering from pain, regardless of the underlying pathology, can facilitate the formation of a cooperative doctor–patient relationship.

Attention to the *description* of pain is important in diagnosing the underlying condition.

An understanding of the cultural background and *social expectations*, as well as the patient's personality, obtained through *questions* and the patient's *demographic data*, helps the physician understand the differing reactions to pain in different patients and also can help him to anticipate and to take appropriate measures to forestall adverse reactions to pain that some patients might manifest. Adverse reactions may be nonreporting of pain in certain stoic individuals, as well as overreaction to it in others.

Because information about pain increases the tolerance for it, physicians should inform patients about potentially painful procedures and prepare them.

Because relief of pain by *placebos* occurs regularly in patients with severe tissue damage, placebos should *never* be used to differentiate organic from functional pain. The deceptive use of placebos in the hospital often undermines the patient's trust in the health-care personnel.

In treating a patient for pain relief, *adequate analgesics* should be given. Since narcotics probably interact with endogenous endorphin systems, some patients may require more narcotics for the relief of pain from the same stimulus if the endorphin system is inadequate or depleted. Narcotic addiction in the course of treatment with narcotic analgesics for pain is rare indeed, the incidence being less than 1%.

Pain may be experienced in the absence of tissue damage. This perception may be determined by *guilt feelings* (expiation), as a *body*

language expression of the need for emotional caring, as a result of *depression* with bodily preoccupation, or as a conditioned response to *anxiety*. Pain-prone patients often come from a milieu in which pain was used as a means of getting attention and love as well as of atoning for guilt feelings. Pain perceived in the absence of peripheral-tissue damage is usually attributed to a part of the body according to the patient's body image and the meaning of the body part, rather than according to the distribution of nerve pathways. Psychogenic pain, however, is often presented as an *added-on* symptomatology to an organic condition, and the presence of secondary gain, or psychological meaning attributable to pain, should not rule out the possibility that there might be an organic disease yet undetected.

For the Community and the Health-Care System

Differences in reaction to pain seen in different ethnic and cultural groups often result in a breakdown of communication between patients and health-care personnel. An understanding by health-care personnel of the cultural expectations of patients can help overcome this. Since anxiety often increases pain perception, the hospital *environment* should be such that patients are exposed to a minimum of anxiety-provoking situations.

The medical community should be aware that the use of narcotics in the treatment of pain is effective and when properly managed is not likely to result in addiction. Medical schools and hospitals should emphasize *adequate relief* of pain through the use of narcotic analgesics, rather than overemphasize the possibility of iatrogenic addiction.

Cultural change and assimilation may result in changes in pain experiences, and ethnic differences in reaction to pain may disappear as cultural stereotypes change.

Recommended Reading

Bonica JJ, Albe-Fessard DB (eds): *Advances in Pain Research and Therapy*, Vol 1. New York, Raven Press, 1976. A very comprehensive multiauthor volume on various aspects of pain, from neurophysiology to psychological factors and surgical management. An excellent up-to-date reference.

Engel GL: "Psychogenic" pain and the pain-prone patient. *Am J Med* **26:**899–918, 1959. A classic paper describing the possible nature of "psychogenic" pain and backgrounds of "pain-prone" patients.

Marks RM, Sachar EJ: Undertreatment of medical inpatients with narcotic analgesics. *Ann Intern Med* **78:**173–181, 1973. This is a must for medical students and physicians to

understand the degree of undertreatment of pain rampant in our hospitals. The conclusion is based on interviews with patients being treated for pain and on questionnaires completed by house staff physicians in two major hospitals in New York.

Snyder SH: Opiate receptors and internal opiates. *Sci Am* **236**:44–57, 1977. This is a comprehensive and lucid discussion of the endorphins (enkephalins) and their actions by one of the discoverers of the opiate receptors in the human brain. Recommended especially for those who would like to read more about the structures and mechanisms of action of the endorphins and the distribution of opiate receptors.

Sternbach RA: *Pain: A Psychophysiological Analysis*. New York, Academic Press, 1968. This is a comprehensive and concise monograph presenting clinical and experimental studies concerning pain. A beautifully written, well-integrated book that also deals with the psychology of pain.

Watkins LR, Mayer DJ: Organization of endogenous opiate and nonopiate pain control systems. *Science* **216**:1185–1192, 1982. This is a comprehensive and interesting review of experimental data concerning the intrinsic pain-control systems and their clinical implications.

References

Akil H, Liebeskind JC: Monoaminergic mechanism of stimulation-produced analgesia. *Brain Res* **94**:279–296, 1975.

Beall JE, Martin RF, Applebaum AE, *et al.*: Inhibition of primate spino-thalamic tract neurons by stimulation in the region of the nucleus raphe magnus. *Brain Res* **114**:328–333, 1976.

Beecher HK: The powerful placebo. *J Am Med Assoc* **159**:1602–1606, 1955.

Beecher HK: Generalization from pain of various types and diverse origins. *Science* **130**:267–268, 1959a.

Beecher HK: *Measurement of Subjective Responses*. New York, Oxford Press, 1959b.

Beecher HK: Increased stress and effectiveness of placebos and "active" drugs. *Science* **132**:91–92, 1960.

Bloom F, Segal D, Ling N, *et al.*: Endorphrins: Profound behavioral effects in rats suggest new etiological factors in mental illness. *Science* **194**:630–632, 1976.

Blumer D, Heilbronn M: Chronic pain as a variant of depressive disease. *J Nerv Ment Dis* **170**:381–406, 1982.

Bond MR: The relation of pain to the Eysenck Personality Inventory, Cornell Medical Index, and Whitely Index of Hypochondria. *Br J Psychiatry* **119**:671, 1971.

Coulter JC, Maunz RA, Willis WD: Effects of stimulation of sensory motor cortex on primate spinothalamic neurons. *Brain Res* **65**:351–356, 1974.

Engel GL: "Psychogenic" pain and the pain-prone patient. *Am J Med* **26**:899–918, 1959.

Eysenck SBG: Personality and pain assessment in childbirth of married and unmarried mothers. *J Ment Sci* **107**:417–430, 1961.

Goldstein A: Opioid peptides (endorphins) in pituitary and brain. *Science* **193**:1081–1086, 1976.

Guyton AC: *Textbook of Medical Physiology*. Philadelphia, WB Saunders, 1976.

Handwerker HO, Iggo Z, Zimmerman M: Segmental and supraspinal actions on dorsal horn neurons responding to noxious and non-noxious skin stimuli. *Pain* **1**:147–165, 1975.

Herrnstein RJ: Placebo effect in the rat. *Science* **138**:677–678, 1962.

Jones A, Bentler PH, Petry G: The reduction of uncertainty concerning future pain. *J Abnorm Psychol* **71**:87–94, 1966.

Kanfer FH, Goldfoot DA: Self-control and tolerance of noxious stimulation. *Psychol Rep* **18**:79–85, 1966.

Kerr FWL, Wilson PR: Pain. *Annu Rev Neurosci* **1**:83–102, 1978.

Kosambi DD: Living prehistory in India. *Sci Am* **216**:105–114, 1967.

Kroeber AL: *Anthropology*. New York, Harcourt-Brace, 1923.

Lasagna L, Mosteller F, von Felsinger JM, et al.: A study of the placebo response. *Am J Med* **16**:770–779, 1954.

Levine JD, Gordon NC, Fields HL: The mechanism of placebo analgesia. *Lancet* **2**:654–657, 1978.

Lynn R, Eysenck HJ: Tolerance for pain, extraversion, and neuroticism. *Percept Mot Skills* **12**:161–162, 1961.

Marks RM, Sachar EJ: Undertreatment of medical inpatients with narcotic analgesics. *Ann Intern Med* **78**:173–181, 1973.

Marx JL: Analgesia: How the body inhibits pain perception. *Science* **195**:471–473, 1977.

McGlashan TH, Evans FJ, Orne MT: The nature of hypnotic analgesia and placebo response to experimental pain. *Psychosom Med* **31**:227–246, 1969.

Melzack R: *The Puzzle of Pain*. New York, Basic Books, 1973.

Melzack R, Torgerson WS: On the language of pain. *Anesthesiology* **34**:50–59, 1971.

Melzack R, Wall PD: Pain mechanisms: A new theory. *Science* **150**:971–979, 1965.

Merskey H: The characteristics of persistent pain in psychological illness. *J Psychosom Res* **9**:291–298, 1965a.

Merskey H: Psychiatric patients with persistent pain. *J Psychosom Res* **9**:299–309, 1965b.

Merskey H, Spear FG: The reliability of the pressure algometer. *Br J Clin Psychol* **3**:130–136, 1964.

Oliveras JL, Besson JM, Builbaud G, et al.: Behavioral and electrophysiological evidence of pain inhibition from midbrain stimulation in the cat. *Exp Brain Res* **20**:32–44, 1974.

Pavlov IP: *Conditioned Reflexes: An Investigation of the Physiological Activity of the Cerebral Cortex* (Anrep GV, transl–ed). London, Oxford University Press, 1927.

Pavlov IP: *Lectures on Conditioned Reflexes* (Gantt WH, transl). New York, International Publishers, 1928.

Pearson DH: Pain pathways: An overview. *Hosp Pract* (special report), Jan 14–18, 1976.

Perl ER: Sensitization of nocioreceptors and its relation to sensation, in Bonica JJ, Albe-Fessard DG (eds): *Advances in Pain Research and Therapy*. New York, Raven Press, 1976, Vol 1.

Pilowsky I: Pain and its management in malignant disease. *Psychosom Med* **31**:400–404, 1969.

Robinson H, Kirk RF, Frye RF, et al.: A psychological study of patients with rheumatoid arthritis and other painful diseases *J Psychosom Res* **16**:53–56, 1972.

Shapiro AK: Attitudes toward the use of placebos in treatment. *J Nerv Ment Dis* **130**:200–211, 1960.

Snyder SH: Opiate receptors and internal opiates. *Sci Am* **236**:44–57, 1977.

Sternbach RA: *Pain: A Psychophysiological Analysis*. New York, Academic Press, 1968.

Sternbach RA, Tursky B: Ethnic differences among housewives in psychophysical and skin potential responses to electric shock, *Psychophysiology* **1**:241–246, 1964.

Timmermans G, Sternbach RA: Factors of human chronic pain: An analysis of personality and pain reaction variables. *Science* **184**:806–808, 1974.

Ulrich RE, Hutchinson RR, Azrin NH: Pain-elicited aggression. *Psychol Rec* **15**:111–126, 1965.

Vernon J, Ulrich RE: Classical conditioning of pain-elicited aggression. *Science* **152:**668–669, 1966.

VonKnorring L: The experience of pain in depressed patients. *Neuropsychobiology* **1:**155–165, 1975.

Wall PD: Modulation of pain by non-painful events, in Bonica JJ, Albe-Fessard D (eds): *Advances in Pain Research and Therapy*, Vol 1. New York, Raven Press, 1976.

Ward NG, Bloom VL, Friedel RO: The effectiveness of tricyclic antidepressants in the treatment of coexisting pain and depression. *Pain* **7:**331–341, 1979.

Watkins LR, Mayer DJ: Organization of endogenous opiate and nonopiate pain control systems. *Science* **216:**1185–1192, 1982.

Winsberg B, Greenlick M: Pain response in Negro and white obstetrical patients. *J Health Soc Behav* **8:**222–227, 1967.

Woodforde JM, Merskey H: Personality traits of patients with chronic pain. *J Psychosom Res* **16:**167–172, 1972.

Woodrow KM, Friedman CD, Siegelaub AB, *et al.*: Pain tolerance: Differences according to age, sex, and race. *Psychosom Med* **34:**548–556, 1972.

CHAPTER 10

Sleep and Dreaming

1. A 40-year-old married woman underwent hysterectomy for uterine fibroids. A few weeks after discharge, she began to experience increasing difficulty in falling asleep. She would also awaken frequently in the middle of the night and wake up at 4:00 A.M. unable to fall asleep again. She felt fatigued, irritable, and unable to concentrate. A depressive syndrome ensued.

2. A 60-year-old widower was admitted to the hospital for elective surgery. During history-taking, the intern found out that the patient had been taking moderately large doses of secobarbital for sleep at night for the past year or so. Also, he would have one or two "shots" of brandy. The intern decided that perhaps the patient should be taken off the sleep medications and, of course, should receive no alcohol at the hospital. The next night, at 3:00 A.M., the patient rushed to the nurses' station, frightened and perspiring, stating that he had had a most vivid and horrible nightmare.

3. A 47-year-old married woman suffering from advanced rheumatoid arthritis became increasingly irritable, complaining of pain and inability to sleep. The irritability subsided when she was prescribed small doses of flurazepam for sleep.

4. Freud's "Dream of the Botanical Monograph": "I had written a monograph on a certain plant. The book lay before me and I was at the moment turning over a folded coloured plate. Bound up in each copy there was a dried specimen of the plant, as though it had been taken from a herbarium" (Freud, 1900/1965).

241
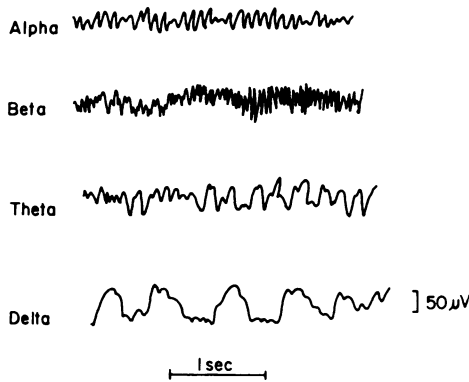
Figure 18. Different types of normal electroencephalographic waves. (From Guyton, 1976. Copyright 1976 by WB Saunders Co. Reproduced with permission from the publisher and author.)

In vignettes 1 through 3, a sleep disturbance heralded a psychiatric illness. Changes in drug regimen can cause sleep disturbance involving dreams, as in vignette 2. Vignette 3 illustrates the interaction between a medical disease and sleep difficulties. Vignette 4 is a famous dream of Freud's that was one of the first dreams analyzed to show its psychological meanings (see Freud, 1900/1965).

Sleep was thought to be a relatively simple and uniform state until the advent of electroencephalographic techniques (see Figures 18–20). During the waking state, the EEG typically shows low-voltage fast waves (beta waves, $\geq$ 14 Hz). When the individual is in a relaxed, awake state, the EEG may show alpha waves (9–13 Hz), especially when the eyes are closed. During sleep, the EEG is characterized by slower waves than in the waking state (see below). Aserinsky and Kleitman (1953) discovered in the 1950s that conjugate, rapid eye movements (REM) appear periodically during sleep. It soon became clear that dreams, especially visual dreams, occurred predominantly during the REM periods.

Stages of Sleep

Typically, in a night's sleep, a person goes through a period of non-REM (NREM) sleep before having his first REM sleep of the night.
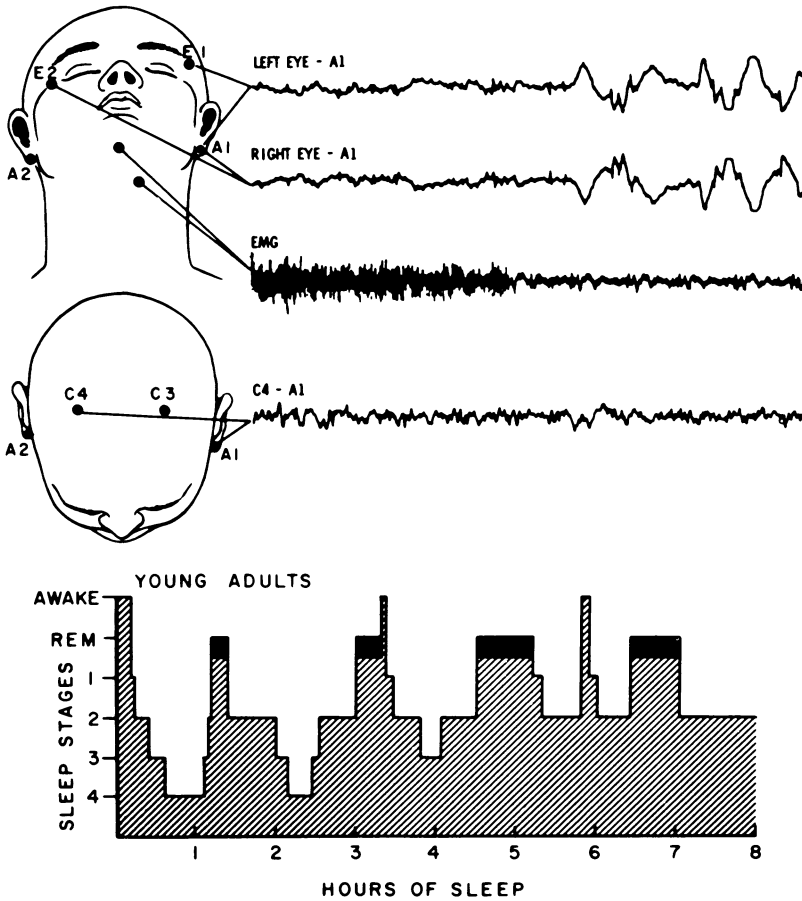
Figure 19. A: Electrode placement in sleep research. The top two tracings show eye movements recorded from electrodes attached laterally to the outer canthus of each eye (electrooculogram) and referred to the ear; this produces out-of-phase deflections in the two tracings for almost all eye movements. The electromyogram (EMG) is recorded from electrodes attached firmly beneath the chin and referred to each other. In the lower tracing, the EEG is derived from a scalp placement referred to the opposite ear. The recordings illustrate the onset of an REM sleep period. First, the EMG decreases sharply; then eye movements appear while the electroencephalographic waves change to low-amplitude, mixed-frequency. (From Rechtschaffen and Kales, 1968.) B: Nocturnal sleep pattern in young adults. Note the absence of stage 4 and the decreased length of NREM periods during the latter part of the night and the short first REM period. (From Kales, 1968. Reproduced with permission from the American college of Physicians and the author.)

Awake – low voltage – random, fast

50 µV

1 sec

Drowsy – 8 to 12 cps – alpha waves

Stage 1 – 3 to 7 cps – theta waves

Theta Waves
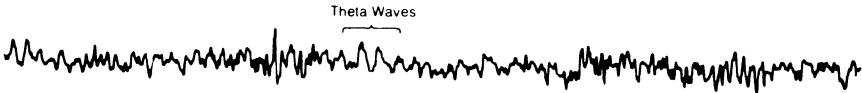
Stage 2 – 12 to 14 cps – sleep spindles and K complexes

Sleep Spindle

K Complex –
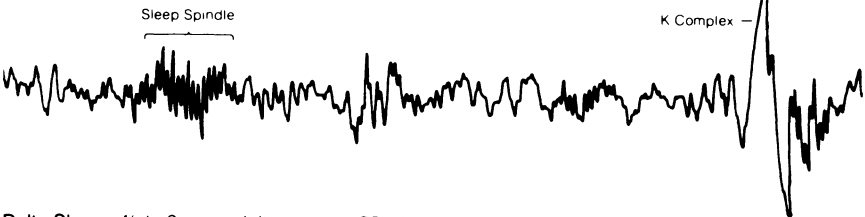
Delta Sleep – ½ to 2 cps – delta waves >75 µV
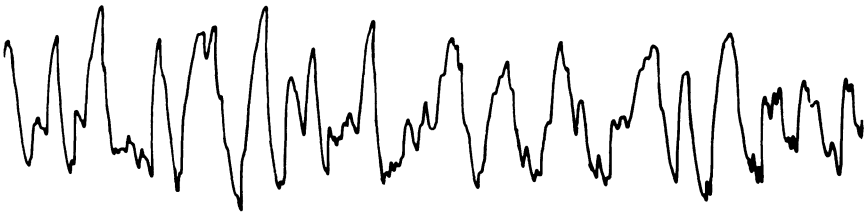
REM Sleep – low voltage – random, fast with sawtooth waves

Sawtooth Waves   Sawtooth Waves

Figure 20. Human sleep stages. (From Hauri, 1977. Copyright 1977 by the Upjohn Co. Reproduced with permission from the publisher and author.)

NREM Sleep

Electroencephalographically, four stages can be identified during NREM sleep: stages 1 through 4. Stages 3 and 4 are sometimes called *delta* or slow-wave sleep (see Figures 19 and 20).

Stage 1. This is a transition phase between wakefulness and sleep. The EEG shows low-voltage, mixed-frequency waves with theta (4–8 Hz) predominating. During this phase, thoughts begin to drift, and thinking is no longer reality-oriented. Short dreams often develop (Foulkes and Vogel, 1965). Many people subjectively feel that they are awake during stage 1 (Hauri, 1977). Stage 1 is usually quite short, in the range of 0.5–7 min.

Stage 2. As sleep progresses, *sleep spindles*, bursts of 12–16 Hz lasting 0.5–2 sec, appear with low-amplitude, fast-frequency activity. K complexes, which are well-delineated, slow, negative EEG deflections followed by a positive component, also appear. Mentation during this stage consists of short, mundane, and fragmented thoughts (Foulkes, 1962). In general, normal adults spend more sleep time in stage 2 than in any other stage.

Delta Sleep (Stages 3 and 4). When the EEG shows at least 20% delta waves (frequency < 4 Hz), delta sleep is reached. Delta sleep may be subdivided into stages 3 (20–50% delta) and 4 (>50% delta). The majority of delta-wave sleep occurs early in the night, when sleep is deepest.

REM Sleep

After the person has been asleep for 80–100 min in NREM sleep, REM sleep appears. During this phase, there occur rapid eye movements that can be demonstrated by the electrooculogram (EOG) (see Figure 19). The *EEG shows a desynchronized pattern similar to stage 1 sleep*, except that "sawtooth" waves are also present (Figure 20). The REM phase is about 5–20 min in duration and alternates with the NREM phase approximately every 90 min. Initially in the night, the REM phase may be skipped or very brief. In addition to *rapid eye movement*, there are several other *characteristic features of REM sleep:* The skeletal muscles are relaxed (Figure 19), although there may be occasional twitches; there is *physiological activation* manifested by irregular (highly variable) heart rate,
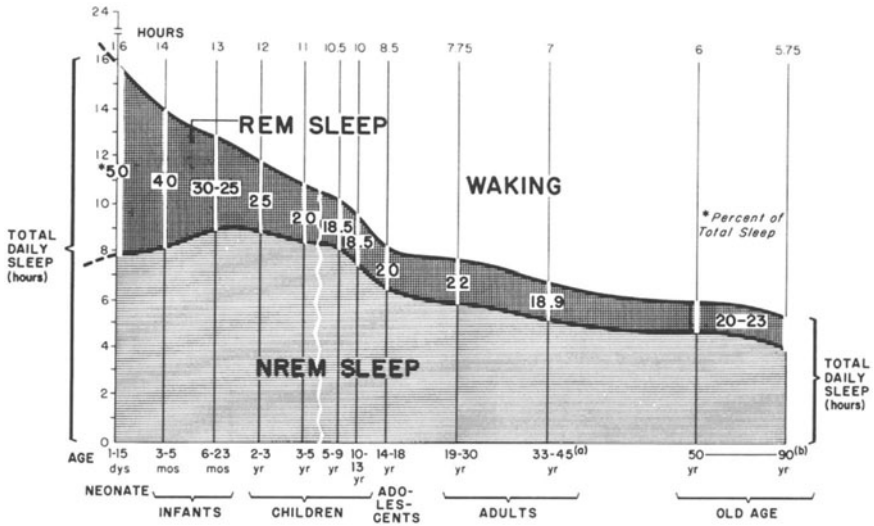
Figure 21. Graph showing changes (with age) in total amounts of daily sleep, REM sleep, and in percentage of REM sleep. Note sharp diminution of REM sleep in the early years. REM sleep falls from 8 hr at birth to less than 1 hr in old age. The amount of NREM sleep throughout life remains more constant, falling from 8 hr to 5 hr. In contrast to the sleep decline of REM sleep, the quantity of NREM sleep is undiminished for many years. Although total daily REM sleep falls steadily during life, the percentage rises slightly in adolescence and early adulthood. This rise does not reflect an increase in amount; it is due to the fact that REM sleep does not diminish as quickly as total sleep. (Revised Figure from Roffwarg H, *et al.*, 1966. Reproduced with permission from the author and publisher.)

respiratory rate, and blood pressure; penile erection also occurs in men. About 80% of subjects awakened during REM are usually able to recall *dreams*, while visual dreams are recalled in less than 20% during NREM awakenings.

The *total time spent in REM* during a typical night is about 20–25% of sleep time; stage 2, about 50%; stage delta, about 20%; and stage 1, 5–10% (see Figure 19B). The amount of *total REM sleep* time *decreases with age*, so that in the neonate, about 50% of total sleep time is spent in REM, in young adults, 20–25%, and in older adults, about 15% (see Figure 21).

Basic Rest–Activity Cycle

Kleitman (1965) proposed that the 90-min REM–NREM cycle represents a *basic rest–activity cycle* (BRAC) that reflects a fundamental pe-

Table 14. REM State in Various Mammalian Species
(Young Adults)[a]

Species	Average length of REM period (min)	Average length of cycle (min)	REM time (% of total sleep)
Human	14	80–90	20–24
Monkey	4–10	40–60	11–20
Cat	10	20–40	20–60
Sheep	—	—	2–3
Rabbit	—	24	1–3
Rat	4–7	7–13	15–20
Mouse	—	3–4	—
Opossum	5	17	22–40

[a] From Hartmann (1970). Copyright 1970 by Little, Brown & Co. Reproduced with permission from the publisher and author. The table is based on data from the work of many authors.

riodicity in the activity of the central nervous system of homothermal animals. He proposed that this particular cycle was easier to discern during sleep because of the EEG changes. There is some current evidence that certain types of activities, including "oral activity" such as eating, drinking, and smoking, as well as waking gastric contractility, follow 90- to 120-min ultradian rhythms (Friedman and Fisher, 1967).

Interestingly, the length of the rest–activity cycle seems to be proportional to the size of the animal species, running from 7–13 min in the rat to 2 hr in the elephant (Table 14).

Biological Rhythms*

The sleep–wake cycle and REM–NREM cycle represents but two of the many rhythmic activities that characterize life. The period of the rhythm may be a day (*circadian*), such as in the sleep–wake cycle; within the daytime (*diurnal*), as in the mood change of depressives between the morning and evening hours; or within the nighttime (*nocturnal*). The period may be greater than 24 hr (*infradian*), such as the menstrual cycle, or less than 24 hr (*ultradian*), as in the REM–NREM cycle.

Many endocrine and metabolic systems follow rhythmic variations, circadian, infradian, or ultradian (Curtis, 1972) (see Figure 22). Circadian

* For a comprehensive review of biological rhythms, the reader is referred to the monograph *Biological Rhythms in Psychiatry and Medicine* (see Recommended Reading).
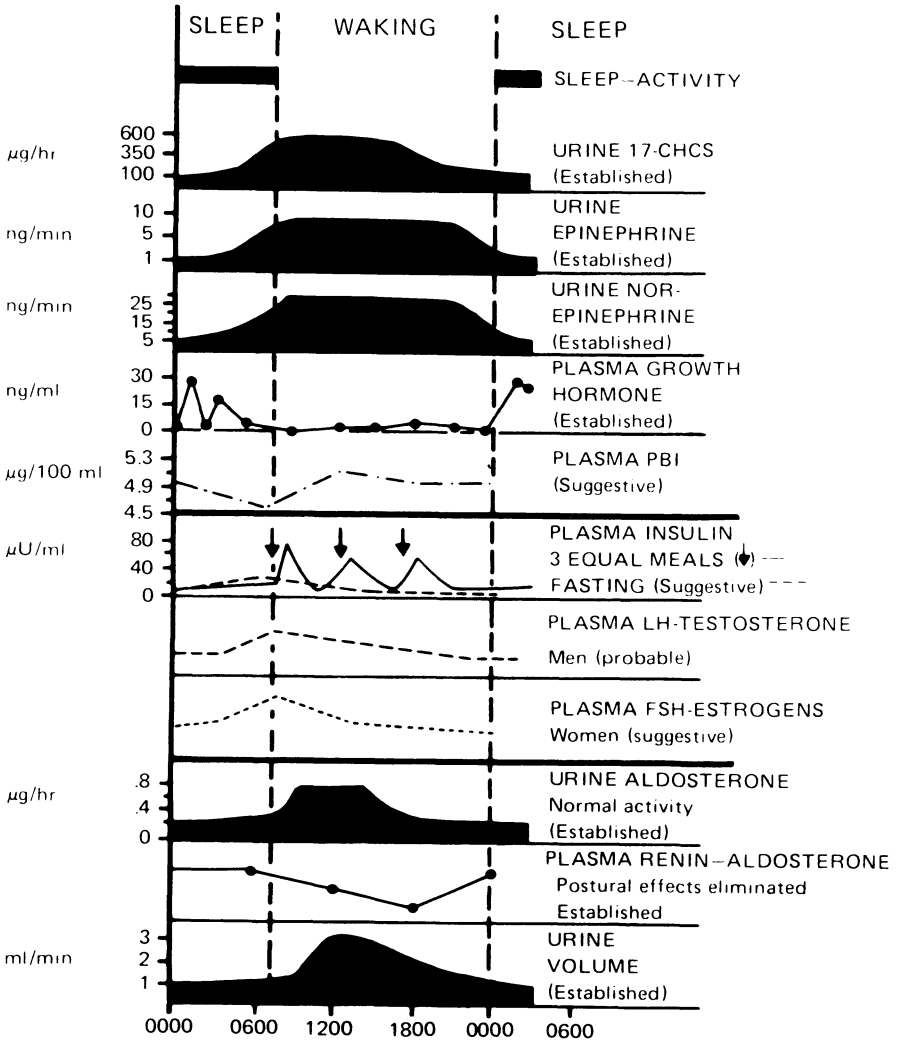
Figure 22. Phase map of daily cycle of endocrine measures and urine volume in humans in relation to habitual sleep and feeding schedule. (From Curtis, 1972. Reproduced with permission from the publisher and author.)

rhythms have been observed in cell mitosis of different organs as well as in the susceptibility of the organism to infectious and toxic agents.

There is evidence that the suprachiasmatic nucleus, which is located above the optic chiasm, provides the "clock" for most circadian rhythms, including the sleep–wake cycle (Hanada and Kawamura, 1981).

An *implication* of this observation of rhythmicity in the biological systems is that the time of the day has to be controlled in all biological experiments, measurements, and interventions. For example, animals have been shown to exhibit the fewest toxic effects to pharmacological agents if the agents are administered before the sleep period. Changes in the rhythmicity of functional systems such as disruption in sleep and REM may be early evidence of dysfunction.

Brain Mechanisms of Sleep

The brain mechanisms of sleep and dreaming are not yet completely understood. *Arousal and wakefulness* seem to depend on the activity of the *ascending reticular activating system*, which is predominately cholinergic [see Figure 7 (Chapter 4)]. The ascending reticular activating system is distributed diffusely throughout the brain stem and limbic system and determines the general tone and arousal of the central nervous system. Continuing high activity of the system inhibits sleep.

Sleep does not automatically occur when sensory input is low. *NREM sleep* is an active phenomenon probably brought about by the activation of the serotonergic neurons of the pontine raphe system [see Figure 12 (Chapter 6)]. Lesions of the raphe nuclei lead to a state of relatively permanent cortical desynchronization associated with behavioral arousal. Drugs that deplete serotonin levels in the brain, such as *p*-chlorophenylalanine (PCPA), cause a reduction in sleep time in animals, involving both slow-wave and REM sleep (Morgane and Stern, 1974). Jouvet (1972a,b) proposed that *serotonergic systems* are involved in *slow-wave sleep* and *noradrenergic* and possibly *cholinergic systems* in *REM and waking*. Jouvet noted that the types of insomnia caused by lesioning of the serotonergic neurons or by drug-induced inhibition of serotonin synthesis have one common characteristic: The decrease in sleep is not followed by a subsequent rebound of sleep. Insomnia can also be caused by activation of the ascending noradrenergic pontomesencephalic system, either by direct stimulation of the mesencephalon, by nociceptive stimuli, by sleep deprivation, or by drugs such as amphetamines. In these instances, either total insomnia or selective suppression of REM occurs, but never a selective suppression of slow-

wave sleep. This type of insomnia is always followed by a rebound of slow-wave or REM sleep or both, the intensity and duration of which are proportional to the duration of the insomnia. This suggests that in this form of insomnia, there may be an increased activity of the "waking system" that affects the biosynthesis and turnover of the serotonin system. Jouvet noted that both forms of insomnia share one common characteristic: They can be suppressed by the inhibition of the synthesis of catecholamines by α-methyl-*p*-tyrosine (AMPT), which decreases the turnover of central catecholamines. There is also evidence that an increase in the serotonin turnover results in true hypersomnia (increases in both slow-wave and REM sleep).

In cats, intravenous injection of the serotonin precursor 5-hydroxy-tryptophan is followed within minutes by a state resembling slow-wave sleep. Reserpine, which depletes both norepinephrine and serotonin in the brain, can suppress both slow-wave and REM sleep totally in the cat. 5-Hydroxy-tryptophan, when given a few hours after reserpine, will restore slow-wave sleep but not REM sleep. Morgane and Stern (1974) conclude that *the presence of serotonin in the brain is a necessary condition for the patterns of sleep, both slow-wave and REM, to become fully manifest.*

Jouvet (1972a,b) found that lesions in the dorsolateral part of the pontine reticular formation totally suppress REM sleep without interfering significantly with slow-wave sleep. Since dorsolateral pontine tegmentum contains the noradrenergic nuclei, *locus ceruleus* [see Figure 23 and also Figures 6 (Chapter 4) and 10 (Chapter 6)] and subceruleus, these structures are considered to be implicated in REM sleep. Bilateral lesions of the caudal part of the locus ceruleus suppress the inhibition of skeletal muscles (relaxation) that occurs during REM sleep. Bilateral total lesions of the locus ceruleus and subceruleus are followed by a decrease in waking and permanent suppression of REM sleep. Following this type of lesion, there is a marked fall in norepinephrine concentration in all parts of the brain.

Hobson (Hobson, 1975, 1978; Hobson and McCarley, 1977) postulates *reciprocal interaction* between the *pontine reticular giant cells (cholinergic)* on one hand and the *noradrenergic locus ceruleus* and the *serotonergic raphe neurons* on the other. He proposes that when the pontine reticular giant cells are activated, the REM state occurs. He also postulates that the cholinergic giant cells are excitatory to themselves and also to the group of monoaminergic neurons, locus ceruleus and raphe nuclei. On the other hand, the monoaminergic neurons (locus ceruleus and raphe) are considered to be inhibitory to themselves as well as to the reticular giant cells (see Figure 24). This model results in an oscillation in activation between the cholinergic giant cells and the monoam-
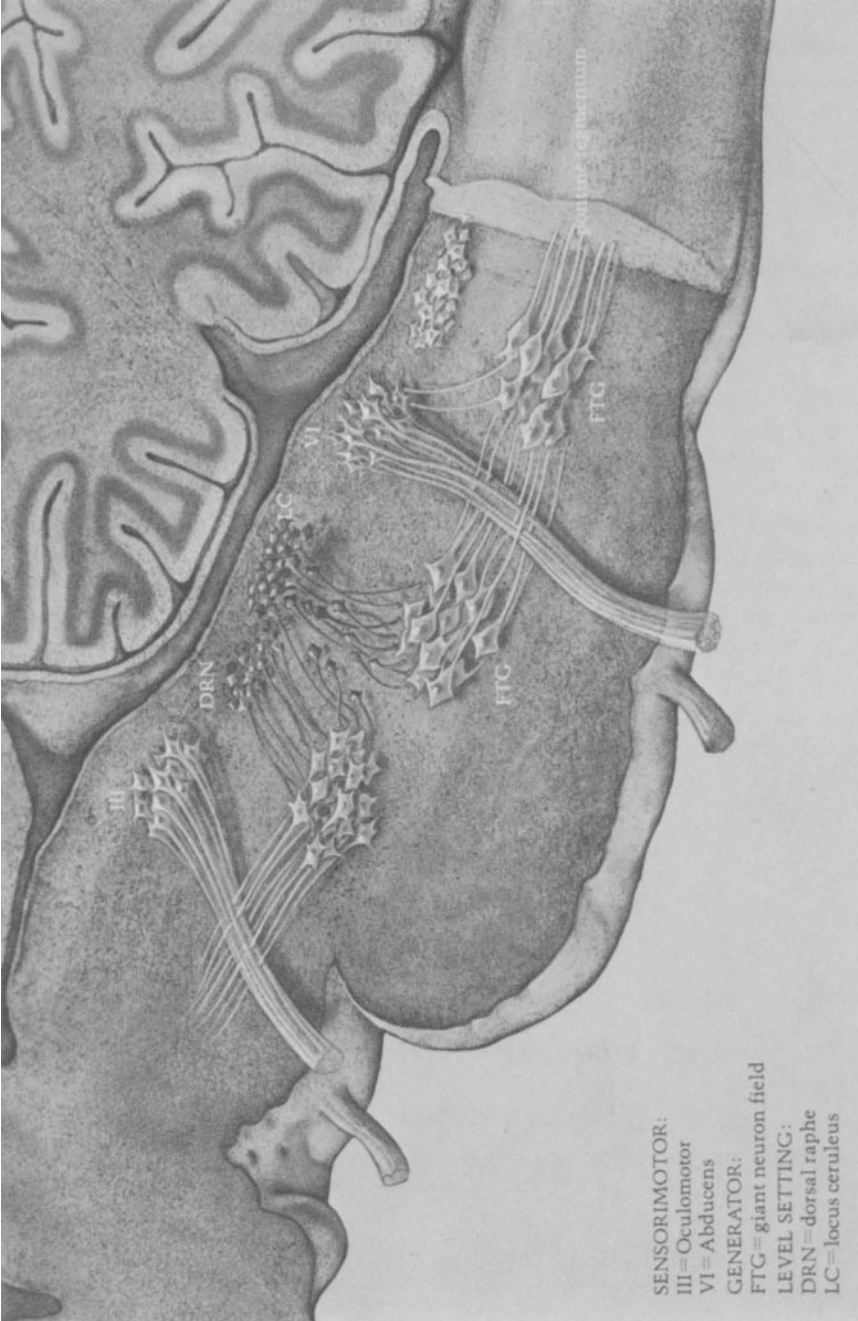
Figure 23. Schematic view of sagittal section of the midbrain and pons illustrating the "dream-state generator." (From Hobson, 1978. Reproduced with permission from Hoffmann-La Roche, Inc, and the author.)
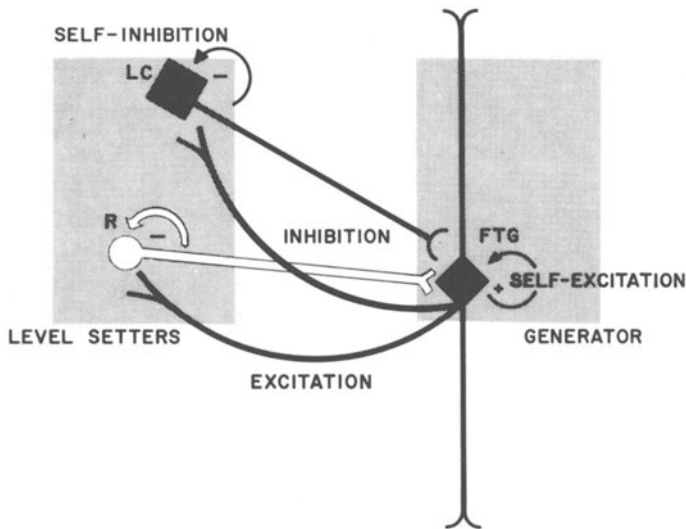
Figure 24. Dream-state generator. Schematic diagram of proposed reciprocal connections between the monoaminergic, "level-setting" neurons in the raphe and locus ceruleus nuclei and the cholinergic, "generator" neurons of the gigantocellular tegmental field. The self-inhibiting and self-exciting collaterals of both groups are also shown. Abbreviations: FTG, giant neuron field; LC, locus ceruleus; R, raphe nucleus. (From Hobson, 1978. Reproduced with permission from Hoffmann-La Roche, Inc, and the author.)

inergic neurons. Hobson and McCarley (1977) found that microinjection of Carbachol, a cholinomimetic substance, into the pontine giant-cell zone potently enhanced the REM state and simultaneously activated giant cells. Stimulation of locus ceruleus had an inhibitory effect on the giant cells.

Hobson (Hobson and McCarley, 1977; Hobson, 1978) discussed the time sequence in the generation of the REM state. In essence, changes in the rate of firing of the pontine reticular giant cells preceded the cortical changes, such as EEG desynchronization, as well as the eye movements. Further, eye movements occurred before EEG desynchronization (cortical activation). The implication of these findings is that *the REM state seems to be primarily generated by the pontine oscillating systems, and the eye movements and dreaming* (cortical activation) *seems to be secondary phenomena to the dream (REM) state* (see Figure 25).

There is controversy concerning the completeness of any of the hypotheses concerning the brain mechanisms of sleep and dreaming. For example, NREM sleep may be governed by an interaction of sero-
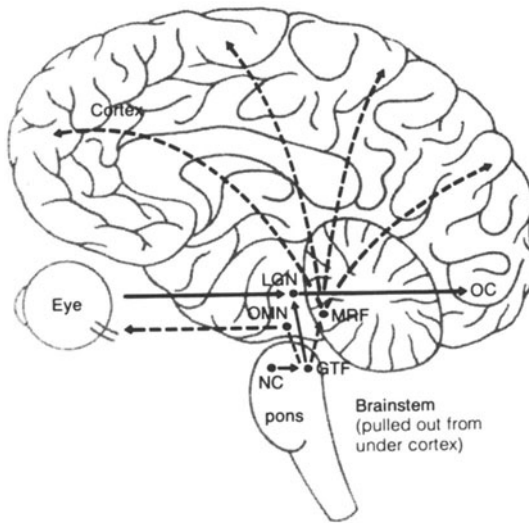
Figure 25. Schematic diagram of the REM system. From the nucleus (NC) to the gigantocellular tegmental field (GTF): (1) discharges from the NC usually inhibit discharges from the GTF during waking and NREM sleep; (2) the neurotransmitter involved in this inhibition appears to be aminergic; (3) this inhibition breaks down just before and during REM, liberating the GTF cells to discharge spikes. From the GTF via the lateral geniculate nucleus (LGN) to the occipital cortex (OC)—the PGO spike: (1) discharges from the GTF enter the LGN, a halfway station on the visual pathway from the retina to the OC; (2) via the LGN, these discharges then enter the OC, where they are apparently interpreted as coming from the retina (seen as pictures in our dreams); (3) because the pathway GTF→LGN→OC was the first one discovered, the discharges are called pontine–geniculate–occipital, or PGO, spikes. Other discharges from the GTF: The discharges from the GTF enter a multitude of other neuronal structures, apparently causing many of the typical phenomena related to REM sleep. Only two are outlined here: (1) discharges enter the oculomotor nuclei (OMN) and cause eye movements; (2) discharges enter the midbrain reticular formation (MRF) and cause general desynchronization of the cortex typical for REM sleep. (From Hauri, 1977. Copyright 1977 by the Upjohn Co. Reproduced with permission from the publisher and the author.)

tonergic, aminergic, and cholinergic neurons, rather than by serotonergic neurons alone (Monnier and Gaillard, 1980). Other neurotransmitters may also be involved in both REM and NREM sleep. For example, factor S is a small peptide isolated from the brain of sleep-deprived goats, rabbits, and cattle. Factor S causes excess delta sleep for several hours if injected into the ventricles of animals (Hauri, 1982) REM-triggering protein has also been isolated (Druckner-Colin *et al.*, 1980).

Dreams

Freud (1900/1965) proposed that dreams represented *fulfillment of wishes* in the form of hallucinations during sleep, motivated by the sleeper's desire to continue sleep. A thirsty sleeper, for example, would dream of drinking water rather than interrupt his sleep to fetch a glass of water. Freud further postulated that wishes that were pushed out of one's awareness during waking life because of their unacceptable nature find fulfillment in dreams, occasioned by the weakening, in the state of sleep, of the part of the personality structure concerned with censoring out unacceptable impulses (*superego*). Thus, during sleep, normally forbidden wishes might be fulfilled through hallucinations. According to Freud, the normally unconscious wishes are not directly transformed to dreams, but undergo distortions and disguises so that they might appear somewhat more acceptable, that is, so as not to alarm the dreamer's personality system to the extent that he would have to interrupt his sleep.

This process of constructing dreams out of unconscious wishes was called by Freud the *dream work*. The dream work involves such processes as compressing several ideas and thoughts into a single image (*condensation*), *displacing* feelings from the original object to another object, and *reversing* feelings or ideas to their opposites. An example of condensation is dreaming of a face that is a composite of features belonging to several different persons. Through reversal, night may be represented in dreams as broad daylight, and very emotional scenes may appear to be quite tranquil.

Recent experiences (especially of the previous day) often appear in dreams, although the particular experience (or image, such as the face of a person) may have been quite incidental. Freud called this the *day's residue*. The day's residue was thought to be otherwise neutral images or experiences that could be exploited to represent unconscious wishes, and it was used as readily available "material" with which to construct the dreams, which are wish fulfillments.

In his classic study of dreams, Freud constructed a purely psychological theory based on psychological data alone, because physiological information concerning sleep and dream states was then largely nonexistent. One of the most important aspects of his discoveries regarding dreams is the demonstration that thoughts rendered in dreams have meaning in relation to the person's ongoing mental life.

Dream research has been revolutionized since the discovery of the association between REM sleep and dreaming. As previously mentioned, in about 80–90% of REM awakenings, dream reports are elicited.

The dreams that occur during the REM periods seem to be especially *visual* and detailed ones. Some thinking or conscious activity also occurs during the NREM periods, but this type of NREM "mentation" usually lacks the visual and elaborate quality of the REM dreams. NREM "dreams" tend to be more the auditory–thinking type of conscious activity, which is usually lacking in detail and vividness.

The content of dreams collected by REM awakenings in the laboratory is rather mundane and not necessarily as bizarre and fantastic as some of the remembered dreams. Many of the dreams that occur relatively early during sleep have content that is temporally closer to the present, while dreams that occur later, toward the morning, seem to contain more material related to early life (Van de Castle, 1970).

According to Hobson, the discovery that the brain electrical activity associated with the REM state (pontine discharges) occurs before cortical activation or oculomotor activation seems to prove that *REM is an autonomously generated state* and not a reaction to an internal or external stimulus threatening the continuation of sleep (Hobson and McCarley, 1977; Hobson, 1978). Consistent with this scheme is the idea that the *content* of a dream is constructed by the cerebral cortex in the presence of the ascending signals from the REM-state generator in the pons. The dream content, then, is closely linked to the ongoing thought processes of the person, as first noted by Freud.

It is significant that only some dreams are remembered, and it is the remembered dreams that are analyzed in psychotherapy. Perhaps, in psychotherapy, *certain dreams are selectively remembered or not remembered* because their content is especially related to experiences, wishes, and images that have particularly important meanings in the context of therapy. These psychological meanings, then, can be explored in psychotherapy.

Functions of Sleep and Dreaming

Hartmann (1973) postulates that the function of *slow-wave* sleep is *physiological restoration*. Studies show that when sleep has been deprived, slow-wave sleep is made up at the first opportunity, followed by restoration of REM sleep. After exercise, slow-wave sleep also increases. Hartmann also hypothesizes that during slow-wave sleep, proteins or RNA or both are synthesized, especially in the central nervous system (Table 15).

According to Hartmann, the functions of *REM sleep* are related to the brain systems having to do with *focused attention*, the ability to maintain an optimistic mood, energy, and processes of *emotional adaptation*

Table 15. Functions of Slow-Wave (S) and REM (D) Sleep[a]

Data Base	Functions	
	Functions of S	
Hints from physiology—chemistry of sleep	Anabolism: macromolecule (RNA or protein) synthesis	Anabolism and synthesis of macromolecules to be used partially in the functions of D
Sleep deprivation	Prevent lethargy or physical tiredness	
Sleep as a response	Restoration after exercise, pain or injury, or excessive catabolism	
Psychology of tiredness	Restoration after "physical" tiredness	
	Functions of D	
Hints from physiology	Repatterning	Repair, reorganization, formation of new connections in cortex and the catecholamine systems ascending to cortex required for optimal attention mechanisms, secondary process, and self-guidance during waking
Sleep deprivation	Focus attention and keep out extraneous stimuli; maintain ego integrity; restore ability for new learning; repattern or consolidate memories	
Long and short sleepers	Restoration after new learning and "psychic strain" including anxiety and depression	
Variable sleepers		
Age changes and pathological states	Restoration at times of new learning and at times of irritability and depression	
Sleep as a response	Restoration of catecholamine systems; restoration after reticular stimulation or hypervigilance; restoration after new learning	
Psychology of tiredness	Restore recent, subtle ego mechanisms and secondary process	
The dream	Shunting out for repair (during D) of certain brain systems necessary for flexible attention, subtle feedback-regulated emotion, continuing sense of self	

[a] From Hartmann (1973). Reproduced with permission from the publisher and author.

to the environment. He also postulates that REM sleep may have a role in *consolidating learning or memory* and in *restoring the catecholamine levels in the brain* (Hartmann, 1973).

Sleep Needs

While the need for sleep seems to change with age (see Figure 21), there is a great *individual variation.* Thus, some individuals sleep for 10–12 hr a night, while others habitually sleep for as little as 1–3 hr a night without any ill effects (Hauri, 1977).

Sleep-Deprivation Studies. Complete sleep deprivation for an entire night usually results in little decrement in performance in young, healthy volunteers (Johnson, 1969). If the total sleep deprivation is continued for two or three nights, small *"microsleeps"* of a few seconds' duration begin to intrude into wakefulness. The microsleeps interfere with attention and, thus, performance. If an individual is totally deprived of sleep for more than three days, microsleeps become longer and more frequent, so that by about ten days, it becomes hard to tell whether a subject is asleep or awake, even if he performs functions normally associated with being awake, such as walking and talking.

When sleep-deprived individuals are allowed to sleep, there is in-
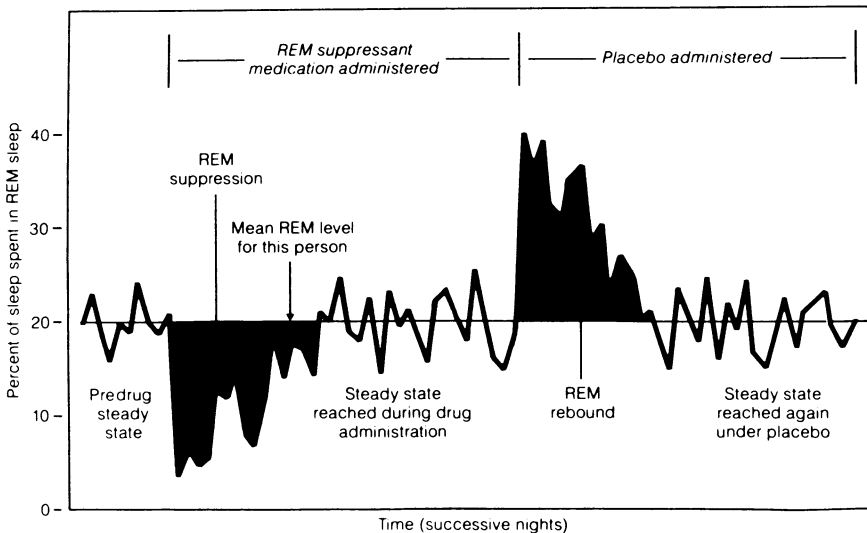
Figure 26. REM deprivation and rebound. (From Hauri, 1977. Copyright 1977 by the Upjohn Co. Reproduced with permission from the publisher and author.)

itially an increase in slow-wave sleep. In fact, on the first recovery night, there may be a decrease in REM sleep. Recovery sleep is usually not equal in length to the lost sleep and is usually not taken in one session.

Selective REM Deprivation. When volunteers are deprived of REM sleep by being awakened each time REM starts, *"REM pressure"* builds up; that is, they have a tendency to go into REM sleep before the customary 90-min lag period and spend more time in the REM state ("REM rebound") (see Figure 26). REM deprivation tends to increase agitation and impulsivity. There is some evidence, however, that REM deprivation has an antidepressant effect in severely depressed patients (Vogel *et al.*, 1976).

Selective Slow-Wave-Sleep Deprivation. Delta-sleep deprivation has a different effect from REM deprivation. Volunteers deprived of delta sleep tend to feel physically uncomfortable, with vague physical complaints and changes in bodily feelings. Their muscles become more sensitive to pressure, and they tend to become withdrawn (Hauri, 1977). A fibrositislike syndrome with musculoskeletal tenderness and heightened sensitivity to pain has been reported (Moldofsky *et al.*, 1975; Moldofsky and Scarisbrick, 1976).

Environmental Factors That Affect Sleep

Noise and temperature, among other things, are known to affect sleep. The amount of noise necessary to awaken a sleeper depends on the sleep stage: Stage 1 is the lightest, stages 2 and REM deeper, and delta sleep the deepest. *During REM sleep, thermoregulation is impaired.* Thus, sleeping in extreme temperatures may have disastrous effects, especially toward the morning when REM periods are longer (Hauri, 1977).

Caloric intake also has an impact on sleep. In experimental animals, weight gain, in general, is associated with longer, uninterrupted sleep, while weight loss is associated with short and fragmented sleep (Jacobs and McGinty, 1971).

Foods containing the serotonin precursor L-tryptophan, such as warm milk, meats, and Ovaltine, increase sleep.

Sleep Disorders

Classification

A classification scheme of sleep disorders recently developed by The Association of Sleep Disorders Centers (1979) proposes that the

sleep disorder syndromes be classified into four major classes: (1) disorders of initiating and maintaining sleep (DIMS); (2) disorders of excessive somnolence (DOES); (3) disorders of the sleep–wake schedule; and (4) dysfunctions associated with sleep, sleep stages, or partial arousals (parasomnias). This classification system is presented in the DSM III (1980) as an appendix.

Insomnia is, of course, an example of DIMS, and hypersomnia, of DOES. Somnambulism, enuresis, and night terrors are examples of parasomnias. The disorders of sleep–wake schedule include the effects of rapid time zone change or of work shift, and more persistent disorders such as delayed or advanced sleep phase syndrome (which may be seen in some severe depressive patients), among others.

A detailed discussion of all these syndromes is beyond the scope of this text. We will, instead, briefly discuss the sleep disorder syndromes that are important in medical practice. In the following discussion, conventional terminology is used for easier recognition.

Insomnia

Since the need for sleep varies among individuals, changes in sleep habits are more important than long-term patterns. Insomnia refers to an inability to sleep that interferes with the person's daytime function and sense of well-being, regardless of the amount of hours slept. Of all sleep disorders, insomnia is the symptom most commonly complained of. Insomnia may be subdivided into *difficulty in falling asleep* (DFA), *middle-of-the-night awakening* (MNA), and *early-morning awakening* (EMA) with inability to fall asleep again.

Insomnia is often a *symptom of a medical disease*, including any condition that causes pain and physical discomfort (Kales and Kales, 1974). Thus, the complaint of insomnia should alert the physician to the possibility of an associated medical condition before it is simply attributed to psychological stress.

On the other hand, *psychological distress* is the *most common cause* of insomnia. The distress may be related to interpersonal or intrapsychic conflicts or to anxiety associated with a medical disease.

Anxiety and depression are the most common psychiatric syndromes that cause insomnia. Anxiety is more often associated with difficulty in falling asleep, while depression is often associated with early-morning awakening. Insomnia associated with other signs of depression, such as loss of appetite, loss of interest, and depressive feelings and guilt, suggests the diagnosis of a depressive syndrome (see Chapter

6). The most common EEG pattern of depressive patients during the night is the waking pattern (insomnia).

A common cause of insomnia is withdrawal from a pharmacological central nervous system depressant, including the discontinuation of a sleeping medication that the patient had been taking chronically. Since many hypnotic agents are also REM suppressants, this may be associated with an REM rebound, including vivid dreams and nightmares (see vignette 2).

Evaluation of Insomnia. Evaluation of insomnia should include a *thorough history and physical examination* to understand the environmental and personal strains as well as to diagnose possible medical conditions causing the insomnia. Attention should be paid to the possibility that the patient may have associated *depression or anxiety.* If they are present, psychotherapy may be necessary in conjunction with drug treatment. Drug treatment may be specific for the depression or for the insomnia (see Chapter 20).

The possibility of *withdrawal from a central nervous system depressant drug (including alcohol)* should also be considered and a careful history taken. Barbiturates should not be withdrawn abruptly, but rather very gradually.

Hypnotic Agents. Most hypnotic agents are habit-forming and also lose potency with continued use. Thus, the use of hypnotics should be well thought out and not prolonged. Flurazepam (Dalmane), 15–30 mg, is a relatively safe and effective hypnotic over relatively long periods, but it depresses delta sleep. Barbiturates such as secobarbital, 100 mg, are effective, but they suppress REM sleep. Chloral hydrate does not affect REM sleep, but the therapeutic index is very low (effective dose is 500–1000 mg and toxicity occurs at as low as 2000 mg). Recently, the serotonin precursor L-tryptophan has been described as a natural hypnotic with minimum toxicity (in 1- to 2-g doses). L-Tryptophan occurs naturally in meats and milk.

Hypersomnia

Hypersomnia refers to excessive sleep. In contrast to narcolepsy (see below), sleep is not irresistible in hypersomnia, and there are no associated symptoms. Hypersomnic sleep may be from many hours to several days. There is often confusion and extreme difficulty in completely awakening from nocturnal sleep (Rechtschaffen and Dement,

1969). Hypersomnic patients, by and large, have *normal EEG sleep patterns* during sleep, although the heart rate and respiratory rate are often elevated before and during sleep.

Chronic hypersomnia may occur as a *symptom of central nervous system lesions,* such as after head injury, brain tumor, and cerebrovascular accidents (Kales and Kales, 1974). It may also be a symptom of *psychiatric disorders,* such as chronic anxiety and depression, in which sleep represents an escape. Especially in bipolar, manic–depressive disorders, hypersomnia may occur as the patient becomes depressed.

In Kleine–Levin syndrome, hypersomnia occurs in conjunction with bulimia (overeating). In Pickwickian syndrome, hypersomnia occurs with obesity and respiratory disorder.

Narcolepsy

Narcolepsy is an episodic disorder characterized by irresistible *sleep attacks* of a few minutes' duration (usually less than 15 min). The sleep attacks often occur after meals and toward the evening. The patient usually feels refreshed after a sleep attack, and there is usually a refractory period of a few hours before the next attack. In the complete narcoleptic syndrome, in addition to sleep attacks, there are three *associated symptoms*. They are *cataplexy* (sudden loss of muscle tone), *sleep paralysis* (momentary inability to move while falling asleep or waking up), and *hypnagogic hallucinations* (vivid visual hallucinations while falling asleep).

Cataplexy is the most common associated symptom and is often related to an emotional stimulus, such as *laughter*. Cataplexy may cause serious injuries. Sleep paralysis and hypnagogic hallucinations often occur together. The relative frequency of the narcoleptic tetrad among narcoleptics is as follows (Kales and Kales, 1974):

Sleep attacks	100%
Sleep attacks + cataplexy	70%
Hypnagogic hallucinations as one of the symptoms	25%
Sleep paralysis as one of the symptoms	50%
Sleep paralysis alone	5% or less
Full tetrad	10%

Narcolepsy usually appears during *adolescence* or *young adulthood*. There is evidence that it has a genetic basis, perhaps a single dominant gene. Whether there is a psychological disturbance in narcolepsy is controversial. EEG studies in narcolepsy show that in the majority of pa-

tients who had a history of cataplexy, both nocturnal sleep and sleep attacks invariably *begin* with REM sleep. Normals usually have 60–100 min of NREM sleep before going into REM. Only 10% of narcoleptic patients without a history of cataplexy have REM at the onset of their sleep attacks. Rechtschaffen and Dement (1969) proposed that in narcolepsy with associated symptoms, the *sleep episodes are attacks of REM sleep* and that *cataplexy is an attack of motor inhibition* similar to the one seen in REM sleep.

Diagnosis. Establishing the diagnosis of narcolepsy is essential, because narcoleptic patients are often misunderstood to be lazy, irresponsible, or emotionally unstable. Diagnosis is primarily on the basis of characteristic history. Sleep-onset REM on EEG is also helpful. Differential diagnosis includes hypersomnia, Kleine–Levin syndrome, Pickwickian syndrome, sleep-apnea hypersomnia, hypothyroidism, hypoglycemia, coma, and epilepsy (Kales and Kales, 1974).

Management. Central nervous system stimulants such as dextroamphetamine and methylphenidate are effective for sleep attacks. They are also REM suppressants. Imipramine (Tofranil), an antidepressant, is effective for the associated symptoms, but not for sleep attacks. Combined therapy with imipramine and dextroamphetamine may be dangerous, since amphetamines release catecholamines while imipramine blocks reuptake and inactivation of catecholamines (see Chapter 20). Thus, in combination, they may cause hypertensive episodes. Monoamine oxidase inhibitors are antidepressants as well as potent REM suppressors and may be effective in narcolepsy.

Narcoleptic patients whose attacks are not satisfactorily controlled with medications *should not be allowed to perform potentially dangerous activities* such as driving an automobile or operating certain machinery.

Somnambulism (Sleepwalking)

Somnambulism occurs more commonly among *children* and preponderantly among *males*. There is usually a positive *family history* and a higher incidence of *enuresis* among sleepwalkers than in the general population (Kales and Kales, 1974). The sleepwalking episode usually lasts for several minutes, and there is complete amnesia for the incident. EEG studies show that somnambulism occurs exclusively during *NREM sleep*, especially during delta sleep. Thus, somnambulism is *not* associated with dreaming. Most children who sleepwalk tend to cease sleep-

walking in a few years. Psychological disturbances are not associated with somnambulism in children, although they are often present in adult sleepwalkers (Sours *et al.*, 1966).

Management. The most important consideration in somnambulism is to *protect the patient from injury*. Contrary to popular belief, a sleepwalker can indeed suffer serious injuries. Locking doors and windows, removing potentially dangerous objects, and having the patient sleep on the first floor are among the prophylactic measures to be used. In children, conveying the information that they usually "outgrow" the condition is reassuring and important. In *adults, psychiatric evaluation* and treatment are often indicated. In severe cases, delta-sleep-suppressant medications, such as *benzodiazepines* (e.g., diazepam), may be tried, although their efficacy has not yet been clearly established (Kales and Kales, 1974).

Enuresis (Bed-Wetting)

Bed-wetting is a relatively *common* condition among children. Approximately 10–15% of all children between four and five years of age continue to wet the bed. Even in adulthood, enuresis occurs in about 1–3% of the population.

Primary enuresis refers to bed-wetting continuing from infancy with no period of consistent dryness for at least a few months (Kales and Kales, 1974). There seems to be a *genetic component* to this condition, and *organic disease* is often the cause of primary enuresis. For example, urethral obstruction in males and ectopic ureter in females can cause constant dribbling. Diverticulum of the anterior urethra, epispadias, ectopia vesicae, and cystitis can also cause bed-wetting.

Secondary enuresis is often associated with psychological factors such as regression due to jealousy of a newborn sibling.

EEG studies in enuresis show that bed-wetting can occur in any sleep stage. Most of the enuretic episodes occur during the first third of the night. If the subjects are awakened and changed, they do not report any dreams and, on falling asleep, have normal sleep patterns. If the subjects continue to sleep-wet during the ensuing REM periods, however, they tend to incorporate the wetness into their dreams.

Management. First, the physician should determine whether the enuresis is primary or secondary. There may be a "maturational lag" in primary enuresis, especially if there is a family history of enuresis. The

child may simply "outgrow" the enuresis in these instances. In secondary enuresis, psychological treatment may be necessary. Parents of enuretic children should be reassured that most affected children eventually outgrow this disorder.

Imipramine (Tofranil) is effective in reducing enuretic frequency if judiciously used. Conditioning devices, such as a bell activated by bedwetting, may sometimes be helpful.

Night Terrors

The night terror is characterized by a subjective feeling of *intense anxiety* associated with oppressive sensations, severe autonomic discharge, "blood-curdling screams," and practically no recall of specifics. In children, this is also called *pavor nocturnus*, and in adults, *incubus*. The night terrors occur during NREM *delta-wave sleep* (especially stage 4 sleep). As in somnambulism, *children usually outgrow this disorder*. In adults, psychiatric evaluation may be necessary. Delta-wave-sleep suppressants, such as benzodiazepines (e.g., diazepam), are useful in suppressing night terrors. Reassurance of parents that children usually outgrow this condition is helpful.

Nightmares

In contrast to night terrors, nightmares are dreams that occur *during REM sleep*. Frightening dreams often occur during the *REM rebound*, concomitant to withdrawal of REM-suppressant medications, including barbiturates and alcohol.

Sleep Apnea

Sleep apnea may be a universal occurrence in normal infants between one and three months of age (Kales and Kales, 1974). It also occurs in Pickwickian syndrome, narcolepsy, and insomnia. Sleep apnea may be subdivided into (1) a central type, (2) a peripheral obstructive type due to upper respiratory tract obstruction, and (3) a mixed type of central apnea followed by upper-airway obstruction.

Sleep apnea in infants occurs mostly *during REM* and is often associated with the *sudden-infant-death syndrome*. Sleep apnea associated with hypersomnia, insomnia, and narcolepsy occurs during both REM

and NREM states. Snoring is a common finding associated with sleep apnea due to airway obstruction.

Medical Conditions Affected by REM Sleep

REM sleep often precipitates symptoms in patients with medical illness. For example, nocturnal angina is associated with REM. In patients with duodenal ulcers (but not in normal subjects), there is an increase in gastric secretion rate during REM.

Hyperthyroidism and Hypothyroidism

Delta sleep is markedly *reduced* in *hypothyroidism* and *increased* in *hyperthyroidism*. With treatment of the thyroid disorder, delta sleep gradually returns to normal. Sleepiness is a frequent symptom of hypothyroidism.

Summary

Sleep consists of NREM and REM sleep. NREM sleep is made up of stages 1 through 4. Stages 3 and 4 are often referred to together as delta or slow-wave sleep. During a typical night, NREM sleep occurs first, followed by REM, the duration of which is about 5–20 min. The cycle repeats itself every 90–100 min. Total REM time per night is about 20% of sleep time. REM is associated with *physiological arousal, skeletal muscular relaxation*, and *visual dreams*.

The sleep–wake and REM–NREM cycles are examples of biological rhythm. Biological rhythms may be circadian, diurnal, nocturnal, infradian, or ultradian. Many metabolic and endocrine systems have rhythmicity.

Brain mechanisms of sleep are not completely understood. Arousal and wakefulness seem to depend on the ascending reticular activating system. Sleep, especially NREM sleep, is probably mediated by the serotonergic neurons arising from the pontine median raphe system. In REM sleep, noradrenergic and cholinergic neurons in the pons (from locus ceruleus and pontine giant cells, respectively) seem to be involved in addition to serotonergic neurons. The electrical activity generated in the pons by the interaction of these systems subsequently result in the

rapid eye movements (REM) and the cortical desynchronization, which may give rise to the subjective experience of dreaming.

The *content* of remembered *dreams* may be especially important in the context of ongoing emotional life, especially in psychotherapy. Freud formulated a way of conceptualizing dreams. According to his theory, dreams represent wish fulfillments through hallucinations during sleep. The wishes thus emerging are often unconscious or repressed during waking life because of their unacceptable nature. *Dream work* is a term used by Freud to refer to the process of constructing dreams out of the unconscious wishes. It includes condensation, displacement, and reversal.

The *function* of slow-wave sleep seems to be physiological restoration, including protein synthesis in the central nervous system. REM sleep may be related to processes of emotional adaptation to the environment as well as consolidation of memory and, perhaps, restoration of catecholamines in the brain.

The *need* for sleep appears to depend on the individual, with wide variations. In sleep deprivation, NREM sleep is restored first, followed by REM. After selective REM deprivation, there is a REM rebound.

Environmental factors such as noise, caloric intake, and content of food affect sleep.

Sleep disorders include insomnia, hypersomnia, narcolepsy, and somnambulism. Somnambulism, night terrors, and bruxism (teeth-grinding) characteristically occur during delta sleep (NREM). The autonomic arousal during REM may precipitate medical symptoms. Delta sleep is reduced in hypothyroidism and increased in hyperthyroidism. Many medical conditions, especially painful conditions, interfere with sleep. Sleep disturbance is often an important and early sign of depression and anxiety.

Implications

For the Patient

Sleep disturbance is often dismissed as being of little consequence. On the other hand, some patients attribute serious symptoms and signs to "lack of sleep" or "sleeping too much," thereby delaying help-seeking behavior. Abuse of sleeping medications is another problem that patients with sleep difficulties often develop. Certain patients become obsessed or frightened by especially vivid dreams that may be associated with an REM rebound phenomenon. A good night's sleep is very important for most patients for a sense of well-being and physiological

restoration. In a stressful environment like the hospital, sleep is especially important for the patient.

For the Physician

Sleep disturbance often indicates the presence of a medical or psychiatric pathology. In serious sleep disturbances, a comprehensive evaluation is necessary. Many *medications* affect sleep; some increase drowsiness and sleep through a depressant action on the central nervous system, others reduce sleep through stimulation of the central nervous system, and still others selectively affect REM (mostly by suppressing REM). It is important to consider the effect of the proposed medication on the patient's sleep and REM. The physician should be aware of the REM rebound phenomenon that can result in nightmares. Hypnotics should be judiciously, and only transiently, prescribed.

For the Community and the Health-Care System

Hospitals should provide an environment conducive to good sleep for patients. This includes reducing noise and light levels and furnishing comfortable beds. Since painful conditions interfere with sleep, which may, in turn, produce physical discomfort, adequate *pain relief* should be provided for patients suffering from pain. Physicians should be educated concerning the need for adequate sleep and pain relief in medical patients. Many hypnotic agents are either ineffective, unsafe, or both. The public should be educated concerning the use of hypnotic agents, especially the dangers of abuse.

Recommended Reading

Biological Rhythms in Psychiatry and Medicine, Public Health Service publication 2088. Washington, DC, National Institute of Mental Health, 1970. This is a very comprehensive and easy-to-read summary of biological rhythms, including circadian rhythms, sleep and dreaming, and even sunspots and emotional rhythms. A little dated, but still good background reading.

Freud S: *The Interpretation of Dreams*. This book, published first in German in 1900, has several translations. We would recommend the James Strachey translation, available in paperback (New York, Avon Books, 1965) or in the *Standard Edition of the Complete Psychological Works of Sigmund Freud* (London, Hogarth Press, 1973, Vols 4 and 5). This work is a landmark in understanding the meaning of remembered dreams. The psychoanalytic perspective concerning dreams and their interpretation is very lucidly and

readably presented. Dreams are seen as "wish fulfillment" through hallucination motivated by the need not to interrupt sleep. Many ideas discussed by Freud in this early work became the foundation of later psychoanalytic theory. Highly recommended.

Weitzman ED (ed): *Advances in Sleep Research,* Vols 1 and 2. New York, Spectrum Publications, 1974 and 1976. These two volumes provide an extensive reference concerning the most current research findings about sleep. The chapters include, just to name a few, chemical anatomy of brain circuits in sleep and wakefulness, the phylogeny of sleep, the cellular basis of sleep cycle control, ultradian rhythms in sleep and wakefulness, maturation of sleep patterns of the newborn infant, sleep disturbances in catatonic patients, and biogenic amines. It is a multiauthored work. Volume 1 has topics of somewhat more general interest than volume 2.

References

Aserinsky E, Kleitman N: Regularly occurring periods of eye mobility and concomitant phenomena during sleep. *Science* **118**:223–224, 1953.

Curtis GC: Psychosomatics and chronobiology: Possible implications of neuroendocrine rhythms: A review. *Psychosom Med* **34**:235–256, 1972.

Druckner-Colin R, de Gomez-Puyou MT, Gutierrez MC, *et al.*: Immunological approach to the study of neurohumoral sleep factors: Effects on REM sleep of antibodies to brain stem proteins. *Exp Neurol* **69**:563–575, 1980.

Foulkes D: Dream Reports from different stages of sleep. *J Abnorm Psychol* **65**:14–25, 1962.

Foulkes D, Vogel G: Mental activity at sleep onset. *J Abnorm Psychol* **70**:231–243, 1965.

Freud S: *The Interpretation of Dreams* (Strachey J, ed). New York, Avon Books, 1965. (Originally published in 1900.)

Friedman S, Fischer C: On the presence of a rhythmic, diurnal, oral instinctual drive cycle in man: A preliminary report. *J Am Psychoanal Assoc* **15**:317–342, 1967.

Guyton AC: *Textbook of Medical Physiology.* Philadelphia, WB Saunders, 1976, p 734.

Hanada Y, Kawamura H: Sleep–waking electrocorticographic rhythms in chronic cerveau isole rats. *Physiol Behav* **26**:725–728, 1981.

Hartmann E (ed): *Sleep and Dreaming.* Boston, Little Brown & Co, 1970, p 408.

Hartmann E: *The Functions of Sleep.* New Haven, Yale University Press, 1973.

Hauri P: *The Sleep Disorders.* Kalamazoo, Michigan, Upjohn Co, 1977.

Hauri P: *Current Concepts: The Sleep Disorders,* ed 2. Kalamazoo, Michigan, Upjohn Co, 1982, p 14.

Hobson JA: The sleep–dream cycle: A neurobiological rhythm, in Ioachim E (ed): *Pathobiology Annual.* New York, Appleton-Century-Crofts, 1975, pp 369–403.

Hobson JA: *The Sleep–Dream Cycle and the Single Neuron.* Nutley, New Jersey, Roche Laboratories, 1978.

Hobson JA, McCarley RW: The brain as a dream state generator: An activation–synthesis hypothesis of the dream process. *Am J Psychiatry* **134**:1335–1348, 1977.

Jacobs BL, McGinty DJ: Effects of food deprivation on sleep and wakefulness in the rat. *Exp Neurol* **30**:212–222, 1971.

Johnson LC: Psychological and physiological changes following total sleep deprivation, in Kales AA (ed): *Sleep Physiology and Pathology.* Philadelphia, Lippincott, 1969, pp 206–220.

Jouvet M: Some monoaminergic mechanisms controlling sleep and waking, in Karczmar AG, Eccles JC (eds): *Brain and Human Behavior.* New York, Springer-Verlag, 1972a, pp 131–161.

Jouvet M: The role of monoamines and acetylcholine-containing neurons in the regulation of the sleep–waking cycle, in *Ergebnisse der Physiologie*, Vol 64: *Neurophysiology and Neurochemistry of Sleep and Wakefulness.* Berlin, Springer-Verlag, 1972b, pp 166–308.

Kales A: Sleep and dreams. *Ann Intern Med* **68**:1078, 1968.

Kales A, Kales JD: Sleep disorders: Recent findings in the diagnosis and treatment of disturbed sleep. *N. Engl J Med* **290**:487–499, 1974.

Kleitman N: *Sleep and Wakefulness.* Chicago, University of Chicago Press, 1965.

Moldofsky H, Scarisbrick P: Induction of neurasthenic mussculoskeletal pain syndrome by selective sleep stage deprivation. *Psychosom Med* **38**:35–44, 1976.

Moldofsky H, Scarisbrick P, England R, *et al.*, Musculoskeletal symptoms and non-REM sleep disturbance in patients with fibrositis syndrome and healthy subjects. *Psychosom Med* **37**:341–351, 1975.

Monnier M, Gaillard JM: Biochemical regulation of sleep. *Experientia* **36**:21–24, 1980.

Morgane PJ, Stern WC: Chemical anatomy of brain circuits in relation to sleep and wakefulness, in Weitzman EE (ed): *Advances in Sleep Research*, Vol 1. New York, Spectrum Publications, 1974, pp 1–131.

Rechtschaffen A, Dement W: Narcolepsy and hypersomnia, in Kales A (ed): *Sleep: Physiology and Pathology.* Philadelphia, Lippincott, 1969, pp 119–130.

Rechtschaffen A, Kales A (eds): *A Manual of Standardized Terminology, Techniques and Scoring System for Sleep Stages of Human Subjects*, National Institutes of Health publication 204. Washington, DC, US Government Printing Office, 1968.

Roffwarg H, *et al.*: Ontogenetic development of the human sleep-dream cycle. *Science* **152**:604–619, 1966.

Sours JA, Frumken P, Indermell RR: Somnambulism. *Arch Gen Psychiatry* **14**:595–604, 1966.

Van de Castle RL: Temporal patterns of dreams, in Hartmann E (ed): *Sleep and Dreaming* Boston, Little Brown & Co, 1970, pp 171–181.

Vogel G, McAbee R, Barker K, *et al.*: REM pressure and improvement of endogenous depression, in Chase MH, Mitler MM, Walter PL (eds): *Sleep Research*, Vol 15. Los Angeles, University of California at Los Angeles, Brain Information Service/Brain Research Institute, 1976.

ON ASSESSING A PATIENT
A Clinical Systems Approach

Approach to a Patient: The Patient Evaluation Grid

1. A 56-year-old man had a routine chest X-ray as a part of his company's physical fitness program. The X-ray revealed a coin lesion in the right lung, indicative of a tumor. When he was told that more medical workup was needed, he felt very perplexed and somewhat angry because he "felt so well." His wife, who was told of the possible implications of the X-ray shadow (cancer), became very anxious, which in turn made the patient more upset. Since he had always been a strong, healthy man, he attempted to dismiss the fact that there was a need for further medical tests and, in fact, did not keep his next appointment with the doctor. Since the X-ray had been done at the company's request, he was eventually told by his superiors that he had to report to the doctor, who in turn had to report to the manager. When the diagnosis of cancer was made, he was retired from work. A depressive syndrome ensued, his physical state deteriorated, and the cancer became symptomatic.

2. A 7½-month-pregnant 24-year-old black woman was admitted to the obstetrical ward because of toxemia of pregnancy. There had also been excessive weight gain. In the hospital, she was found eating white starch powder from a box. The intern learned that this was a common custom during pregnancy in some Southern subcultures.

3. A 15-year-old girl was admitted to the surgical service for correction of tetralogy of Fallot (a form of congenital heart disease). In the hospital, she displayed excessive anxiety that was not controlled by diazepam (Valium). When the intern asked her detailed questions about her family, she found that the patient's parents had agreed to separate when their daughter's heart

273

condition had been repaired. The patient was anxious about the impending separation of her parents!

4. A 45-year-old man had an operation for coronary artery disease. During the second postoperative day, he began to have frightening visual hallucinations. He was also tremulous and febrile. Since the symptoms were very similar to delirium tremens, the astute medical student on the case asked the family about the patient's drinking habits. It turned out that he customarily drank at least half a dozen cocktails a day when he was working. In fact, even during his hospitalization, his wife had supplied him with several bottles of vodka that were hidden in his room. He lost access to alcohol only after the operation!

Once a person comes to the physician for medical care, it is the doctor's responsibility to carry out a comprehensive evaluation, to diagnose disease if present, and to treat the patient to the best of his or her ability. As noted in earlier chapters, the physician and health-care personnel will, to a large degree, determine through their demeanor and behavior how much cooperation the patient will give in the assessment and treatment process and, further, what the patient's attitudes and expectations will be in future contacts with the health-care system.

The chapters in this section focus on the critical task of clinical assessment. Specifically, we discuss a system for organizing data and for thinking about patients and their problems. The aim of this system of approach is to facilitate the organization of relevant biological, psychological, and environmental information about the patient in a comprehensive and integrated fashion. This system of organized information can then be the basis for diagnosis and for treatment planning.

Before information about a patient can be organized and evaluated, it must first be gathered, and this is not a simple task. To obtain information effectively, the physician must have (1) extensive knowledge of clinical medicine, (2) high degrees of skill in conducting medical interviews and in managing interpersonal relationships; and (3) extensive knowledge of human psychology, particularly in respect to pathogenesis and to the experience of medical illness.

Medical history-taking—a specialized instance of interviewing—is an art and a skill that the physician continuously practices and aims to develop and improve throughout his entire professional career. Guided by knowledge of signs, symptoms, and natural history of disease, the physician uses the interview to formulate a series of (diagnostic) hypotheses, which he or she continually tests against the new information that is elicited from the patient as the interview proceeds. New information then leads to changing hypotheses and seeking further data in a series of algorithmic steps.

Eventually a final diagnostic hypothesis—the "differential diagnosis"—is subjected to a series of empirical tests, i.e., the physical examination, selected laboratory tests, and special diagnostic procedures. The heuristic value of the biological data obtained in the later diagnostic steps is dependent on the medical knowledge that guided the selection of procedures and on the physician's skill in eliciting personal and historical as well as purely medical information.

As mentioned above, medical interviewing is a special form of interviewing; its basic guidelines derive from the same set of general principles that govern all interview procedures. But one special feature distinguishes the medical history: It takes place in the context of medical help-seeking behavior (usually accompanied by pain, suffering, or worry). The effectiveness of the medical interview depends on the nature of the developing doctor–patient relationship. At the same time, the patient's experience of the interview contributes to shaping that relationship. Thus, the effectiveness of the medical history-taking also depends on personal qualities: the physician's capacity to be empathic, to respond to the patient with sensitivity and with understanding of his needs for compassion and respect. These qualities not only shape the nature of the doctor–patient relationship initially but also later exert a continuing influence on the patient's clinical condition and response to treatment.

The potential for developing the art, skill, and human qualities mentioned above is present in most, if not all, students when they choose medicine as a career. There are, however, many factors in medical education that understandably but regrettably tend to blunt these capabilities. But the fact is that nurturance and full development of these qualities should constitute a major goal of medical teachers and students alike, and for that matter of all physicians throughout their entire careers. But this goal depends on more than good intentions and common sense. There are well-established principles to be learned and skills to be practiced. Learning about medical interviewing constitutes a separate subject—too extensive and too important to be relegated to an abbreviated and inadequate textual development that space limitations of this introductory text would dictate. More extensive specialized texts and monographs should be studied as guides for developing this highly important medical skill.*

---

* Two good books on interviewing are *Patient Interviewing, The Human Dimension*, by Reiser and Schroder, and *The Clinical Approach to the Patient*, by Morgan and Engel (see Recommended Reading).

The Patient, the Patient's Components, and the Patient's Environment

To evaluate a diseased organ thoroughly, it is necessary to consider it in three broad perspectives that reflect the three levels of organization that are cogent to its function: (1) the function and state of the organ as a whole; (2) the function and state of components or subsystems of the organ, such as component cells and tissues, blood supply, and chemical subsystems; and (3) the function and state of the larger system of which the organ is a part (e.g., the cardiovascular or digestive) and even its relationship with other organs (e.g., heart and kidney, liver and brain). For example, a thrombus in a coronary artery (part of the cardiovascular system) results in anoxia of myocardial tissues (chemical), leading to infarction (tissue), and thus reduces cardiac contractility (organ), resulting in congestive heart failure (system).

Similarly, in repairing an automobile, the mechanic must know the state of the engine, radiator, and other *components* of the automobile; how the car is functioning as a *whole* (such as its maximum speed, handling characteristics, and appearance); and the *interaction* of the automobile with external factors (such as how the owner is maintaining it and whether it is habitually driven on particularly rough roads).

In approaching patients, a similar conceptual approach is useful—that is, a thorough evaluation of the patient that includes organizing data according to three dimensions: (1) the *biological dimension*, that is, the structural and functional state of physiochemical components and subsystems, including of course, healthy and possibly diseased organs; (2) the *personal dimension*, that is, the psychological state and behavior of the whole person (patient); and (3) the *environment dimension*, that is, the environmental–interpersonal data, including interaction of the patient with the physical environment and with family, work, and the health-care system, among others (Figure 27). Each of these levels or dimensions can be conceptualized as a *system*, with subsystems at each level and with interactions both within and across dimensions (see Chapter 22).

Disease, Illness, and Distress

A patient, by definition, is a person seeking help for experienced distress. The cause of the distress and the factors that contribute to it reside in and relate to the three dimensions discussed above, that is, the *biological*, the *personal*, and the *environmental–interpersonal*.
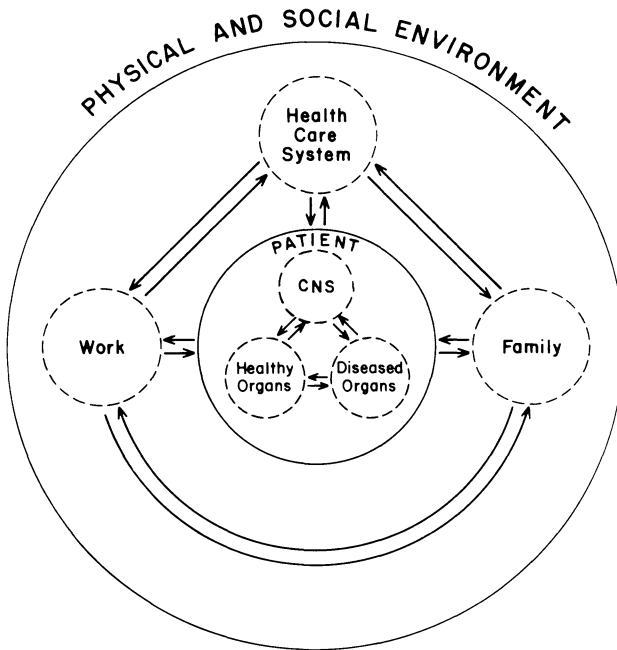
Figure 27. The patient, the patient's components, and the patient's environment.

The person experiences distress, such as anxiety, suffering, and depression (see Part II), which together with the symptoms emanating from the biological components constitutes the *illness* (Feinstein, 1974), and these then interact with each other and with components of the environment.

Disease at the organ level often produces the experience of illness, but a disease process may exist without illness for extended periods (e.g., asymptomatic progression of a cancer, as in vignette 1), and a patient may experience an illness without evidence of disease [e.g., "psychogenic" pain (see Chapter 9)]. Factors at the personal level may contribute to the pathogenesis of disease, such as a habit of smoking or excessive drinking (vignette 4).

At the environmental level, interpersonal difficulties may result in the experience of anxiety and in heterothetic [problems of living presenting as a symptom (see Chapter 1)] help-seeking behaviors. Furthermore, help-seeking behavior is often prompted by family or friends of the patient. Environmental factors may also contribute to the pathogenesis of disease (Rahe, 1972); examples of such factors are environ-

mental poisons, occupational toxins (e.g., heavy metals), and interpersonal loss (e.g., bereavement). Assessment of the environmental system is also important in the management of the patient, since treatment plans should take the patient's family and work situation into account.

Patient Evaluation Grid (PEG)

If informatioin pertaining to the three basic dimensions is to be of practical help in the management of patients, it must be organized in such a way as to be applicable in the clinical arena. We propose such a method that organizes information in the three dimensions according to three longitudinal *time contexts* taking the patient's help-seeking behavior as the fixed point of time reference. These three time contexts are (1) *current*, (2) *recent*, and (3) *background*. They expand in scope and complexity with retrograde movement along a time continuum from the present to the remote past.

The *current context* refers to the cross-sectional *state* in each dimension at the time of help-seeking behavior. The *recent context* refers to *recent state* changes and events in each system. This includes the appearance of symptoms and signs, as well as such stressful events as bereavement, retirement, recent hospitalization, and recent disease. The *background context* refers to *traits* rather than states, i.e., congenital and early childhood diseases, long-standing personality traits, and early environmental, interpersonal, and cultural influences. Early experience, heredity, constitution, personality style, family background, cultural heritage, and other determinants are included in the background context.

A three-by-three grid, the Patient Evaluation Grid (PEG),* displays the intersection of data regarding the three dimensions of organization (biological, personal, and environmental) with the three longitudinal time contexts (current, recent, and background), creating a nine-compartment grid that we have found useful in teaching and research and in everyday clinical practice (see Figures 28 and 29).

In diagnosis, the PEG can serve as a useful guide for (1) taking a complete medical history, (2) performing the physical examination, and (3) planning further special diagnostic procedures (e.g., laboratory tests). It is useful to have the PEG in mind while interviewing the patient and, eventually, to enter information into the squares with dates. Relevant information covering some matters can, when appropriate, be

---

* The PEG was developed by H Leigh, MF Reiser, and AR Feinstein in 1978.

DIMENSIONS	CONTEXTS		
	CURRENT (Current States)	RECENT (Recent Events and Changes)	BACKGROUND (Culture, Traits, Constitution)
BIOLOGICAL			
PERSONAL			
ENVIRONMENTAL			

Figure 28. Framework of the PEG.

DIMENSIONS	CONTEXTS		
	CURRENT (Current States)	RECENT (Recent Events and Changes)	BACKGROUND (Culture, Traits, Constitution)
BIOLOGICAL	physical symptoms, signs physical exam medications abnormal lab values	onset/change in symptoms and signs change in physical condition change in medications recreational drugs	past history of disease family history of medical disease family history of psychiatric illness
PERSONAL	chief complaint chief psychiatric complaint mental status expected treatment	change in psychiatric condition change in mood/behavior change in habits recent help-seeking	personality traits defense/coping mechanism past psychiatric history
ENVIRONMENTAL	who lives with patient occupation stress physical environment	change in living condition change in occupation LCU (Holmes & Rahe) change in physical environment	parents occupation and socioeconomic class schools attended early relationships marriage/jobs

Figure 29. PEG: Organization of relevant information. Biological dimension refers to the components of the patient, such as the organ systems, tissue, and chemical composition. Personal dimension refers to attributes of the whole person, including the psychological and behavioral aspects. This includes personal habits such as smoking and drinking. Environmental dimension refers to the psychosocial and physical environments surrounding the patient.

obtained from family members as well as the patient, e.g., matters pertaining to cultural and family expectations of the sick role, the meaning of illness, personality style, and so forth (Leigh *et al.*, 1980). In this chapter, we will discuss each of the elements of the PEG briefly to provide the reader with an overview. More detailed discussion of each of the contexts follows in subsequent chapters, which will also illustrate its application in patient management.

What Goes into the PEG

Current Context. This column, which concerns the current state of the patient, has most immediacy for both the patient and the doctor, since the information indicates the pressing needs of the patient as well as any constraints that may have to be taken into account in formulating therapeutic plans.

Biological Dimension: This square contains information about the patient's physical state, vital signs, and the classic data of the present illness, including physical symptoms and laboratory findings, which constitute the basis of clinical diagnosis and many therapeutic decisions.

Personal Dimension: Data concerning the chief complaint, appearance, current psychological state, mental status, and the anxiety level are contained here. The chief complaint relating to physical state belongs in both the biological and the personal dimension to the extent that the patient is suffering from it. This square also contains answers to questions such as the following: Is the patient coherent, confused, frightened, or depressed? What does the patient think about the symptoms? What does the patient expect (e.g., hospitalization, a "shot," an operation)? The information is valuable for an understanding of how the patient experiences illness and of his motivation in seeking help. Recognition of the patient's immediate need for help may consolidate the doctor–patient relationship and increase patient cooperation.

Environmental Dimension: Examples of data that belong in this square are answers to the following questions: Who accompanied the patient? Who lives with the patient? Marital status? Who suggested seeing a doctor? What effects will the patient's illness have on his family and occupation? The value of this square is its information about the patient's "significant others." At a very practical level, the next of kin, family, or friend is an important source of information, support, and, occasionally, consent.

Recent Context. This column contains highlights of *recent changes and events*, including symptoms, illness, and disease processes and their effects on the patient's life and family, as well as changes in long-standing patterns such as habits. This column also includes information about possible contributing, complicating, or precipitating factors of the illness and help-seeking behavior. "Recent" is used here as a relative term, its range being measured in days, weeks, or months—often as much as 6–12 months, seldom longer. All entries here should include the *date* or approximate *duration* of the event.

Biological Dimension: Recent physical change, weight gain or loss, injuries, discovery of hypertension, recent surgery, and medications are examples of data to be included here. Data found in this square provide information concerning early signs of current disease and possible contributing or antecedent factors, as well as physical effects of recent environmental and behavioral changes.

Personal Dimension: This square contains such information as data concerning personality change, change in habits (e.g., increased drinking), mood change (e.g., depression), and preoccupation with disease (e.g., conviction that he has cancer). Mood and habit changes may contribute to disease (e.g., depression causing self-neglect and malnutrition and susceptibility to infection) or may be symptoms of a disease process (e.g., personality change in frontal lobe tumor).

Environmental Dimension: This section, which denotes possible environmental stressors causing disease or illness behavior (Mechanic, 1962; Rahe, 1972; McWhinney, 1972), is concerned with such data as change in residence or job, marriage, separation, divorce, bereavement, recent travel, exposure to doctors or hospitals, and recent illness in the family.

Background Context. The background factors indicate previous illnesses and medical experiences and predispositions to disease. They also set the tone and attitude of the patient toward illness and help-seeking behavior and adaptation to the sick role through personality, constitution, predisposition, and cultural experiences. The items appearing here represent relatively long-standing, stable characteristics of the patient and are usually resistant to change. This information is important in setting realistic goals for care as well as in understanding the patient's needs.

Biological Dimension: This square contains information concerning congenital conditions, constitutional vulnerability, predisposition, genetic endowment, and acquired tendency for specific diseases. Family history of hereditary disease and early injuries, diseases, and deformities

also belong here, as do answers to such questions as history of functional disorders under stress and allergies.

Personal Dimension: Data such as the patient's personality type (see Chapter 17), habitual psychological defenses, and coping styles (see Chapter 5) belong here. They include answers to such questions as the following: Does the patient tend to deny the presence of symptoms and signs? Habitual complaining of minor symptoms? Tendency to be exacting and somewhat obsessive? Tendency to become overly dependent? The patient's habits (e.g., alcohol, smoking), hobbies, ambitions (e.g., always wanted to be a doctor), and educational and intelligence level are also pertinent data here. How information is processed and interpreted by the patient, what current illness means to the patient, and how the patient may respond to planned therapeutic approaches may be determined by the factors found in this square.

Environmental Dimension: The patient's cultural background, early physical environment, ethnic origin, and religion are examples of data to be entered into this square. Answers to questions such as the following also belong here: Early exposure to illness in family? Cultural sick-role expectations? Cultural myths about illness and treatment? Religious qualms about procedures? Early experiences with doctor or hospital? Information contained in this section may provide clues to the patient's current attitudes and expectations. Early contact with the physician or hospital may be related to unrealistic or irrational attitudes toward medical care.

Significance of Contexts

The organization of longitudinal data into current, recent, and background contexts, makes clear the immediacy or novelty of or degree of habituation to a particular phenomenon. For example, chest pain appearing in the recent context *de novo* has greater immediacy for the patient than chest pain that first appeared in the background-context column. Although we are dealing here with chronological material along a time dimension, the material is seen from the vantage point of the patient's help-seeking behavior. Thus, the background, recent, and current contexts may affect one another in both forward and backward directions. For example, if the patient is confused currently, he may not be able to give a complete or accurate past history.

The need for intervention or treatment and the ease with which this can be done are usually greatest in the current context and decrease progressively in the recent and background time frames. Although the

background context information, in general, most often points to limiting factors in approaching the patient, certain background phenomena are amenable to change. For example, the patient's expectations and fantasies about a medical procedure may change if specifically discussed. In vignette 3, the congenital heart disease is a background context factor that is amenable to change. Also, the expectation that the parents would separate after the girl's recovery could be discussed and coped with by counseling and possibly family therapy.

Significance of Dimensions

The *biological dimension* provides information concerning the structure and status of the component parts of the person. Information referable to this dimension forms the basis of the diagnosis of disease. Data can be systematized most thoroughly at this level thanks to the expansion of our knowledge in this dimension through research in physiology, biochemistry, pathology, and other disciplines. Obviously, there are a number of subsystems or levels within the biological level, e.g., organs, tissues, cells, and molecules. Disease may involve alteration at any of these levels of organization within this dimension. In using the PEG, the entries in this dimension should include the important laboratory and physical findings as well as diagnosed and suspected diseases. In this dimension, as in others, the current-context physical or physiological state requires immediate attention and treatment. Information concerning the etiological and predisposing biological factors is usually to be found in the recent and background contexts.

The *personal dimension* denotes qualities and characteristics ascribable to the patient's total personality, including appearance, disposition, habits, feelings, idiosyncrasies, fantasies, and prejudices. Since the central nervous system, especially the brain, is the control center for the whole organism, the attributes of the personal dimension often reflect the condition of the central nervous system, although they are not confined to it.

Background-context personality or habits may contribute to or serve as markers of vulnerability to certain diseases. For example, smoking contributes to lung cancer and coronary disease.

Recent-context events or changes, such as personality change or mood change, may be secondary to disease (e.g., carcinoma of the tail of the pancreas) or an interpersonal event (e.g., bereavement) and in turn may give rise to a disease (e.g., depression resulting in malnutrition and infection) or to a change in behavior (e.g., increasing intake of alcohol resulting in problems at work and unemployment).

Current-context information such as chief complaint (suffering), mental status, and behavior indicate the motivation for help-seeking behavior. Care must be directed toward the immediate need (relief from suffering that motivated the help-seeking behavior) if the patient is to receive satisfactory medical treatment, since he will be less motivated to comply with the physician's plans if the plans omit measures to relieve or ameliorate the specific current suffering. Relief of the immediate and specific suffering, such as overwhelming anxiety or pain, may be necessary before further evaluation can proceed (vignette 3). An important factor at the personal level is the patient's fantasies and expectations concerning the health-care system. Elucidation of these is essential for any patient who has difficulties in complying with medical procedures or regimen, since simple direct reassurance or education may correct unrealistic fantasies and expectations.

The *environmental dimension* provides information concerning the physical and interpersonal environment in which the patient lives and functions. This environment, in the background context, helped shape the patient's personality and cultural aspirations and in a real sense provided the patient with a large part of his constitutional endowment. Previous contact with the health-care system and health-care personnel provides information concerning the patient's sick-role behavior. Changes in the physical environment (e.g., getting a job in a chemical factory) may provide a clue to the cause of illness (e.g., heavy-metal poisoning). Customs unique to subcultures may provide clues to unusual behavior (e.g., weight gain and unusual eating habits, as in vignette 2).

Recent life changes and life situations are the sources of the patient's current joys, sorrows, and concerns and may have provided contributing factors in illness (Rahe, 1972; Holmes and Rahe, 1968). The impetus to seek help at a particular time usually comes from a recent change or event, for example, interpersonal tension (McWhinney, 1972) or a relative falling ill with a similar disease.

The current interpersonal context provides information concerning the significant others involved with the patient. At a very practical level, the next of kin or friend who accompanied the patient to the physician or who is most readily available on request is an important resource— a source of information concerning the patient and for obtaining consent for procedures when necessary.

Rational Patient Management

In formulating a rational patient-management plan, consideration of the interrelationships among findings in the nine compartments of

the PEG is useful. Although illness (the suffering or distress leading to help-seeking behavior) usually occurs in the presence of a disease, it does not always have a one-to-one correlation with it. For example, suffering (illness) may be present in the absence of disease (e.g., bereavement) and may result from the treatment of disease rather than from the disease (e.g., pain caused by surgical operation to remove an asymptomatic malignant tumor of the breast). Problems of living presenting as a physical symptom are another example (McWhinney, 1972). The PEG will show, in each instance, the precipitating and contributing factors in one of the squares in the recent or current context. Events such as bereavement, unemployment, and psychological depression may be found in conjunction with biological changes leading to a disease, and such associations raise questions for research concerning the physiological mechanisms involved. For example, in a patient with current streptococcal pharyngitis and recent bereavement, one may wonder about the possible role of the central nervous system state (induced by bereavement) in (1) lowering immunological resistance and (2) influencing the likelihood of subsequent development of rheumatic fever or glomerulonephritis. On the other hand, the combination of the presence of systems and signs in the absence of identifiable disease and the absence of convincing evidence that illness behavior can be attributed to the personal and environmental–interpersonal dimensions may indicate the need for a further search into the possibility of a yet unidentified or incompletely understood disease.

In most situations, the PEG will identify the strains in the biological (disease), personal (illness), and environmental–interpersonal systems. These identified problems form the basis of the three-dimensional diagnoses to be entered into the PEG-Management Form (Figure 30). On the basis of the three-dimensional diagnoses, rational three-dimensional short-term and long-term management plans will emerge. In developing a comprehensive management plan, priority has to be assigned to each proposed intervention. An examination of the PEG will help prioritize the management plans; for example, current-context factors such as overwhelming anxiety may require immediate treatment before definitive etiological treatment for the disease can be given. Although intervention at the level of etiology would be most effective in many situations, it is not always possible, for example, in many viral diseases, fractures, or states of pain without identifiable tissue damage.

Setting priorities in management is a matter of clinical judgment. For example, treatment of a grave disease may take priority over the patient's possible loss of a job due to prolonged hospitalization. On the other hand, relief from an anxiety state may take priority over discussing

PATIENT EVALUATION GRID (PEG) - Management Form

DIMENSIONS	DIAGNOSIS	THERAPY PLANS	
		SHORT TERM	LONG TERM
BIOLOGICAL			
PERSONAL			
ENVIRONMENTAL			

Figure 30. On the basis of the PEG, diagnoses are entered in each dimension. The therapy plans for the diagnoses need not be in the same dimensions; for example, a short term therapy plan for depression (personal dimension) may be antidepressant drugs (biological dimension).

elective surgical treatment plans for a patient with a chronic disorder such as inguinal hernia. For a patient with a congenital heart disease (background context, biological dimension) who comes to the physician with vague symptoms and depressive affect (current context, personal dimension) and who has recently lost a spouse (recent context, environmental dimension), the priority may be, first, relief from the depression, which in turn may relieve the vague symptoms; second, interpersonal support; and third, possible treatment of the congenital heart disease. On the other hand, the priority in a chronically depressed patient (background context, personal dimension) who presents with exacerbated old or even new symptoms arising out of current problems of living might well be to deal first with them, rather than with the chronic depression.

To operationalize the use of the PEG for patient-care planning, the following steps may be taken:

1. Underline the factors that appear important:
   a. in the patient's current suffering;
   b. in the pathogenesis of his disease;
   c. in his attitude and fantasies concerning his illness;
   d. in his perception and attitudes concerning the health-care system.
2. Also underline major limiting factors in possible intervention and care, such as personality style, nature and quality of interpersonal relationships (supportive persons), and occupation.
3. On the basis of the PEG and, especially, the underlined factors, fill in the PEG-Management Form.

Summary

Comprehensive assessment of a patient must include evaluation of three levels of systems (dimensions): the biological (component) dimension, the personal (the whole patient) dimension, and the environmental–interpersonal dimension that surrounds the patient. *Disease* is a concept that implies a dysfunction in the biological dimension, while *illness* refers to the biological dysfunction plus the suffering and social problems attendant on the disease. Illness behavior, however, may be present in the absence of disease, and a disease need not produce an illness, especially in early stages.

From the vantage point of medical practice, it is useful to evaluate the three dimensions cited above in terms of three longitudinal time

contexts; current, recent, and background. This is the *systems–contextual* approach to patients. The three dimensions (biological, personal, environmental) intersected by the three contexts (current, recent, background) form the *Patient Evaluation Grid* (PEG), which is the operational method by which information about a patient can be organized.

The PEG allows the clinician to anticipate problems relating to patient care and to assign priorities to management plans formulated in the three dimensions. In each dimension, the current-context information represents the current state and pressing needs, the recent-context information represents the contributing and precipitating factors, and the background-context information represents the broad background factors from which the recent and current problems have emerged and which are often resistant to change and so may impose limits in formulating plans for management.

Implications

For the Patient

The patient brings with him three separate but interacting systems when he seeks help. Although the chief complaint may refer to distress (personal dimension) caused by a disease (biological dimension), the implications of hospitalization, the disease, and treatment plans inevitably involve his family, work, and physical surroundings. Factors in any of the dimensions may become intervening variables that influence the patient's clinical course and behavior in the sick role.

For the Physician

By the use of the PEG and the systems–contextual framework in evaluating patients, the physician can approach or aspire to a comprehensive understanding of the patient. This understanding will complement a narrower medical model of disease that leads to the diagnosis and treatment of diseases rather than patients. Such an approach enables the physician to pay attention to (1) the pressing needs of the patient in the personal dimension [including felt distress (see Chapter 3)], (2) the contributing and precipitating events, and (3) the factors that must be kept in mind as stable and limiting influences (e.g., the patient's personality type and cultural sick-role expectations) on the disease in the narrower sense.

For the Community and the Health-Care System

The need for a holistic, patient-centered perspective in medicine is increasing in urgency (Barondess, 1974). Such a perspective can be obtained by a general systems model of illness and health (Menninger, 1963; Engel, 1960, 1977). The systems–contextual approach we use is also based on a general systems model (Leigh and Reiser, 1977), but it is in practice closely linked with and complementary to the medical model through the use of the PEG (Leigh *et al.*, 1980). We feel that this (or a similar) approach could be adopted with benefit especially in medical schools, where physicians of the future may, from the very beginning, learn the importance and interactions of the nine areas represented by the PEG. With this comprehensive framework, the science of medicine can develop side by side with the art of medicine.

Recommended Reading

Feinstein AR: *Clinical Judgment.* Huntington, New York, Robert E Krieger Publishing Co, 1974. This is an excellent and scholarly book on the science and art of clinical judgment in medicine. It includes discussions of the way physicians collect and use clinical data and the concepts of disease, illness, treatment, and response, among others. It has very interesting and thought-provoking chapters on diagnostic taxonomy, including how the concept of disease has changed throughout history. For example, some of the diseases in Hippocratic times were fever, cyanosis, consumption, and asthma. Highly recommended reading for students in medicine.

Leigh H: The evolution of psychosomatic medicine and consultation–liaison psychiatry. *Adv Psychosom Med* **11**:1–22, 1983. A review of the history of the mind–body relationship in medicine. This article traces the evolution of psychosomatic medicine that led to a recognition of the importance of a comprehensive approach to the patient and to the development of the Patient Evaluation Grid.

Leigh H, Reiser MF: Major trends in psychosomatic medicine: The psychiatrist's evolving role in medicine. *Ann Intern Med* **83**:233–239, 1977. In this article, we give an overview of the development of psychosomatic medicine. We show that the emphasis of psychosomatic medicine is the integration of biopsychosocial factors in the comprehensive management of patients. This is based on the emerging general systems model for all diseases in which the biological, personal, and environmental systems interact.

Menninger K: *The Vital Balance.* New York, Viking Press, 1963. This is a classic work by one of the most venerated psychiatrists in this country on a general systems model of psychiatric disorders. Although Menninger's taxonomy of emotional disorders (e.g., the "orders of dyscontrol") has not achieved wide acceptance, this represents an interesting alternative to the medical model of mental illness. There is extensive discussion on classifications in medicine as well as on the role of the physician in mental illness.

Morgan WL, Engel GL: *The Clinical Approach to the Patient.* Philadelphia, WB Saunders Co, 1969. A classic book on diagnostic work with a patient.

Reiser DE, Schroder AK: *Patient Interviewing, The Human Dimension.* Baltimore, Williams and Wilkins, 1980. An excellent introduction, with many examples, to the art and science of interviewing. It provides a particularly sensitive discussion of the importance of the human dimension in taking medical histories, and guidelines for acquiring requisite skills.

References

Barondess JA: Science in medicine: Some negative feedbacks. *Arch Intern Med* **134**:152–157, 1974.

Engel GL: A unified concept of health and disease. *Perspect Biol Med* **3**:459–485, 1960.

Engel GL: The need for a new medical model: A challenge to biomedicine. *Science* **196**:129–136, 1977.

Feinstein AR: *Clinical Judgment.* Huntington, New York, Robert E Krieger Publishing Co, 1974.

Holmes I, Rahe RH: The social readjustment rating scale. *J Psychosom Res* **1**:213–218, 1968.

Leigh H, Reiser MF: Major trends in psychosomatic medicine: The psychiatrist's evolving role in medicine. *Ann Intern Med* **83**:233–239, 1977.

Leigh H, Feinstein AR, Reiser MF: The patient evaluation grid: A systematic approach to comprehensive care. *Gen Hosp Psychiatry*, **2**:3–9, 1980.

McWhinney JR: Beyond diagnosis: An approach to the integration of behavioral science and clinical medicine. *N Engl J Med* **287**:384–387, 1972.

Mechanic D: The concept of illness behavior. *J Chronic Dis* **15**:189–194, 1962.

Menninger K: *The Vital Balance.* New York, Viking Press, 1963.

Rahe RH: Subjects' recent life changes and their near future illness suspectibility. *Adv Psychosom Med* **83**:2–19, 1972.

Current Context

1. A 42-year-old man was brought to the emergency room of the general hospital by police officers who had found him lying on the street. The patient, unmarried, was identified as a known alcoholic who had been evicted from his room. The presumptive diagnosis after careful examination was alcoholic intoxication. After a few hours, he was alert and coherent, and his vital signs were stable. The emergency room physician felt that he could be discharged without any further treatment.

2. A 33-year-old married man carrying a paper bag containing a week-old sandwich came to see his doctor. He was obviously anxious and agitated. He told the doctor, "I am sure I am being poisoned by my wife. Please examine me and find out what poison she has been using. I brought this sandwich she made for me last week; I am sure that it is poisoned!" For several months the patient had suspected that his wife was having an affair and, recently, had been secretly following her to catch her with her lover. He was convinced that her lover was his boss. As evidence, he cited a recent raise in salary he had received. "Unless he has a guilty conscience, why would he give me a raise?" Although physical examination and laboratory tests showed no evidence of poisoning, the patient refused to believe that he was not being poisoned. In fact, he accused the doctor of colluding with his wife.

3. A 45-year-old woman was admitted to the psychiatric ward because of serious depressive symptoms, including suicidal ideation. On physical examination, her blood pressure was moderately elevated, and it was found that she had been taking a "blood pressure pill" for several weeks. The psychiatrist called her internist, who informed him that the blood pressure

pill was reserpine, a medication known to cause depression. The medication was discontinued.

In assessing patients, it is essential to understand the immediate current situation of the patient and its relation to his help-seeking behavior. In vignette 1, the police sought help for the patient because he was lying on the street drunk. On arrival in the emergency room, he was found to need no further acute medical treatment. For another patient, this would have been enough reason for discharge from the hospital, but this patient could not be sent home—he had no home to go to. The immediate *social* situation, in this case, dictated that he be sent to a temporary shelter with referral to an alcohol rehabilitation program.

In vignette 2, the help-seeking behavior was due to a *psychological state* (paranoid psychosis). In this psychotic state, no amount of reassurance by the physician would change the conviction; rather, the physician himself became an object of suspicion. In vignette 3, the psychiatric problem (depression) was probably due to the effects of the antihypertensive drug reserpine on the central nervous system. Here, the patient's immediate *biological state* determined the nature of the illness that occasioned the help-seeking behavior. What is important to recognize here is that current social, psychological, and biological factors and states can and do participate and interact in influencing illness and help-seeking behavior.

Current Context in the Biological Dimension

The patient's biological state, that is, the state of the component systems, organs, and tissues, is, of course, most often the underlying basis of his illness and help-seeking behavior. The diagnosis of the disease underlying the distress and symptoms often provides the physician with a logical approach to treatment of the *disease*. An understanding of the disease, combined with an understanding of the person and his environment, will lead to a rational management plan for the *patient*.

History, physical examination, and laboratory tests are the tools that provide information concerning the current biological state. In this dimension, one is concerned with bodily states—condition of tissues, organs, organ systems, and other components (e.g., Is the heart enlarged because of overwork? If so, what is causing the overwork? Hyperthyroidism? How is the thyroid gland behaving? What is the plasma thyroid hormone level?)

Current Context in the Personal Dimension

At the level of the whole patient as a unit system, the psychological state and current behavior should be considered. The patient's current behavior includes such observations as whether the patient is complaining of suffering such as pain or is experiencing anxiety related to symptoms such as grossly red (bloody?) urine. The concept of patient behavior (McWhinney, 1972) leading to doctor–patient contact is useful here (see Chapter 1). In addition to the current help-seeking behavior and chief complaints, other behaviors, such as manner of speech, tremor, perspiration, and gait, should be considered.

The patient's psychological state can be ascertained by a mental-status examination and by direct observation of his behavior. The significance of the patient's current psychological state is that it colors the history that can be obtained from the patient, and it influences the efficacy or inefficacy of specific approaches to the patient for optimal management. For example, in the case of vignette 2, the patient's paranoid psychosis resulted in a definite distortion in history. The physician's reassurances only resulted in the breakdown of the doctor–patient relationship. Another example is the depressed patient, whose history is often distorted systematically, that is, thoroughly permeated by a pessimistic, hopeless outlook and perspective as a result of the depressive syndrome. A confused patient will obviously give a confused history.

The mental status consists of the following components: (1) appearance, (2) levels of consciousness and orientation, (3) status of the communicative facilities (speech and movement), (4) cognitive processes (attention, concentration, comprehension, memory, perception, thinking logical thoughts, abstraction, judgment), (5) content of thought, and (6) affect (expression, experience, lability)—both subjective and objective.

Outline of the Mental-Status Examination

Appearance. Appearance is an excellent indicator of the sum total of a patient's mental status at a given point. Sloppy, disheveled appearance often signifies self-neglect or preoccupation and distraction. Flushed appearance and the smell of alcohol on the breath, combined with characteristic drunken behavior, point to the diagnosis of alcoholic intoxication. Pale, emaciated appearance accompanied by malodorous and sloppy dress may indicate the presence of depression or cachexia. Some patients with a lesion in certain areas of the nondominant hemisphere of the brain may dress only one side of the body, completely oblivious

to the presence of the other side. Such patients may pay attention only to one half of the visual field.

Notations on appearance should include observations on the general impression made by the patient (e.g., sloppy, neat, emaciated), including any unusual features (e.g., completely shaved scalp, unusual bodily habitus, dress).

Levels of Consciousness and Orientation. Awareness of self and environment constitutes *consciousness* in the mental-status examination. Consciousness may be subdivided into *content* of consciousness and *arousal.* The sum total of mental functions, including the ability to *remember* and to *think,* comprises the content of the consciousness, while the appearance of wakefulness and response to stimuli form the bases of inference concerning arousal. The content of consciousness is largely a function of the cerebral hemispheres, while the state of arousal is largely a function of the reticular activating system in the brain stem (Plum and Posner, 1972) [see Figure 7 (Chapter 4)].

Arousal: Levels of consciousness may be classified as follows:

- *Hyperalert* state—Increased state of arousal in which the patient is acutely aware of all sensory input. Anxiety and certain central nervous system stimulants can cause this state.
- *Alert* state—Normal degree of alertness.
- *Dullness* and *sleepiness*—May be due to fatigue and insomnia, as well as to sedating medications (as either primary effect or side effect). Metabolic derangements due to disease can also result in dullness and sleepiness, such as in uremia and hypercalcemia.
- *Clouding* of consciousness—A state of reduced wakefulness in which periods of excitability and irritability often alternate with periods of drowsiness. Illusions, especially visual, may occur, and the patient is often startled. Mild to moderate toxic states, withdrawal states, and metabolic derangements can cause this.
- *Confusional* states—In addition to clouding of consciousness, there is consistent misinterpretation of stimuli and shortened attention span. There is disorientation at least to time and often to place. Memory is often poor, and the patient appears perplexed. This is a more severe degree of clouding of consciousness.
- *Delirium*—When used to denote a particular and often fluctuating *level* of consciousness, delirium may include a florid state of agitation, disorientation, fear, misperception of sensory stimuli, and, often, visual hallucinations. The patients are often loud, talkative, and suspicious and are sometimes completely out of contact

with the environment. The degree of contact may vary. Delirium usually occurs in moderately severe toxic states and metabolic derangements of the central nervous system, including withdrawal from central nervous system depressants such as alcohol and barbituates.

The term *delirium* is *sometimes* used to denote *all reversible organic brain syndromes* due to metabolic encephalopathy. When used in this sense, delirium is in contrast to dementia, which implies irreversible changes in the brain. Agitation and florid psychotic picture may be lacking in patients with delirium in this broader sense; that is, the patient with reversible confusion and disorientation may be placid and drowsy rather than agitated.

- *Stupor*—In this state, the patient is unresponsive to stimuli unless their application is very strong and repeated. Usually caused by diffuse cerebral dysfunction.
- *Coma*—Complete unresponsiveness to stimuli. Even strong and repeated stimuli cannot arouse the patient. This occurs in severe dysfunction of the brain, such as serious intoxication or severe head trauma.

Content of consciousness: Orientation refers to the person's consciousness of the orienting markers, such as correct awareness of *time, place,* and *person.* Impairment of orientation results in *confusion.* The orientation of a patient is determined by asking questions such as, ''What day of the week is it today?'' ''Where are you right now?'' ''What is your name?'' In case of insufficiency in the cerebral cortical functions for any reason (most often due to metabolic derangement of the brain or neuronal destruction), orientation may be impaired to varying degrees. Impairment of orientation usually occurs in the order of time, place, and person. In hospitalized patients, disorientation as to date is not uncommon, perhaps due to the change in daily schedule following hospitalization, distractions by the medical procedures, and other disruptions in the patient's usual routine. In the absence of cerebral insufficiency, however, most patients are oriented to the month and year if not to the exact date. Orientation as to person, especially to the patient himself, usually is not impaired until the very latest stage of cerebral insufficiency, although the patient may often forget the names of others, especially those persons encountered recently. Disorientation to the self despite relatively normal mental-status examination in other areas strongly suggests a dissociative disorder rather than cerebral insufficiency (organic brain syndrome).

Status of Communication Facilities (Speech and Movement). In assessing the communicative facilities of a patient, one should consider the integrity of the apparatuses, the effect of learning and psychological state, and the content of the communication.

Integrity of apparatuses: The organs related to speech and movement should be assessed. Weakness of the tongue or facial muscles may produce dysarthria (difficulty in articulating words). Hemiplegia may cause the patient to gesticulate with only one hand. A painful lesion in the mouth may force the patient to be verbally noncommunicative. Deafness may result in nonresponse to a question.

Disorders of language (*aphasia*) caused by brain lesions may be present. Aphasia should be distinguished from dysarthria; the former is due to problems with language itself at the brain level, while the latter refers to difficulty in articulation. In aphasia, written language as well as verbal speech is affected. Aphasias may be roughly classified into *expressive* and *receptive* types. Expressive aphasia is related to lesions of the motor speech (Broca's) area in the dominant frontal lobe of the brain. The patient with expressive aphasia has major difficulties in translating thoughts to symbols; thus, what the patient wishes to express may come out in a distorted form or not at all. He is usually aware of this distortion or difficulty in his own speech and, for this reason, is usually reluctant to speak (or write). Receptive or sensory aphasia is due to lesions of the sensory speech (Wernicke's) area of the dominant temporal lobe. In this condition, the patient has difficulties in comprehending language, including his own speech. Thus, the patient's speech may be garbled, but he may *not* be aware of the problems with his speech. Unlike patients with expressive aphasia, those with the receptive form are usually fluent, although often incomprehensible to others. There are varying combinations and subtypes of aphasias; for more complete discussion, the reader is referred to textbooks of neurology.

Effect of learning and psychological state: Given intact apparatuses for communication, the form of communication often depends on the patient's psychological state and the effect of learning. The effect of learning determines the language in which the patient will express his feelings and thoughts as well as the fluency and facility of the language. For example, middle-class patients are more likely to use grammatically correct syntax and the elaborated code (see Chapter 3). Some patients may use dialects or culturally specific expressions. The current psychological state also determines speech and nonverbal communication. A euphoric patient is more likely to be effusive, verbose, and flamboyant; a depressed patient may be uncommunicative and withdrawn.

Content of communication: What the patient is communicating forms the content of communication. Understanding the content of the patient's communication is essential for the physician to be helpful to the patient. This is more than just accepting the patient's spoken language at face value; it involves an attempt to understand what the patient really *means* through his communication. For example, when a patient comes to the doctor and asks, "Is there a cure for Hodgkin's disease?," much more may be involved than simple intellectual curiosity.

The content of communication often reveals the patient's psychological state, for example, themes of hopelessness and death in depressed states, and bizarre contents in psychotic states. Extreme suspiciousness and ideas of persecution may indicate paranoid psychosis (as in vignette 2).

The content of communication includes the chief complaint, which is the perceived distress for which the patient is seeking help. For a further discussion of its significance, the reader is referred to Chapter 3. Pain is one of the most common chief complaints, and Chapter 9 should be referred to for a fuller discussion. The presence of delusions and paranoid thinking can also be determined by paying attention to the content of the communication (see below).

Cognitive Processes. These are the *processes* that determine the content of consciousness. The processes include attention, perception, memory, concentration, comprehension, abstraction, logical thinking, and judgment. Diminution in the function of any of these areas may indicate the presence of pathology in the cerebral cortex or limbic system. It should be noted, however, that what is important is a *decrease* in function *from the premorbid state* and not necessarily the absolute level of functioning, since the absolute levels of abstract thinking, comprehension, and other processes may be determined by background and by long-term variables such as constitutional endowment, educational level, and habitual functional level, as well as by illness. For example, cerebral pathology is more probably present in a college professor who cannot remember the names of the past five presidents than in a blue-collar worker with a tenth-grade education who evinces the same inability.

The cognitive processes can be tested both indirectly and directly. *Indirectly,* inferences can be made concerning the patient's memory, judgment, concentration, comprehension, and other abilities by asking the patient to describe the present illness and his personal history. Does the patient remember the dates (or years) of graduation from schools, marriage, and other significant events? Does the patient comprehend the nature of his illness and the proposed procedures? Does he remem-

ber what has been told him by the physicians? Does he seem to be aware of the possible risks and complications?

When there is any question concerning the patient's ability to concentrate, comprehend, or think in a logical fashion, a direct and formal testing of the cognitive functions may be necessary.

Direct tests of cognitive processes: In introducing direct tests of cognitive processes, it may be helpful to explain that they evaluate memory, concentration, and so forth and so are useful in evaluating possible mental effects of medications, procedures, and the illness itself. For example, sedating medications may need to be reduced if the patient is found to be too drowsy or if concentration is diminished. This type of reassurance may put the patient at ease about possible errors he may make and gives the testing a medical context.

1. Orientation. The significance of this was discussed above. When doing a direct cognitive testing, it is useful to ask questions concerning orientation first. The questions may be, "What day is today? What is the date (or day of the week)?" If the patient does not know, then "What month is it now?" "What year?" may follow. Mild disorientation as to time (e.g., not knowing the date) is common even among normal persons, but severe disorientation (e.g., not knowing the month and year) is indicative of cerebral dysfunction. "Where are you right now? The name of this place?" These test orientation as to place. If the patient does not know, then he may be asked, "Are you in a hospital, a hotel, or a supermarket?" The patient may know that he is in the hospital (or a doctor's office), but may not know the name of the hospital or clinic, which indicates a milder degree of dysfunction than not knowing the nature of the place or confusing it with somewhere else, such as a hotel room. The next question (orientation as to self) might be, "What is your name?" As discussed previously, dysfunction in orientation proceeds in an orderly manner from time to place to person. In fact, except in cases of very severe brain disease, orientation as to self is usually well preserved. Of course, a delusional patient may have a distorted orientation as to self, for example, "I am Napoleon Bonaparte."

2. Memory, attention, concentration, comprehension. Presidents: "Who is the President of the United States now? If the patient answers correctly, continue asking, "Yes—and before him?," until four or five names have been given correctly. This tests recent memory and information of the patient. Most patients with average high-school education can remember four or five recent presidents.

Calculations: Asking the patient to do simple calculations can test the patient's ability to attend to and comprehend the physician's instructions and to concentrate and utilize immediate memory. "How

much is 15 plus 17?" "25 minus 7?" If the patient has difficulty, an easier calculation involving single digits should be tried. Unlike additions and subtractions, simple multiplications, such as 4 times 6, are easier tasks, since they involve primarily long-term memory (which is resistant to decay) and comprehension of the instructions. Thus, if the patient can do 4 times 6 but not 15 plus 17, then one might wonder whether the patient has difficulties with concentration and immediate or recent memory but not with remote memory and comprehension (indicating possible brain dysfunction). On the other hand, if the patient has difficulties with both, low educational level or mental retardation might be suspected.

If there is reason to suspect difficulties on the basis of simple calculations, serial 7s and digit span might be done. *Serial 7*s are done by asking the patient to subtract 7 from 100 and to keep subtracting 7s from the results. This tests sustained attention and concentration as well as short-term memory. If serial 7s are too difficult for the patient, serial 5s or 2s may be tried. *Digit span* is tested by asking the patient to repeat a number of digits, such as 5—8—3—9—6—2. Digit span backward is tested by asking the patient to repeat in reverse order the numbers that you give him. For example, "If I say 1—2—3, please say '3—2—1.'" This tests primarily short-term memory and concentration. Most patients without brain dysfunction can do at least six digits forward and four digits backward.

Memory, especially recent memory, is very sensitive to dysfunction of the brain. There is some evidence that the limbic system structures, especially the hippocampus, are involved in the coding of recent memory into the long-term memory mechanisms [see Figures 4 and 5 (Chapter 4)]. Any metabolic derangement and structural damage to the limbic system (which is especially sensitive to anoxia) and the cerebral hemispheres can result in problems with memory.

When memory dysfunction is suspected, the physician can test the registration, retention, and recall of memory by the following steps: First, ask the patient if he remembers your name. If he does not, give him your name and ask him to repeat it (immediate memory: registration and immediate recall). Then, tell him that you would like him to remember the names of four objects, such as a pen, a telephone, a flashlight, and a book. Tell him to repeat the names of the four objects immediately. In about five minutes or so, ask the patient if he remembers your name; also, the names of the four objects (recent memory: retention and recall). The patient may be able to remember only one or two objects (diminished recent memory). If the patient cannot recall the names at all, ask the patient, "Please say 'yes' if any of the objects I name now

is one of the objects I named before: pencil, pen, television, telephone, book," etc. (If the patient can identify the articles but could not remember them, it may indicate the presence of retention but difficulty with recall).

3.   Abstraction. Similarities: This tests the ability of the patient to see similarities among objects and to categorize them on the basis of the similarities. For example, "What is the similarity between a cat and a dog?" The patient may answer, "They are both animals" (a good abstraction) or "They both have legs" (a concrete response). In case of the latter, you might ask, "Then how about a dog, a cat, and a snake?" At this point, the patient may be able to abstract and say, "They are all animals." Proverbs: For example, "What does the old saying, 'People who live in glass houses shouldn't throw stones' mean?" "Don't cry over spilt milk," etc.

In testing for abstraction, one should recognize that these are most subject to influences of educational level, cultural background, and language. For example, those from non-English-speaking cultures may have great difficulty in abstracting English-language proverbs.

A concrete response in tests for abstraction may indicate possible brain dysfunction, low educational level, low intelligence, or formal thought disorder, as in schizophrenia. An *idiosyncratic* or *bizarre* response may indicate an unusual way of thinking, as in psychosis. For example, "What is the similarity between a cat and a dog?" "They are both my enemies." "What does the proverb, 'People who live in glass houses shouldn't throw stones' mean?" "That means that even if you have enough money to buy a glass house, you should not throw away money. Stones are gems, you know, which cost a lot of money."

4.   Logical thinking and judgment. Patients with brain dysfunction may show varying degrees of difficulty with judgment. Judgment means the ability to act appropriately in social and emergency situations. Many questions concerning judgment also involve the ability to think logically. For example, "If you were in a crowded theater and happened to discover fire and smoke coming from the ceiling, what should you do?" A good answer would be, "I would tell the usher or manager." If the patient replies, "I would yell 'Fire'", the physician might ask, "If you yelled 'Fire,' what would happen?" The patient with intact logical thinking may then say, "I guess that would cause panic . . . perhaps I should not yell 'Fire.'" Other judgment questions include, "What would you do if you found an envelope with an address and a stamp on it on the street?"

Patients with personality disorders without organic brain dysfunction may give idiosyncratic or inappropriate responses to judgment

questions. For example, an impulsive patient may say, "I would try to put out the fire by throwing my can of soda on it."

5. Perception. The patient's perception can be tested by first observing whether the patient seems to be aware of the tester's presence and whether the patient seems to be responding to visual or auditory hallucinations (e.g., carrying on a conversation or touching). Then, the patient can be asked questions such as, "Have you ever had any experiences of hearing things or seeing things that others couldn't see or hear?" Any experiences of things changing shape or becoming distorted?"

6. Delusions and paranoid thinking. A delusion is an idea firmly held by a patient that is not corroborated by reality. Delusions may be grandiose ("I am God"), persecutory ("Everybody is out to get me"), or depressive ("Worms are eating my brain out"). Some delusions involve diseases, such as, "I know I have cancer, no matter what the tests show." The term "paranoid" is often used to describe patients who have persecutory ideas or delusions.

The presence of delusions is usually manifested by the content of the patient's communications. Delusion formation is a process by which perceptions are put into some kind of perspective. Thus, strange bodily sensations, due to whatever cause, may be attributed to "poisoning" and continuing presence of anxiety to "people spying on me." Obviously, when cognitive processes are not functioning optimally, and when the anxiety level is high (such as in a hosptialized patient with preexisting cognitive difficulties due to poor blood supply to the brain), the risk of delusion formation is greater; it is easier to misperceive stimuli or attribute confusing stimuli to a cause unrelated to reality (e.g., "The doctors are trying to kill me so that they can give my kidneys to someone else").

In addition to indicating the possible presence of cognitive difficulties, delusions give clues concerning the emotional state of the patient. For example, persecutory delusions are associated with anxiety, grandiose delusions with euphoria, and depressive delusions with a depressive syndrome.

Affect and Mood. Affect refers to feeling and is synonymous with emotion, although the latter is sometimes used to denote the physiological aspects of affect [e.g., emotional response (see Chapters 4 and 6)]. Mood refers to prevailing and relatively enduring emotional tone.

Affect can be documented by observation and direct questioning. By observation, one can see whether the patient's affect is *appropriate* or *inappropriate* relative to the topic of conversation (does the patient smile

while talking of sad events?) and whether it is *stable* or *labile*. Labile affect, as manifested, for example, by lauging one minute and crying the next, may be indicative of organic brain dysfunction, in which case there will be additional signs of cognitive difficulties. *Flat affect* means the absence of any display of affect and is often associated with extreme use of isolation as a defense mechanism (Chapter 5) or with schizophrenia.

Direct questions about affect might be, "How do you feel right now?" "Do you feel anxious?" Psyiological signs such as sweating, rapid heart rate, and facial expressions also reveal affective states.

Mood can be ascertained by asking the patient how he has been feeling recently, for the past week or two. Family, friends, and relatives of the patient may also provide useful information concerning the patient's mood. Depression or chronic feelings of anger may contribute to illness and help-seeking behavior.

Current Context in the Environmental Dimension

It is very useful to pay attention to the patient's environment as he comes to the doctor for help. This includes observing who has accompanied the patient and at whose prodding or suggestion the patient decided to seek help, as well as who is available to help the patient should the need arise. For example, who might be able to sign a consent form for procedures in case the patient himself becomes incapable of doing so?

The patient's occupation and work environment are an important area of consideration in terms of possible expense of hospitalization and loss of wages due to illness, as well as in terms of possible toxins associated with certain types of work environments (e.g., heavy-metal poisoning in chemical factories).

The effect of the patient's assumption of the sick role on his family and work should also be considered. The physician may have to help the patient decide whether or not he should delegate his work to someone else during his hospitalization, on the basis of the expected incapacity, the prognosis, and the necessity of the patient's personal involvement in work.

Interaction among the Current-Context Dimensions

The psychological state of the patient (central nervous system), the biological state (state of the organs and tissues), and environmental fac-

tors are closely correlated and influence each other. For example, in anxiety states, there is often an elevation of blood pressure, pulse rate, and gereralized sympathetic nervous system discharge, as well as increases in corticosteroid hormone levels. The anxiety related to hospitalization alone may result in increases in plasma levels of adrenocortical and, possibly, thyroid hormones. On the other hand, organ diseases and endogenous and exogenous toxins can influence the patients' psychological state, including depression, anxiety, personality change, and even psychosis (see Table 16).

An often frightening state in which a patient may present himself to the physician is the state of catatonia—the patient is mute, unresponsive, and rigid. When the physician changes the patient's posture by, for example, lifting up his arm, the arm remains in the new position ("waxy flexibility"). The vital signs are usually normal in this state. This *catatonic syndrome* has been regarded as typical of a form of schizophrenia. Many other conditions and diseases, however, can give rise to many features of the catatonic syndrome [as in Table 6 (Chapter 7)]. The presence of one or more features of the syndrome should not lead the physician to make the diagnosis of schizophrenia and forego further workup to rule out underlying medical or neurological diseases.

As in the case of depression, the presence of any psychiatric syndrome should alert the physician to the possibility that it might be *secondary* to a disease in the biological dimension or to an environmental factor.

Summary

The current context of help-seeking behavior can be assessed at the environmental, personal, and biological levels. The *environmental*-level information is important in understanding the patient's interpersonal resources, which in turn is helpful in planning rehabilitation and convalesence, among other things, and also in understanding possible contributory factors to illness, such as noxious environmental factors.

At the *personal* level, the current psychological state can be assessed by the mental-status examination. This assessment is important because it provides the physician with information on the immediate pressing needs and concerns of the patient as well as the immediate limitations (such as psychosis or confusion).

The current *biological*-level information is the basis for the diagnosis of disease, and the disease itself is often the cause of the patient's distress. There are many biological disease processes and organ states that

Table 16. Partial List of Medical Diseases Often Associated with Psychiatric Syndromes

Anxiety
  Hyperthyroidism
  Pheochromocytoma
  Carcinoid syndrome
  Menopausal syndrome, premenstrual
    tension
  Hyperinsulinism (including insulin
    overdose)
  Withdrawal from a CNS-depressant
    drug (alcohol, narcotics, sedatives)
Depression
  Carcinoma (especially tail of pancreas,
    but any occult cancer can cause
    depression)
  Hypothyroidism
  Hyperthyroidism
  Diabetes mellitus
  Cushing's syndrome
  Addison's disease
  Administration of ACTH
  Antihypertensive therapy (especially
    reserpine, but also with methyldopa
    and propranolol)
  Any electrolyte imbalance
  Uremia
Euphoria/mania
  Hyperthyroidism
  Steroid psychosis
  Multiple sclerosis (depression is also
    common in this syndrome)
  Ingestion of a CNS stimulant (e.g.,
    amphetamines)
Personality change
  Frontal lobe lesion of the brain due to
    any cause, including metastases,
    diffuse encephalopathies, such as
    Alzheimer's disease, and focal lesions
    in lupus erythematosus, and genetic
    disorders such as Wilson's disease
    and Huntington's disease
Paranoid psychosis
  Steroid psychosis
  Hyper- and hypothyroidism
  Hypoglycemia
  Amphetamine psychosis
  Withdrawal from a CNS-depressant
    drug

Organic brain syndromes and toxic
  psychosis
  Electrolyte imbalance
    Acid–base imbalance
    Sodium imbalance (especially
      hyponatremia)
    Potassium imbalance (especially
      hypokalemia)
    Magnesium imbalance (especially
      hypomagnesemia)
    Calcium imbalance
  Drugs (withdrawal or intoxication)
    Alcohol withdrawal
    Barbiturate withdrawal
    Intoxication by any licit or illicit drug
  Overmedication
    Tricyclic antidepressants
    Phenothiazines
    Anticholinergics
    Sedatives
    Analgesics (opiates, pentazocine)
    Digitalis
  Undermedication
    Analgesics
  Environmental toxins
    Heavy metals (e.g., lead, arsenic)
    Organic phosphate compounds
  Ventilation–perfusion problems
    Hypoxia (e.g., anemia, chronic
      obstructive lung disease)
    Congestive heart failure
    Hypovolemia (dehydration,
      hemorrhage, shock)
  Neurological diseases
    Cerebrovascular accidents (CVA)
    Tumor
    Infection
    Trauma
    Epilepsy
    Degenerative diseases (e.g.,
      Alzheimer's disease)
  Vascular diseases
    Arteriosclerosis
    Inflammatory disease of blood vessels
      (e.g., systemic lupus
      erythematosus)

Table 16. (*continued*)

Endocrine diseases	
Hyper- or hypothyroidism	Lack of stimulation
Hyper- or hypoparathyroidism	Poor lighting
Addison's disease	Other
Cushing's syndrome	Hypoglycemia
Deficiency states	Hepatic encephalopathy (ammonia
General malnutrition	intoxication)
Pernicious anemia (B_{12} deficiency)	Uremia
Thiamine	Fever
Pyridoxine	Remote effects of malignancy
Pellagra (nicotinic acid deficiency)	Postanesthesia
Environmental factors	Porphyria
Room location (noisy or too secluded)	Blood dyscrasias

are associated with psychiatric syndromes. In many of these patients, the psychiatric syndrome (e.g., depression) may be the presenting complaint. Treatment of the underlying medical disease is essential for relief from the psychiatric syndrome in these patients.

Implications

For the Patient

The current-context factors such as chief complaint, physical state, and interpersonal strain have the greatest urgency and immediacy for the patient, and help-seeking behavior is determined by, and directed toward, these factors. The current-context variables may also limit the patient's ability to communicate (e.g., stuporous or psychotic state) or call for immediate intervention (e.g., state of hypotensive shock).

For the Physician

Understanding the patient's immediate needs, concerns, and limitations allows the physician to formulate immediate management plans. It is essential to understand the patient's immediate surroundings, including who accompanied the patient to the physician, since the family or friends can be invaluable as information sources and in health-care planning. The physician should be aware that a number of organ states and *diseases*, such as occult carcinomas, can present with *psychiatric syn-*

dromes. Thus, the presence of a psychological abnormality such as depression or psychosis should not be automatically taken as an indication that the patient is a "mental" patient whose problems are caused by factors at the environmental or personal level only. Even those psychiatric patients who have long-standing psychiatric illness may have, in addition, a medical disease.

For the Community and the Health-Care System

Medical education should place more emphasis on understanding patients in all three dimensions: biological, personal, and environmental. Medical records should have provisions for documenting the factors at all three levels. Hospitals should pay attention to the immediate physical and social environments of patients and provide them with comfortable surroundings as well as a place for the family and friends accompanying patients. Hospital staff should be educated concerning the management of patients who are in altered states of consciousness, including stupor and psychosis. Since psychosis occurs in medical conditions requiring medical treatment, medical personnel should be trained to deal with such patients.

Recommended Reading

Alpers BH, Mancall EL: *Essentials of Neurological Examination.* Philadelphia, FA David, 1971. A concise and lucid manual for neurological examination that will complement the mental-status examination discussed in this chapter.

Harvey AM, Johns RJ, Owens AH Jr, *et al.: The Principles and Practice of Medicine*, ed 19. New York, Appleton-Century-Crofts, 1976, Section 1 (The approach to the patient), pp 1–70. A good introduction to obtaining history, the diagnostic process, and approaches to management.

Plum F, Posner JB: *The Diagnosis of Stupor and Coma.* Philadelphia, FA David, 1972. This is an authoritative book on the evaluation of patients showing altered states of consciousness. A very good reference book.

References

McWhinney JR: Beyond diagnosis: An approach to the integration of behavioral science and clinical medicine. *N Engl J Med* **287**:384–387, 1972.

Plum F, Posner JB: *The Diagnosis of Stupor and Coma.* Philadelphia, FA David, 1972.

CHAPTER 13

Recent Context

1. A 50-year-old white married man, accompanied by his wife, comes to the psychiatrist's office with the chief complaint of severe depression that has become progressively worse over the past two months. The patient has trouble sleeping, has lost 20 pounds, and has difficulty concentrating. He has not gone to work for the last three days because, he says, "I feel so bad, I can't do anything." He has been constantly thinking about how worthless he is and has been contemplating suicide. On close questioning, the physician finds that three months ago, the patient was passed over for a promotion at work.

2. A 30-year-old single woman reports increasing asthmatic attacks that began about six months ago. History reveals that she had broken up with her fiancé about seven months ago, after which her drinking increased. Incidentally, she has a cat in her apartment which was given to her by a friend about six months ago.

3. A 66-year-old widower is brought to the hospital by his neighbors because of his disruptive behavior. Lately, he has been observed walking around outside his apartment only half dressed and urinating in the street in full view of his neighbors. He also dumped his garbage in front of his neighbor's door. When the neighbor tried to speak to him, he was argumentative and assaultive. The neighbors could smell a strong stench coming from his apartment. On admission to the hospital (involuntarily, by commitment), he is found to have a tumor in the frontal lobe of the brain.

Also reread the vignettes in Chapter 11.

Recent changes and events in the patient's life often provide the physician with clues concerning the disease process and the factors con-

309

tributing to it. In fact illness itself is a recent change that causes help-seeking behavior. The changes may be a symptom of the disease process, as in vignette 3. Frontal-lobe tumors often cause behavioral changes characterized by the loss of customary social behavior. In vignette 1, the loss of hoped-for promotion was responsible for the depressive behavior. Either the acquisition of the cat or the loss of the relationship with the fiancé, or the two together, may have been active in aggravation of the asthmatic syndrome in vignette 2.

Recent Context in the Biological Dimension

Focusing on the events in this dimension in the recent context forms the *present illness* part of medical history. Recent changes and events in the biological dimension determine the present state and functioning of the organism. The precursors of disease and evidence of organ malfunction may be manifest in the recent past. Information concerning this may be obtained by careful history-taking. Such history should include specific questions about medications, including nonprescription and street drugs. Any history of recent visits to a doctor or hospitalization should be inquired into and information obtained from the physician or hospital.

Recent diseases, whatever the outcome, determine the level of resistance of the patient and the vulnerability to new disease processes. For example, the recent administration of a medication may have started an allergic reaction, setting the stage for a severe hypersensitivity reaction to subsequent intake of the same drug.

Recent Context in the Personal Dimension

The psychological and behavioral aspects of the patient in the recent past comprise the recent context in this dimension. This includes changes in mood, interest, habits, and thoughts and other behavioral changes and events, for example, changes in smoking or drinking habits and changes in sleep patterns, appetite, and sexual activity.

Depression is an important recent-context phenomenon that increases the risk of morbidity from any illness (see Chapter 6). For example, individuals who had an elevation of the depression scale on the Minnesota Multiphasic Personality Inventory (MMPI) were more likely to develop myocardial infarction without preexisting angina pectoris (Bruhn *et al.*, 1969) and also had a poor prognosis once an infarction occurred. Depressed persons, of course, have a tendency to neglect per-

sonal hygiene and have poor dietary habits, which may contribute to increased likelihood of becoming ill. Fatigue, anxiety, and depressed mood may be manifestations of illnesses, such as in the case of hepatitis and infectious mononucleosis. Depression also often occurs secondary to cancer of the tail of the pancreas and other occult malignancies. The presence of depression in the recent context should draw the physician's attention to the possibility of an occult carcinoma or other medical disease that might cause depression [see Table 4 (Chapter 6)].

Changes in personal habits such as drinking and smoking can directly contribute to pathogenesis of such diseases as coronary thrombosis, gastritis, pancreatitis, and liver failure. Increased drinking and smoking may be associated with strains in the environmental–interpersonal system, such as problems at work or with family.

Changes in interests may indicate changes in mood. For example, loss of interest in activities that usually provide the patient with gratification, such as hobbies and sex, is often associated with depression. On the other hand, getting involved in numerous activities and hypersexuality may be associated with a manic disorder. Certain drugs, such as amphetamines, and certain medical diseases, such as hyperthyroidism, can also cause hypomanic or manic behavior (see Chapter 6).

Another important area to be considered in the recent context at the personal level is the sleep pattern, which is an excellent example of the interrelationship among the biological, personal, and environmental dimensions (see Chapter 10).

Recent Context in the Environmental Dimension

Changes in the *physical environment* of the patient can result in diseases associated with noxious agents. Such noxious influences include chemical poisons (e.g., lead and polyvinyls), infectious agents (e.g., Legionnaire's bacillus), and changes in temperature, barometric pressure, noise level, or other factors in the patient's living and working environment. History of foreign travel may explain an unusual infection in some patients.

Contact with street drugs and the drug culture is an important area to be evaluated, especially in relatively young patients. For example, a young man complaining of a peculiar sensation under his skin—that insects are crawling under the skin—may be suffering from the symptoms of cocaine intoxication ("cocaine bug"). In managing a young man admitted with multiple fractures after an automobile accident, it is important to ascertain whether or not he has been habitually using drugs,

especially barbiturates, in order to prevent possible serious withdrawal reaction while in traction on the surgical intensive care unit. Barbiturates should never be withdrawn immediately from addicted patients, because convulsions and even deaths occur frequently during withdrawal. Information concerning drug use or exposure to drugs is often not volunteered. Direct questioning of the patients and friends or relatives is important.

Many heavy-metal poisonings, including lead and mercury poisoning, produce behavioral and neurological manifestations and have to be differentiated from a psychiatric disorder.

The physical environment has effects not only in the biological system of the patient but also in the personal system *through psychological meanings*. A patient who has been accustomed to a large, single private office may become depressed or irritable in a crowded office after a change of job. Thus, environmental changes that may not, of themselves, be sufficient to cause disease directly may nevertheless cause an illness through perception of the change at the personal level.

Changes in the interpersonal–social environment have an important effect on help-seeking behavior in two ways: first, interpersonal strain may increase help-seeking behavior (Mechanic, 1962) (see Chapter 1); second, certain changes in the interpersonal environment may contribute to illness or disease through mechanisms not as yet clearly understood (see Chapters 4 and 6). For example, persons with a past history of residential or job mobility have higher rates of coronary disease than persons who have not had such mobility (Syme *et al.*, 1965; Jenkins, 1976). Significantly increased mortality and morbidity rates were found in bereaved first-degree relatives within the first year of bereavement (Parkes, 1972; Jacobs and Ostfeld, 1977; Rees and Lutkins, 1967). Increased blood pressure was found in persons who were experiencing job loss (Kasl and Cobb, 1970).

Holmes and Rahe (1968) developed the concept of "life change units" (LCUs) and the relative degree of necessary adjustment required to common life changes such as marriage and divorce. The relative weights were determined on the basis of questionnaires given to a large number of subjects. According to this scale, called the social Readjustment Rating Scale, death of spouse has a value of 100 LCUs; divorce, 73; marriage, 50; all the way down to minor violations of the law, 11 (see Table 17). Rahe and his colleagues found that those who had fewer than 150 LCUs for a given year reported good health for the following year, and of those who had between 150 and 300 LCUs, about half reported illness in the following year (Rahe, 1972). Seventy percent of those who had more than 300 LCUs during the year had illnesses in the

Table 17. Life Change Events and Life Change Units[a]

Events	LCU values
Family	
Death of spouse	100
Divorce	73
Marital separation	65
Death of close family member	63
Marriage	50
Marital reconciliation	45
Major change in health of family	44
Pregnancy	40
Addition of new family member	39
Major change in arguments with spouse	35
Son or daughter leaving home	29
In-law troubles	29
Spouse starting or ending work	26
Major change in family get-togethers	15
Personal	
Detention in jail	63
Major personal injury or illness	53
Sexual difficulties	39
Death of a close friend	37
Outstanding personal achievement	28
Start or end of formal schooling	26
Major change in living conditions	25
Major revision of personal habits	24
Changing to a new school	20
Change in residence	20
Major change in recreation	19
Major change in church activities	19
Major change in social activities	18
Major change in sleeping habits	16
Major change in eating habits	15
Vacation	13
Christmas	12
Minor violations of the law	11
Work	
Being fired from work	47
Retirement from work	45
Major business adjustment	39
Changing to different line of work	36
Major change in work responsibilities	29
Trouble with boss	23
Major change in working conditions	20
Financial	
Major change in financial state	38
Mortgage or loan over $10,000	31
Mortgage foreclosure	30
Mortgage or loan less than $10,000	17

[a] From Gunderson and Rahe (1974), with minor modification. Reproduced with permission from Charles C Thomas.

following year. A number of retrospective and prospective studies showed a significant relationship between mounting life changes and the occurrence of sudden cardiac death, myocardial infarction, accidents, athletic injuries, tuberculosis, leukemia, multiple sclerosis, diabetes, and other conditions (Rabkin and Struening, 1976). The presence of social support systems may have a protective effect against the occurrence of illness despite high LCUs (Dean and Lin, 1977). Thus, an understanding of the nature of and changes in the interpersonal environment of the patient is an important part of a comprehensive evaluation of the patient.

To elicit the data related to the recent context in the environmental dimension, the physician should ask *specific questions* in addition to obtaining a chronological history of the present illness. The specific questions include the following: Has there been any change in residence in the past five years or so? Any changes in your job? Any changes in your family such as divorce, separation, remarriage, death of a family member? Did you go on a vacation, and, if so, where? Do you have any difficulties at work? Have you had any trouble with the law? Have you known anyone who became ill recently or who had an operation or an accident?

Summary

The recent context of help-seeking behavior consists of recent events and changes in the environmental, personal, and biological dimensions. In the environmental dimension, exposure to physical toxins and changes in interpersonal systems (such as bereavement and job changes) should be considered. At the personal level, changes in personal habits, attitudes, and mood are important factors. Depression has been shown to be associated with increased morbidity. The recent context in the biological dimension includes recent diseases, surgical procedures, and medications. It also includes biological rhythms, which are influenced by factors in the environmental and personal dimensions and which in turn have far-reaching effects in all dimensions.

Implications

For the Patient

The recent-context events and changes are frequently the sources of current concerns, joys, and sorrows of the patient. Examples include

recent diagnosis of a disease, marriage, or bereavement. Many events and plans that occur in contact with the health-care system will be interpreted by the patient in relation to the recent-context variables. For example, a patient whose friend recently obtained good relief from back pain by surgical procedures would expect surgical procedures if he develops back pain himself. A patient who is newly married may be more concerned about long-term health and more inclined to engage in help-seeking. On the other hand, a patient who has been depressed recently is likely to neglect personal hygiene and not seek medical help despite obvious symptoms and signs. Certain life changes and recent changes may increase the likelihood of the patient's becoming ill.

For the Physician

Understanding the recent events and changes in the patient's bodily functions, behavior and mood, and environment often provides the physician with clues concerning the contributing and precipitating factors of current illness and possible associated conditions that must be considered in managing the patient. They include changes in habits, such as increased drinking or smoking that may contribute to liver and respiratory disease. Environmental changes such as trips or changes in working conditions may be responsible for a rare infection or poisoning. Mood changes such as depression may need to be treated in addition to the disease at hand. Changes in sleep patterns and other biological rhythms often provide the physician with important information.

For the Community and the Health-Care System

In developing a health-care system centered around the patient rather than merely the disease, medical education should emphasize the recent events in the patient's life and environment that have an important impact on the patient's current feelings and attitudes. Understanding of the recent-context information should lead to preventive measures and improvement of the overall quality of the patient's life by managing aspects of the patient's recent experiences that are amenable to intervention. This includes teaching better coping skills.

Recommended Reading

Gunderson EKE, Rahe RH (eds): *Life Stress and Illness*. Springfield, Illinois, Charles C Thomas, 1974. A symposium on the topic of life stress and illness. Multiauthor, mul-

tifaceted discussion on the role of life changes, especially in heart disease and depression.

Jacobs S, Ostfeld A: An epidemiological review of the mortality of bereavement. *Psychosom Med* **39**:344–357, 1977. This is a succinct and comprehensive review of literature concerning the increased risk of morbidity and mortality following bereavement. A critique of the methodologies used in the studies as well as a discussion of possible mediating factors in the increased morbidity can be found in this article.

Jenkins CD: Recent evidence supporting psychologic and social risk factors for coronary disease, Parts 1 and 2. *N Engl J Med* **294**:987–993, 1033–1938, 1976. This is a comprehensive review of the psychosocial risk factors in coronary disease, the most common cause of death in the United States. Many recent-context factors, such as anxiety, depression, and interpersonal problems, seem to be precursors of angina pectoris. Sleep disturbance is shown to herald all presentations of coronary disease. Jenkins also discusses the association between coronary disease and certain background-context risk factors, such as status incongruity, dietary habits, and the type A personality pattern. Highly recommended.

References

Bruhn JG, Chandler B, Wolf S: A psychological study of survivors and nonsurvivors of myocardial infarction. *Psychosom Med* **31**:8–19, 1969.

Dean A, Lin N: The stress-buffering role of social support. *J Nerv Ment Dis* **165**:403–417, 1977.

Gunderson EKE, Rahe RH (eds): *Life Stress and Illness.* Springfield, Illinois, Charles C Thomas, 1974.

Holmes I, Rahe RH: The social adjustment rating scale. *J Psychosom Res* **11**:213–218, 1968.

Jacobs S, Ostfeld A: An epidemiological review of the mortality of bereavement. *Psychosom Med* **39**:344–357, 1977.

Jenkins CD: Recent evidence supporting psychologic and social risk factors for coronary disease, Parts 1 and 2. *N Engl J Med* **294**:987–994, 1033–1038, 1976.

Kasl SV, Cobb S: Blood pressure changes in men undergoing job loss: A preliminary report. *Psychosom Med* **32**:19–38, 1970.

Mechanic D: The concept of illness behavior. *J Chronic Dis* **15**:189–194, 1962.

Parkes CM: *Bereavement: Studies of Grief in Adult Life.* New York, International Universities Press, 1972.

Rabkin JG, Struening EL: Life events, stress, and illness. *Science* **194**:1013–1020, 1976.

Rahe RH: Subjects' recent life changes and their near future illness susceptibility in Lipowski ZJ (ed): *Advances in Psychosomatic Medicine.* Vol 8: *Psychosocial Aspects of Physical Illness.* Basel, Karger, 1972, pp 2–19.

Rees WD, Lutkins SG: Mortality of bereavement. *Br Med J* **4**:13–16, 1967.

Syme SL, Hyman MM, Enterline PE: Cultural mobility and the occurrence of coronary heart disease. *J Health Hum Behav* **6**:178–189, 1965.

Background Context

1. An 18-year-old single white man was admitted to the hospital for an open-heart procedure. He was a thin young man who appeared anxious and sickly. At an early age, he had been diagnosed as having a congenital heart defect (tetralogy of Fallot). His mother was observed by the staff to be hovering over him always and generally overprotective. The patient was considered by the staff to be immature and impulsive.

2. A 19-year-old single white man, John, has just graduated from high school with honors. This was a source of deep satisfaction for his family physician, because John has superior intelligence despite phenylketonuria, a hereditary disease that can cause severe mental deficiency if it is not diagnosed and treated early. In John's case, the condition was diagnosed at birth and treated with a specific (phenylalanine-free) diet. His family, with the help of the doctor, has successfully prevented the mental deficiency.

3. If certain birds, such as mallard ducks, are first exposed to a human after hatching, they will follow that person and for the rest of their lives behave toward him as if he were their mother. This phenomenon, called "imprinting," occurs only during a limited period in early life, roughly between 16 and 32 hours after hatching.

4. A 43-year-old married black man was admitted to the hospital with bronchial pneumonia. On admission, he was considered by the staff to be guarded and suspicious. History revealed that he had grown up in the South and as a child had spent an entire year in the hospital. The prolonged stay was due to chronic recurrent complications following surgery, and the child and his family had felt somewhat mistreated.

Constitution (genetic plus early experiential factors); congenital conditions; perinatal, infantile, and early childhood environment; physical

and personality development and familial influences; and broad cultural factors constitute the background context of help-seeking behavior. Some of the factors are biologically programmed, for example, genetic factors and possibly imprinting phenomena similar to those observed in animals (vignette 3). Generally, traits reflecting background-context factors are relatively stable and not easily modified by the time the patient seeks help. Certain congenital anomalies, however, may be amenable to curative intervention at any time in one's life, as in vignette 1.

The physician should recognize that alleviation of a long-standing disability may require major psychological adaptation by the patient and the family. For example, some patients who have been blind for a long time due to cataracts may paradoxically become psychotically depressed or paranoid after an operation that is successful in restoring vision.

Background Context in the Biological Dimension

The background-context factors in the biological dimension include the genetic endowment and other constitutional factors of the patient. The latter include nonhereditary congenital anomalies, such as the sequelae of rubella infection of the mother during the first trimester of pregnancy.

A genetic abnormality does not necessarily result in phenotypic expression. For example, in phenylketonuria, proper diet can completely prevent the mental retardation (vignette 2).

Certain physical and laboratory findings may serve as "genetic markers" of an abnormality or a predisposition to a disease. For example, long fingers and toes in conjunction with high arched palate indicate Marfan's syndrome. Persons with Marfan's syndrome are particularly predisposed to develop serious vascular disease (dissecting aneurysm) due to connective-tissue abnormality. Elevated serum pepsinogen level (which is a genetic trait) is a marker of those who are at risk for developing duodenal peptic ulcer disease. Interestingly, elevated serum pepsinogen is associated with certain psychological characteristics common in duodenal peptic ulcer patients (Weiner *et al.*, 1957). This suggests that the psychological characteristics may result from complex interactions among the effects of infant behavior, the genes that cause elevated serum pepsinogen, and the behavior of the mother during the child's development. For example, Mirsky (1958) postulated that infants with this genetic trait may have a higher than normal need for nurturance and loving contact, thus setting the stage for frustration of these needs if extra amounts of care are not provided. This may lead to development

of the psychological characteristics that result from conflict over dependency wishes. Background-context data at the biological level should include family history of diseases and illnesses, including alcoholism, substance abuse, psychiatric illnesses, and suicide.

Background Context in the Personal Dimension

The relatively stable (trait) characteristics of the patient as a whole constitute the background context in this dimension. They include the personality type, intelligence, general outlook and tendencies, attitudes, habits, and habitual psychological defense mechanisms and coping styles in the face of illness. The patient's personality type and his habitual defense mechanisms are important in his adaptation to the sick role (see also Chapters 5 and 17). The background-context personality factors generally represent stable *"givens"* that the physician and the health-care system must respect and work with, rather than "problems" to be treated. This is especially true in short-term treatment situations such as brief hospitalizations.

Information concerning the patient's *habits* may be helpful both in shedding light on the diagnostic process and in suggesting management approaches. Personal habits include drinking and smoking, drugs, personal hygiene, exercise, sleeping, and diet. Specific questions should be asked about habit patterns. For example, "How much do you drink? Every night?" "How long have you been smoking?" "Have you been smoking more or less recently, say, in the past month?" A history of excessive drinking in a jaundiced man suggests a diagnosis of alcoholic cirrhosis, and hemoptysis in a middle-aged man who is a heavy smoker suggests possible lung cancer. A patient who has a history of heavy drinking is at risk for developing delirium tremens if an acute medical condition prevents him from drinking. It may be necessary to give sedatives such as benzodiazepines prophylactically to such a patient who may be in the hospital for an acute medical condition unrelated to alcoholism.

Attitude toward illness is usually long-standing and determines the patient's illness behavior as well as sick-role performance. It is influenced by many factors, including the patient's socioeconomic status, religion, other cultural expectations, and personality.

The *coping style* in the face of illness is a concept described by Lipowski (1970). The concept is closely related to the patient's personality type (Chapter 17) and defense mechanisms (Chapter 5). In the presence of the specific threatening situation of illness, many patients tend to

show one of the following two *cognitive* coping styles: *minimization* and
vigilant focusing. Patients who minimize tend to use selective inatten-
tion—not hearing, not seeing, not understanding, or ignoring the facts
or the significance of the illness or its consequences or both. Obviously,
this type of cognitive coping tends to occur in conjunction with the
psychological defense mechanisms of *denial, repression,* and *rationaliza-
tion.*

Vigilant focusing is characterized by hypervigilance and is often
associated with the orderly, exacting personality. The patient copes with
the threat by paying sharp attention to the problem and attempting to
know everything associated with it. There is often a constriction of
awareness so that matters not relevant to the problem at hand may be
ignored. *Isolation of affect* and *intellectualization* are defense mechanisms
that are frequently used in conjunction with this cognitive coping style.

Personality as a Factor in the Pathogenesis of Disease

Some specific personality types and conflicts have been considered
to be especially associated with certain specific medical diseases (Alex-
ander, 1950). For example, peptic ulcer patients were observed to show
exaggerated aggressive, ambitious, and independent attitudes as an over-
compensation for repressed dependency needs and longing for love.
Such a psychological conflict (between dependency needs and inhibition
of them because of personal and social expectations) was considered to
contribute importantly to the etiology of the *"psychosomatic" diseases.*
Although there is often an association between patterns of psychological
conflicts and certain medical diseases, such association is no longer con-
sidered to be etiological (although "core conflicts" may be related to
vulnerability to disease, and their activation may play some role in pre-
cipitation of disease states) (Leigh and Reiser, 1977).

More recently, overt behavior patterns such as the *type A behavior*
described by Friedman and Rosenman (1974), characterized by ambi-
tiousness, competitiveness, and a sense of time urgency, have been
associated with an increased risk of coronary disease, especially in
younger individuals, but it is still unclear whether (or, if so, in what
way) this behavior pattern may be causally related to the development
of cornoary artery disease.

Theoretical concepts have been changing rapidly in response to con-
stant accumulation of new research data (for a fuller discussion of this
topic, see Reiser, 1975, 1984).

Background Context in the Environmental Dimension

Broad cultural influences, including the patient's ethnic origin, socio-economic status of his family during childhood, the region or country in which he spent his childhood, native language (or bilinguality), religious background, and the size of the family (e.g., number of siblings, presence or absence of extended family), are among the factors that comprise the background context in the environmental dimension.

As already discussed in Chapter 3, *language* is often associated with the patient's socioeconomic status as well as with his early cultural background. Whorf (1956) postulates that language actually shapes the way experiences are perceived and processed. For example, the Hopi Indians do not have words that denote time as an entity. Thus, time is perceived as continuous and flowing, without the familiar sense of the *amount* of time and *division* of time (such as "two days ago" or "three years from now"). The Indo-European languages (which includes most of the Western languages, including English) have a tendency to divide events into nouns, adjectives, verbs, and adverbs. This analytic mode of thinking is also the basis of scientific thought. However, there is a tendency to equate the word with an entity; that is, if a word exists, there is a tendency for us to assume that it also has a body and substance (*reification*). Creating a word for a condition or disease also gives a sense of control over it—thus, the desire of patients to know the name of the disease.

The Whorfian hypothesis of language determining thought is controversial; for example, Chomsky (1972) has shown that the "deep structures" of language, which are ultimately reflected in thought processes, may be universal across cultures, while the "surface structure," the actual syntactical arrangement and use of words, may be culture-bound.

Religion plays a role in personality development and in the patient's relationship to illness. Durkheim, in his classic book, *Le Suicide* (1897/1966), postulated that the higher suicide rate among Protestants in Europe in comparison to Catholics was related to the spirit of free inquiry, desire for knowledge, and weakening of the communal bond in Protestantism. This was called egoistic suicide—suicide due to exaggerated individualism in which life becomes empty and loses its meaning. The other types of suicide described by Durkheim are "anomic suicide," due to the lack of regulation in activity, as in achievement and times of financial well-being, and altruistic suicide, in which the social bonding is so strong that cultural goals are found beyond individual existence (see Chapter 6). Members of certain fundamentalist sects and of some Catholic households may see illness as an act of God and take a fatalistic attitude toward it and, consequently, may be less likely to seek medical

help. Some medical procedures may come into conflict with the patient's religious background, such as abortion in Catholics and blood transfusion in Jehovah's Witnesses.

It is important to recognize that patients have *varying degrees* of religiosity and also that the childhood religion or parental religion is *not* necessarily identical to the patient's. Thus, some patients derive great comfort from seeing a minister or priest in the hospital, while others feel upset or angry when visited by the clergy. It is necessary to *ask* the patient about religious matters and the desirability of involvement with the clergy. Some patients assume that the involvement of the clergy necessarily means that their illness is terminal.

Early family environment is probably the single most important factor in the background context in the environmental dimension. This includes physical aspects, such as nurturance, dietary provisions, and contact behavior. For example, in the treatment of phenylketonuria (vignette 2), it is essential to provide the child with a phenylalanine-free diet. Parental cooperation is obviously essential, and in turn, is determined by their involvement with the child, whether the child was planned and wanted or not, and the quality of the marital relationship. Experiments with monkeys by Harlow and his associates show that *tactile sensation* is an important element of attachment behavior (Harlow and Harlow, 1966a,b). They showed that if infant monkeys were given inanimate dummies with apparatus for the provision of milk as mother surrogates, they invariably chose the soft terrycloth-clad surrogates ("terrycloth mothers") as opposed to "wire mothers." In fact, even if the terrycloth mothers did not provide nurturance, the infants clung to them rather than to the tactilely unsatisfying wire mothers. The monkeys who were reared by such artificial mothers had difficulties in social and sexual behavior in adulthood, but some of the adverse effects were neutralized by exposure to peers in childhood.

In mice, early isolation results in relatively lower blood pressure in adulthood, but if the isolated mice are exposed to intense psychosocial stimuli through situations of dominance challenge, their blood pressure rises to a significantly greater extent than that of nonisolated controls (Henry *et al.*, 1975). Thus, early experiences of a social interactional nature may determine the reactivity to situations in later life.

Family interactions play an important role in generating predisposition to psychiatric illness. For example, Lidz *et al.* (1965) found that the families of adolescent schizophrenic patients were often characterized by marital schism or marital skew. In the *schismatic families,* each spouse was caught up in his or her own personality difficulties with a chronic failure to achieve complementarity of purpose or role reciprocity.

In the *skewed families*, the psychopathology of one marital partner dominated the home so that often very bizarre and distorted ideas and behaviors were taken as the norm in the families.

Intersystems Interaction in the Background Context— Development

The process of the patient's ontogeny (*development*) occurs through time by the interaction of the three systems—biological, personal, and environmental. The process of physical maturation itself is genetically programmed, including the development of secondary sexual characteristics. The development of the person, including the personality characteristics and assumption of social roles, is a result of complex interactions of factors at all levels of organization. A detailed discussion on development is beyond the scope of this book, so the reader is referred to a standard textbook of child development (e.g., Lewis, 1971).

In this section, we will briefly and schematically review the stages of libido development as originally described and conceptualized by Freud* and the developmental schemata of Erikson and Piaget. All these theories are *epigenetic;* that is, a later development stage is built on the foundation of the preceding stage, so that difficulties in any stage may result in difficulties in the next stage. On the other hand, successful overcoming of a difficulty in any given stage can help to resolve difficulties carried over from a previous stage.

Freud

Freud's earliest and major developmental theory is based on the *libido theory,* e.g., the vicissitudes of the sexual drive. He postulated that the primary erogenous zones of the sexual libido were concentrated in the mouth during the first year of life—the *oral stage.* In other words, the mouth is the organ predominantly involved in the experience of pleasure during this period. The pleasure may be in sucking and, later, in biting. Initially, during this stage, the object of attachment or love is considered to be the self (autoerotic); that is, pleasure is derived from the self in the act of sensual sucking. Later in the same stage, the infant

---

* This omits important later additions by Freud and others to psychoanalytic theory concerning the psychology of the ego and its development. (See recommended readings by Brenner, 1965 and Meissner, 1985.)

comes to recognize that the mother provides him with the food, the breast, and comfort. Thus, his mother becomes the love object. Passive longings and dependency are thought to be associated with this stage.

The second stage of development according to Freud is the *anal stage,* which occurs during the second year of life simultaneously with toilet training. During this stage, the child's major bodily concern shifts to the anus and its control—which provides a sense of mastery. The act of excretion itself, as well as retention of feces, can be pleasurable. Sadistic impulses and concerns over control and mastery are associated with this stage. The oral and anal stages are considered to be *pregenital* stages as compared to the next stage of development, the phallic or Oedipal stage.

The *phallic* or *Oedipal stage* takes place between the ages of approximately 3 and 6 and is ushered in with the discovery of the genitals by the child. In this stage, erotic sensation concentrates in the genital organs, and the child experiences strong love directed toward a parent. Freud postulated that boys and girls differ in experiencing this stage. For both boys and girls, the mother is the initial object of love. During this stage, the boy feels an intense desire to possess his mother and replace his father as her primary lover. This incestuous wish, however, results in the fear of retaliation by the father in the form of castration (*castration anxiety*). The boy gradually relinquishes his incestuous wishes and, instead, identifies with his father—to become like him, so that in the distant future, he would possess a woman like his mother. Thus, the boy's interest, for the time being, turns away from love and sex, and a period called "latency" sets in. Freud thought that the discovery by the boy that girls do not have a penis was a major factor confirming the fear that castration could actually occur.

In the case of a girl, Freud postulated, the discovery that she does not have a penis results in a serious disappointment in her mother, her initial love object. Thus, according to Freud, she feels defective and becomes envious for a penis (*penis envy*). Because of this feeling of having been castrated, the girl turns her love from the mother toward the father, the person who, unlike the mother, has a penis. Freud postulated that the girl thus has the fantasy of obtaining a penis from her father, which becomes transformed into a wish to have a baby, a penis substitute. In the girl, too, latency occurs because of the fear of complete loss of love from her mother—which produces an identification with the mother.

The phenomenon of little boys and girls experiencing an intense love toward the parent of the opposite sex is called the *Oedipus complex*, after the Sophocles tragedy *Oedipus Rex*. Psychoanalytic theory postu-

lates that unsuccessful resolution of the Oedipus complex is the cornerstone of the development of certain types of neurosis.

If the child has difficulties in progressing from one stage to another because of severe frustrations or excessive gratification, psychoanalytic theory postulates that there may be a *fixation* at that developmental stage; that is, the characteristics of the stage may persist throughout life. Thus, conflicts over dependency or over control and mastery may be the result of fixation at the oral or anal stage, respectively. Problems with sexuality and love often arise as a result of difficulties in resolving the Oedipal situation successfully. When a person encounters difficulties, there may be a tendency for him to return to the behavioral and psychological patterns of an earlier developmental stage in which more gratifications were experienced or a sense of mastery occurred. This phenomenon is called *regression* and is considered to be an important mechanism in psychopathology. Regression may be responsible for secondary enuresis in a child who feels jealous of a newly arrived sibling. [Regression may also be adaptive (see Chapter 5).]

The period of *latency* lasts from about age 7 until adolescence. During this period, children relinquish intense sexual interest and tend to play with playmates of the same sex.

With the onset of adolescence, there is a resurgence of heterosexual interest. The true *genital stage* sets in as the person finds a spouse and is involved in a mature love relationship.

Although the phenomena of infantile sexuality and Oedipus complex are well established, the concept of penis envy has undergone modifications. Thus, the girl's penis envy may be more related to the culturally inferior expectations of girls in the past rather than to the anatomical difference *per se.* In the boy, a wish to fuse or identify with the mother and a wish to be able to bear children may also have important consequences in development.

Erikson

Erik Erikson (1963) described eight developmental stages in humans on the basis of *developmental tasks.* Erikson, who is himself a psychoanalyst, has paid special attention to the relationship of the developing individual to the demands of society and culture. His stages are as follows:

1. Basic trust vs. basic mistrust. This stage is essentially identical to the Freudian oral stage, in which the infant, through the experience of a trusting relationship with the society (family) in the form of feeding,

comfort, and other aspects, learns to develop a sense of basic trust and confidence. Unsuccessful outcome results in a basic sense of mistrust.

2. *Autonomy vs. shame and doubt.* This stage roughly parallels the Freudian anal stage, in which a sense of regulation or control develops. "Holding" and "letting go" are the two social modalities the child *experiments* with. Successful outcome results in a sense of autonomy, a sense of self-control without loss of self-esteem. Difficulties with the developmental task result in a pervading sense of shame and doubt.

3. *Initiative vs. guilt.* This stage is parallel to the Freudian stage of the Oedipus complex (phallic stage). During this stage, the child is more energetic, more loving, brighter in his judgment, and, at the same time, more relaxed. There is a quality of planning and "attacking" a task. During this stage, as a result of regulation between inner drives and internalized inhibitions (due to a developing conscience), the child gradually develops a sense of moral responsibility and unconflicted initiative. Difficulties in this period result in complete repression of initiative or in overcompensatory "showing off."

4. *Industry vs. inferiority.* Paralleling the Freudian latency period, the child learns to win recognition by learning and producing things. Children receive systematic instruction during this stage and acquire the basic skills (such as the three Rs—reading, 'riting, and 'rithmetic) required for work. Unsuccessful outcome results in a sense of inadequacy and inferiority.

5. *Identity vs. role confusion.* Erikson considers the period of adolescense to be of great importance in the development of the sense of identity. This is the period in which the person develops a sense of inner sameness and continuity, a sense of direction and self. Career choices are also made during this stage. The danger in this stage is "role confusion."

6. *Intimacy vs. isolation.* This covers the period of young adulthood, in which the person is ready and eager to fuse his identity with those of others. This involves commitment to concrete affiliations and partnerships with others. Mature love relationships, colleagueships, and friendships develop. Difficulty in achieving this developmental task results in a sense of isolation and self-absorption.

7. *Generativity vs. stagnation.* This is the stage of middle adulthood, in which the primary concern is the establishment and guidance of the next generation. Generativity includes the concepts of creativity and productivity. Childbearing and childrearing are common generative activities. Failure to participate in generative tasks often results in a sense of stagnation and personal impoverishment. Erikson states that such individuals may regress to an obsessive need for pseudointimacy and

indulge themselves as though they were their own or one another's only child. Early invalidism may become a vehicle of self-concern.

8. Ego integrity vs. despair. In old age, acceptance of the individual's life with satisfaction is reflected in "ego integrity," a sense of assurance of order and meaning for humankind. The lack of this integrity is signified, according to Erikson, by fear of death and despair that the time is now too short to attempt to "start another life or try out alternative roads to integrity."

It will be noted further that the Eriksonian developmental stages are comparable to the Freudian ones except that the Freudian stages are based on sexual drives, while the Eriksonian stages are based on "ego tasks," and that Erikson's stages span the whole life cycle, while Freud's stages stop with the development of mature genitality.

Piaget

Jean Piaget, a Swiss psychologist, studied the development of intelligence in children. Piaget's developmental stages, then, are based on the structure of intelligent thinking, rather than on love relationships, as in Freud, or on social tasks, as in Erikson. According to Piaget, one can distinguish three periods of intellectual development: (1) the sensorimotor period (birth to 2 years), (2) the period of representative intelligence and concrete operations (2 to 11 years), and (3) the period of formal operations (past 12 years through adulthood).

1. Sensorimotor period. During the first 18 months to 2 years of life, the infant learns through repetition—initially, from blind imitative repetition to repetitions in anticipation of results and invention of new methods through combinations of previous experiences. Response–feedback loops called "circular reactions" form an important part of the experience.

2. Period of representative intelligence and concrete operations. This stage may be subdivided into the preoperational phase (ages 2 to 7 years) and the phase of concrete operations (ages 7 to 11 years).

   a. Preoperational phase (2 to 7 years). During the period of 2 to 4 years of age, the child begins to use symbols; that is, he begins to use a word or an object to stand for or represent something else that is not immediately present. Symbolic play (e.g., making believe that a piece of cloth is a pillow) and language soon develop.

   b. Phase of concrete operations (7 to 11 years). When the child develops the notion of conservation (of volume or weight), he

is in the period of concrete operations. Now the child knows that the same amount of clay can look as though there were more clay if divided into a number of smaller balls. The child also has a clear notion of hierarchical classifications, especially if concrete objects are involved. For example, he can answer correctly the question, "Which would make a bigger bunch, one of all the primulas or one of all the yellow primulas?" (Ginsburg and Opper, 1969).

3. *Period of formal operations.* As the child reaches adolescence, his thought processes move from concrete operations to a more flexible, abstract mode. The thought transcends here and now. With adolescence, the person begins formal, propositional thinking, in which logical thinking and hypothesis-testing are prominent features.

Piaget's developmental stages are based primarily on normal children, and his theory does not form the basis of psychopathological understanding. However, it emphasizes the process of "discovery learning"—derivation of a higher level of intellectual functioning by the incorporation of experience.

Summary

The background context includes hereditary, constitutional, personality, and early environmental factors such as language, religion, and family interactions. Early physical environment may also play a major role in the present state of the patient.

At the personal level, personal habits, including drinking and smoking, personality and coping styles, and behavior patterns should be evaluated. Genetic givens and laboratory findings often provide clues to constitutional vulnerabilities to illness.

Development is a complex intersystems event. Freud formulated developmental stages on the basis of sexual drives, with emphasis on the love relationships in children. Erikson formulated developmental stages on the basis of social tasks, and the whole life cycle is seen as having period-specific tasks. Piaget studied the intellectual development of children.

Implications

For the Patient

The factors in the background context determine the patient's stable expectations of himself, the environment, and the health-care system.

They also determine his skills, talents, and limitations. Often, patients are not aware of the specific ways in which the background-context factors determine their current attitudes and feelings.

For the Physician

Understanding the patient's background-context factors in all three dimensions is essential in arriving at rational management plans. The physician should also consider what background factors tend to reinforce, and what factors tend to conflict with, the patient's recent and current context factors. For example, a Catholic patient whose depression followed a therapeutic abortion may be influenced by the background-context factor of childhood religion, although she may no longer profess Catholicism. In this instance, recognition of the source of guilt feelings in childhood may aid in alleviating present depression. Early experience of mistreatment may determine present perception of the health-care system, as in vignette 4. The physician should ask specific questions concerning early family environment, socioeconomic status, and residence, parents' occupations, ethnicity, religion, and other background factors when obtaining a family history and past history.

For the Community and the Health-Care System

Medical education should include an understanding of the role of background environment and habits in the pathogenesis of illness. Educational programs concerning smoking and excessive drinking can have major preventive effects. More research is needed in order to understand optimal environment and childrearing techniques.

Recommended Reading

Brenner C: *An Elementary Textbook of Psychoanalysis*. New York, International Universities Press, 1955. An exceptionally lucid and concise introduction to the psychoanalytic theory.

Erikson EH: *Identity, Youth and Crisis*. New York, WW Norton & Co, 1968. This contains discussion on the identity problem of adolescence and a review of Erikson's ideas concerning the developmental stages throughout the life cycle. This work and Erikson's *Childhood and Society* are very important in understanding the social–cultural influences in the personality system.

Freud S: *The Complete Introductory Lectures on Psychoanalysis*, Strachey T (ed). New York, Norton, 1966. A lucid and easy-to-read exposition of Freud's basic ideas. Highly rec-

ommended for an understanding of Freud's early developmental theory and psycho-
analysis.

Ginsburg H, Opper S: *Piaget's Theory of Intellectual Development: An Introduction.* Englewood
Cliffs, New Jersey, Prentice-Hall, 1969. This is a very readable and substantive review
of Piaget's contributions in understanding intellectual development in children. It
describes the major experiments done by Piaget with good discussions of the impli-
cations.

Lewis M: *Clinical Aspects of Child Development.* Philadelphia, Lea and Febiger, 1971. This
is a concise and readable textbook on child development. It is an overview of devel-
opment from the prenatal period to early adolescence. it also contains a chapter on
psychological reactions to bodily stresses such as acute illness, birth defects, and sur-
gery. Highly recommended.

Meissner WW: Theories of Personality and Psychopathology: Classical Psychoanalysis, in
Kaplan HI and Sadock BJ (eds): *Comprehensive Textbook of Psychiatry,* ed. 4. Baltimore,
William and Wilkins, 1985, Vol 1, pp. 337–418. An up-to-date and comprehensive
review of the psychoanalytic theory.

Reiser MF: Changing theoretical concepts in psychosomatic medicine, in Reiser MF (ed):
American Handbook of Psychiatry, ed 2. New York, Basic Books, 1975, Vol 4, pp. 477–
500. A comprehensive and historically oriented review of theoretical concepts in psy-
chosomatic medicine.

Reiser MF: *Mind, Brain, Body: Toward a Convergence of Psychoanalysis and Neurobiology.* New
York, Basic Books, 1984. Contains more detailed conceptualizations concerning the
role of psychological factors in disease.

Shapiro D: *Neurotic Styles.* New York, Basic Books, 1965. For a very vivid description of
some personality styles.

References

Alelxander F: *Psychosomatic Medicine.* New York, WW Norton, 1950.

Chomsky N: *Language and Mind.* New York, Harcourt Brace Jovanovich, New York, 1972.

Durkheim E: *Suicide,* Simpson G (ed). New York, The Free Press, 1966. (Originally pub-
lished in French, in 1897.

Erikson EH: *Childhood and Society,* ed 2. New York, WW Norton & Co, 1963.

Friedman M, Rosenman RH: *Type A Behavior and Your Heart.* New York, Alfred A Knopf,
1974.

Ginsburg H, Opper S: *Piaget's Theory of Intellectual Development: An Introduction.* Englewood
Cliffs, New Jersey, Prentice-Hall, 1969.

Harlow M, Harlow H: Affection in primates. *Discovery* 27:11–17, 1966a.

Harlow H, Harlow M: Learning to love. *Am Sci* 54:244–272. 1966b.

Henry JP, Stephens PM, Axelrod J, *et al.*: Effect of psychosocial stimulation on the enzymes
involved in the biosynthesis and metabolism of noradrenaline and adrenaline. *Psy-
chosom Med* 37:227–237, 1975.

Leigh H, Reiser MF: Major trends in psychosomatic medicine: The psychiatrist's evolving
role in medicine. *Ann Intern Med* 87:233–239, 1977.

Lewis M: *Clinical Aspects of Child Development.* Philadelphia, Lea and Febiger, 1971.

Lidz T, Fleck S, Cornelison AR: *Schizophrenia and the Family.* New York, International Uni-
versities Press, 1965.

Lipowski ZJ: Physical illness, the individual and the coping process. *Psychiatry Med* 1:91–
102, 1970.

Mirsky LA: Physiologic, psychologic, and social determinants in the etiology of duodenal ulcer. *Am J Dig Dis* **3:**285–314, 1958.

Reiser MF: Changing theoretical concepts in psychosomatic medicine, in Reiser MF (ed): *American Handbook of Psychiatry*, ed 2. New York, Basic Books, 1975, Vol 4, pp 477–500.

Reiser MF: *Mind, Brain, Body: Toward a Convergence of Psychoanalysis and Neurobiology.* New York, Basic Books, 1984.

Slobin DI: *Psycholinguistics.* Glenview, Illinois, Scott Foresman & Co, 1971.

Weiner H, Thaler M, Reiser MF, *et al.:* Etiology of duodenal ulcer: I. Relation of specific psychological characteristics to rate of gastric secretion (serum pepsinogen). *Psychosom Med* **19:**1–10, 1957.

Whorf BL: *Language, Thought, and Reality,* Carroll JB (ed). New York, Technology Press and Wiley, 1956.

ON MANAGING A PATIENT

The Case of the "Sick Tarzan": A Challenging Case History

The following case is presented in some detail in order that the reader may use a systems–contextual framework in analyzing the history. Rational and comprehensive management plans should emerge from the data arranged according to the Patient Evaluation Grid (PEG).

A 59-year-old married executive of a small company was admitted to the intensive care unit with nausea and severe chest pain radiating to the left arm that had developed suddenly in the morning following a hot bath. He immediately called his wife and asked her to call for an ambulance. Electrocardiograms (EKGs) and enzyme studies on admission confirmed the diagnosis of massive myocardial infarction (MI). This was the patient's first admission to a hospital, and there was no past history of serious illness. Family history was not significant except that his father had died of MI at age 72. His mother had died of pulmonary edema at age 75.

On admission to the intensive care unit, absolute bed rest was prescribed, and continuous monitoring of the EKG was instituted. Orders were written for administration of diazepam (Valium), 5 mg four times a day, and meperidine (Demerol), 50 mg every four hours for pain. Procainamide, 500 mg intramuscularly every six hours, was prescribed when he developed bursts of paroxysmal ventricular contractions (PVCs), an irregularity of the heartbeat that is common after MI and indicates hyperirritability of the heart muscle.

335

On the day of admission, he posed no problem for the doctors and nurses. His complaints were confined to the "gas pain" in his upper abdomen. On the second day, however, he was found sitting up in a chair beside the bed, despite the doctor's orders to stay in bed. He appeared agitated at night and asked for large doses of Valium to make him relax. The nurse wrote, "Patient is very arrogant but easily subdued with firmness." On the fourth day after admission, he became agitated and refused to have his blood drawn for laboratory tests. He screamed to the nurses that he wanted to sign out on the same day and was found exerting himself trying to lift up his bed. He was reproached by the nurse with no effect. He refused to cooperate in measuring his fluid intake and output and poured urine on the floor. The next day, he said to the nurse, "My doctor thinks I had a heart attack. All I need is to get back my strength, which I lost from being in here too long." He was again observed lifting up his bed. He told the nurse that he wanted to exercise by lifting up his bed 40 times a day. He was flushed and sweaty, and multiple PVCs occurred. Finally, the nurses pursuaded the cardiologist to ask for psychiatric consultation.

The following are transcribed segments of the tape-recorded interview with the psychiatrist:

DOCTOR: Could you tell me something about how you came to be in the hospital?

PATIENT: Doc, when I came here, I was sick . . . . I admit I was sick and I wanted to be helped. Now after the first stages are over, I get impatient. I can stay in bed for a certain amount of time. You know, the first day I was here, I was jumping out of the bed every two seconds—I didn't know why, but I'm accustomed to sleeping on a flat board, and the bed was at an angle and my back just broke on me. I couldn't sleep. The second night, they gave me sleeping pills so I was able to take it. Do you understand?

DOCTOR: Yes. I think so. Let's backtrack a little bit. Could you tell me what happened in the first place that brought you to the hospital?

PATIENT: You mean the heart attack? You want me to tell you something? I laid my wife that morning—let's be frank. And instead of resting a minute, I jumped into a hot tub of water, boiling hot water. I didn't give my heart a chance to rest, and it was a little too much for me. If I had rested just one minute, you know, lay alongside her and relaxed a minute, nothing woulda happened. In the beginning, I came downstairs, and I sorta felt squeamish, so I said to myself, "You know, I oughta stand in front of the open window and breathe deeply." I've got a *big* chest so I can take in a lot of air. Then, I had

a drink of pineapple juice. You know, I don't drink small amounts of anything! I guzzled that, I am telling you, and when I guzzled that, all of a sudden, she gave me a bang in the heart. And I knew right away I had a heart attack.

DOCTOR: I see. It must have been frightening to you.

PATIENT: No, I don't get frightened at nothing—nothing scares me. All I did was holler up to my wife. I've got a bull voice—you know what I mean, I can't help myself. You understand, I got a powerful chest so it comes out strong. So, she came down, and I says, "Call the doctor, tell him I had a heart attack." As I said, in the hospital, I got uncomfortable staying in bed. They had my bed raised, and I couldn't sleep. So I got up and walked around.

DOCTOR: What kinds of thoughts were you thinking as you got up and walked about?

PATIENT: You want to know something, Doc? *Nothing bothers me.* I am just telling you. I am an impatient guy. I don't let nothing bother me. I found that a long time ago, that if you don't let nothing bother you, you're the happiest guy in the world. Let them call you a dope, let them call you anything, but you got *peace of mind.*

DOCTOR: Could you tell me more about that? You learned a long time ago. . . .

PATIENT: At age 37, I had pains in my head, and I had diarrhea for years, and my family, every one of my aunts and uncles died of cancer, and I took an X-ray and I figured, "Gee, I got cancer, too." Let's be frank. I had a cousin at 31 die of cancer, too. He died of leukemia, so I figure it was me. But when they told me it was all from nerves, I says, "You see this book. It's open right now. This book is closed. If you ever see me get excited again, you can insult me from morning to night, you can call me anything you want, and I will look at you and I don't give a damn." And you want to know something—from that day on, I don't give a damn about anything. I learned one thing. If you got a calm mind, nothing is gonna hurt you. You know, a lot of doctors don't realize it.

After more discussion about past history, the doctor asks about the patient's wife.

PATIENT: My wife? In earlier years, we used to fight like hell. Now, I love her so much I could kiss her. And she knows it. And I let her do what she wants. She likes folk dancing, I let her folk dance. She wants me to stay home with her on Saturdays, I stay home with her.

DOCTOR: How has she reacted to your being in the hospital?

PATIENT: Oh, she was very shocked. The thought of me, *me* being sick. She couldn't take it, you know. I'm imperishable. That's the way she looks at me. You want to know why? Because I look at myself that way. I looked at myself as imperishable. In other words, nothing could hurt me.

DOCTOR: Could you tell me some more about your being imperishable?

PATIENT: In my youth, I was so strong that in order to get any satisfaction, I had to wrestle with four or five fellows at one time. I used to wade into a gang of fellows, 14, 15 fellows, just for the excitement of a little exercise. I had muscles like this here; I had two calves under here, like watermelons. I was what they called a natural strong man . . . but today I feel sorta weak—not full of pep, if you know what I mean.

DOCTOR: You're feeling weak?

PATIENT: I think it's because they don't let me move my blood in the hospital.

DOCTOR: Move your blood?

PATIENT: Yeah. I'll explain it another way. Do you remember, years ago, they had a couple of operations in the African veldt? Now, these operations that they had in the fields, everyone survived, yet the ones they had in the hospital, those guys died. You want to know why? Those guys had to get off the bed and pull themselves along to get home. They moved their blood. I am convinced that moving the blood is the most important thing to stay alive. Once you start laying in bed without moving and letting your blood move, you might as well bury yourself. That's how I feel. I tell you the honest truth, doctor. And, someday, you fellas are going to come to the same conclusion—that moving the blood is the most important thing of all.

The case of the sick Tarzan constituted a medical emergency. In the early phase of MI, maximal myocardial rest is essential. His insistence on lifting the bed to "move his blood" was seriously jeopardizing his life. In fact, there were increasing signs of myocardial distress, as evidenced by increasing cardiac arrhythmia. To consider rational management plans for this patient,* however, we must first consider certain important factors in the management of patients in general.

* Discussed in Chapter 21.

The Doctor–Patient Relationship

1. *"A pint of blood was extracted from his right arm, and a half-pint from his left shoulder, followed by an emetic, two physics, and an enema comprising fifteen substances; the royal head was then shaved and a blister raised; then a sneezing powder, more emetics, and bleeding, soothing potions, a plaster of pitch and pigeon dung on his feet, potions containing ten different substances, chiefly herbs, finally forty drops of extract of human skull, and the application of benzoar stone; after which his majesty died."*

The foregoing is a description of a heroic treatment of King Charles II (Van Dyke, 1947*).

2. *A 35-year-old woman suddenly developed a toxic megacolon and was admitted to the hospital on an emergency basis. She is known to have ulcerative colitis, which has been under control until now. Her physician had left the country on vacation two days prior to this acute exacerbation.*

The history of medical therapeutics until about 100–150 years ago is largely that of the doctor–patient relationship and the "placebo effect." Most ministrations by physicians would have been more harmful than beneficial to the patient, but for the natural recuperative powers of the human organism supported by the beneficial effects of the doctor–patient relationship. Four of the most famous medications used by physicians until about the 18th century were unicorn's horn, used to detect and protect against poisons in wines; benzoar stones, as antidotes for all kinds of poisons; theriac, as a universal antidote; and powdered Egyptian mummy, as a universal remedy, including wound healing (Shapiro, 1960). The unicorn's horn usually came from the ivory of the

---

* As quoted by Shapiro (1960). Reprinted with permission.

narwhal or elephant, according to Shapiro. Doctors were dangerous to patients! What is surprising, however, is that despite such noxious treatments, many patients in fact got better or even recovered completely.

The benefits of the placebo effect are psychologically determined by *expectations* and *hopes* shared by the patient and the doctor. Action, ritual, faith, and enthusiasm are important ingredients (Findley, 1953). The physiological mechanism and the pathways by which these effects are mediated are largely unknown, but at least one has been clarified in recent experiments (Levine *et al.*, 1978) that have demonstrated the physiological mechanism underlying one important type of placebo effect, that is, relief of *dental* pain by injection of a pharmacologically inactive (placebo) solution. Patients with dental pain who responded to placebo injection (placebo responders) and a matched sample of control patients with dental pain who did not (placebo nonresponders) were given placebo injections plus naloxone, a drug that blocks opiate-receptor sites in the brain. Naloxone blocked the pain relief in the placebo responders, but had no effect on the pain in control patients. These findings strongly suggest that placebo responders experience relief from pain by secreting endorphins, which are known to relieve pain by activating opiate receptor sites (see Chapter 9). Hypnotic relief of pain, on the other hand, does not work by this mechanism—it is *not* blocked by naloxone (Goldstein, 1976).

Balint (1964) calls the doctor "the most frequently prescribed drug." In this context, the doctor–patient relationship exerts major therapeutic influence. Sometimes this may be dramatically demonstrated when a patient, suddenly realizing that his trusted physician is not available, has an acute exacerbation of disease. This is especially true when the physician "covering" for the absent doctor is new to the patient and does not have an ongoing relationship with the patient (vignette 2).

The doctor–patient relationship involves, in addition to expectation of help, some specific therapeutic elements. *First,* an ongoing supportive relationship with a respected and competent professional may serve as a buffer against excessive anxiety and strain for the patient as he attempts to cope with illness. *Second,* as we will see in Chapter 19, many of the physician's activities (as in taking the medical and personal history and in performing the physical examination) may in themselves have intrinsic psychotherapeutic effects and serve to consolidate the supportive, trusting aspects of the relationship. *Third,* by providing the patient with an opportunity to share life problems, the physician may relieve the sense of helplessness and hopelessness that goes with feeling alone in the face of vexing, threatening, and sometimes overwhelming conflictual situations and feelings. *Fourth,* the physician as an objective

observer may serve as a source of information (education) and counseling that help the patient to cope with his problems. *Fifth*, we know that the patient brings to the relationship with the doctor preexisting unconscious feelings, attitudes, and expectations. The latter are reactivated feelings, attitudes, and expectations that the patient originally experienced as a child toward caretaking adults, most often parents and older members of the nuclear family, and often, too, the family physician or pediatrician who cared for the patient in childhood. The reexperiencing of these reactivated inner (memory) feelings, attitudes, and expectations *as current responses belonging to the physician* is called *transference*. The phenomenon of transference may exert a powerful influence on the patient's mental and emotional state, and the concomitant physiological events may then influence pathophysiological processes (see Chapter 4). A "positive" (i.e., trusting, loving, respectful) transference component in the doctor–patient relationship may help to modulate stress responses, induce relaxation, and permit recuperative forces to take hold, and so serve to augment beneficial effects of medications and other therapeutic measures. These phenomena will be further discussed in Chapter 19.

Of course, the physician also brings unconscious attitudes, feelings, and expectations to the relationship and should be alert to elements in his own makeup that may intrude into his relationship with particular patients.

Obviously, a good ongoing doctor–patient relationship makes it easier for the physician to recognize heterothetic help-seeking behavior and to deal with it appropriately. And the physician who knows his patient and family and takes the opportunity to stay abreast of these aspects of the patient's life on the occasion of regular physical checkups or on other occasions will be optimally situated to help his patient when trouble of any sort develops. In a good doctor–patient relationship, the patient feels that the physician will be available to answer questions, listen to life problems, and provide helpful information and counsel. Reentering a trusting relationship with the physician may re-create the physiological and psychological states associated with comfort and healing (Adler and Hammet, 1973).

Clarification and education are important aspects of the modern doctor–patient relationship. Participation in a shared belief system, by rendering anxiety-producing symptoms comprehensible, may in itself exert beneficial effects through reduction of anxiety and associated physiological processes. In the past and in other cultures, symbolic manipulation as practiced by shamans in rituals has been shown to be effective in healing certain medical conditions (Levi-Strauss, 1963). Clarification

and education allow the patient to understand and share the assumptions on which the physician bases his treatment plan. To achieve this, the physician must first understand the patient's ideas and concepts about the illness and how it may be managed. Kleinman *et al.* (1978) suggest the following line of inquiry to elicit the patient's ideas about the illness:

1. What do you think has caused your problem?
2. Why do you think it started when it did?
3. What do you think your sickness does to you?
4. How severe is your sickness?
5. What kind of treatment do you think you should receive?
6. What are the most important results you hope to receive from this treatment?
7. What are the chief problems your sickness has caused you?
8. What do you fear most about your sickness?

Once the patient's model is understood, the doctor is in a position to discuss with the patient the *discrepancies* in their respective models and to share his own understanding of the illness. Some *compromise* may be necessary if elements of the patient's model, although medically incorrect, have such preemptive importance for the patient that he cannot give them up, for example, in the case of the "sick Tarzan" (Chapter 15), the idea of "moving blood" as being so important in treatment.

The educational activities of the physician need not and should not be confined just to the illness at hand, but should include education concerning prevention of disease and hygienic principles of living and should be ongoing in nature.

The modern doctor–patient relationship is rooted in magic and primitive superstitions (e.g., of the shaman), and it exploits placebo phenomena when and as appropriate. It has developed into *much more than that, however*, and more, too, than simply the "art of medicine" as practiced by intuitive, emphathic, and humane physicians. It now also includes implicit psychotherapeutic elements and explicit specific technical procedures that can be understood as appplications of basic principles of human behavior and of mental phenomena to the practice of medicine. Properly managed, its effects can articulate synergistically with those of other therapeutic procedures, such as use of drugs and corrective surgery. The patient's expectation that the doctor will help is based, then, not only on magical beliefs but also on the scientific principles of modern medical biology and behavioral science (see Chapter 19).

Open and comfortable communication between the patient and the doctor is essential for this synergistic linkage to occur. In its absence, patients may fail to comply with the doctor's prescriptions, otherwise effective medications may turn out to be less than optimally effective, and valid surgical procedures may have unexpected adverse side effects because of the patient's anxiety and mistrust. Conversely, some patients may fall victim to scientifically unsound treatment methods (e.g., laetrile) administered by the equivalent of modern-day shamans rather than seek help from a competent physician.

Summary

Despite ineffective and often harmful methods, prescientific medicine was often effective, probably because of the effects of placebo phenomena and nonspecific supportive aspects of the doctor–patient relationship.

Current understanding of the doctor–patient relationship regards it as rooted in shamanistic magic and placebo effects, but specific psychotherapeutic elements are now included that link it effectively and understandably to the effects of conventional medical procedures. An ongoing, open channel of communication between the physician and the patient is essential for an effective doctor–patient relationship to develop. In addition, the physician must understand the patient's own ideas and models of illness and educate the patient so that he and the patient can ultimately share common belief systems and common goals for the treatment.

Implications

For the Patient

The patient's ideas about illness and the pattern of his participation in the doctor–patient relationship are based on his past experiences and cultural expectations. *Discrepancy* between the patient's and the doctor's models of illness may interfere with development of an optimal doctor–patient relationship because a shared belief system—an important ingredient of a good doctor–patient relationship—does not exist. A patient's *lack of compliance or cooperation* with the medical regimen may be a sign of a poor doctor–patient relationship or deterioration of a previously good one.

For the Physician

An understanding of the patient's ideas and models about illness and health care is essential for an optimal doctor–patient relationship. *Specific questions* are necessary to elicit this information. The patient's cultural background and his family's attitude toward illness are important data in this regard. The physician may judiciously utilize the powerful doctor–patient relationship as a *therapeutic tool* and should pay attention to it in his work with patients.

For the Community and the Health-Care System

Education of the general public concerning illness and health care is important, since shared expectations play an important role in fostering good doctor–patient relationships. Medical education should inform students about the therapeutic significance and use of the doctor–patient relationship in addition to the technical aspects of treatment in the practice of scientific and effective medicine.

Recommended Reading

Balint M: *The Doctor, His Patient, and the Illness*. New York, International Universities Press, 1964. This is a classic, describing the discussions between Dr. Balint, a psychoanalytically oriented psychiatrist, and a number of general practitioners in England about their patients. A good discussion on the relationship between the doctor and the patient and also on psychological understanding of patients. Has interesting vignettes and case histories.

Kleinman A, Eisenberg L, Good B: Culture, illness, and care: Clinical lessons from anthropologic and cross-cultural research. *Ann Intern Med* **88:**251–258, 1978. A good discussion on the cultural patterning of sickness and care, with emphasis on understanding the patient's models concerning illness.

Levi-Strauss C: *Structural Anthropology*. New York, Basic Books, 1963. A classic. Contains very interesting and vivid descriptions of symbolic manipulation of shamans. Fascinating reading.

References

Adler HM, Hammet VBO: The doctor–patient relationship revisited: An analysis of the placebo effect. *Ann Intern Med* **78:**595–598, 1973.

Balint M: *The Doctor, His Patient, and the Illness*. New York, International Universities Press, 1964.

Findley T: The placebo and the physician. *Med Clin North Am* **37:**1821–1826, 1953.

Goldstein A: Opioid peptides (endorphins) in pituitary and brain. *Science* **193:**1081–1086, 1976.

Kleinman A, Eisenberg L, Good B: Culture, illness, and care: Clinical lessons from anthropologic and cross-cultural research. *Ann Intern Med* **88:**251–258, 1978.

Levine JD, Gordon NC, Fields HL: The mechanism of placebo analgesia. *Lancet* **2:**654–657, 1978.

Levi-Strauss C: *Structural Anthropology*. New York, Basic Books, 1963.

Shapiro AK: A contribution to a history of the placebo effect. *Behav Sci* **5:**109–135, 1960.

Van Dyke HB: The weapons of panacea. *Sci Mon* 64–322, 1947.

CHAPTER 17

The Patient's Personality

1. A 67-year-old man was admitted to the hospital for evaluation of fainting episodes. On admission, he started complaining about the "sloppy service," wanted to have food specially brought in from a restaurant every day, and called the office of the president of the hospital to complain about the delay in the nurses' responding to his calls. History revealed that he was a self-made man who was still active as the chairman of the board of a large corporation. Soon the patient was considered by the doctors and nurses to be a "pain in the neck" and a "crank," and they tried to avoid dealing with him as much as possible. This resulted in further delays in medications and responses to his calls.

2. The doctor asked, "Do you have any pains in your chest?" The patient replied, "Yes, doctor, I have so much pain in my chest—like constant pressure and pins and needles, and then I have these terrible aches and pains in my stomach, I have indigestion and constipation, and pains in my spine. My head hurts, too, and all my joints."

"It sounds like you are suffering a lot."

"I suffer all the time, what with my pain and my luck . . . you know, when I had my operation for my back, the wound would not heal, then I had an abscess which was treated with penicillin—then I had this reaction to penicillin so they had to stop it. My husband left me while I was still in bed from the operation, and the house burned down. That's when my arthritis flared up, and my headaches started. My old doctor gave me aspirin for it, which caused my stomach ulcers . . . when they started bleeding and I had to have an emergency operation. . . . "

347

Personality and Character

How an individual reacts to illness, and the willingness with which he assumes the sick role, are determined in large measure by his personality. *Personality* is a broad general term that refers to a person's *characteristic patterns of thought, behavior, and feelings*. It includes, among other things, cognitive style, temperament, tolerance for anxiety, and patterns of defense and coping. In its broadest sense, personality includes the concepts of character and "neurotic styles," the latter referring to habitual symptomatic ways in which a person perceives and thinks (Shapiro, 1965). The term "character" is often incorrectly used as a synonym for personality.

Character was used in a special sense by Wilhelm Reich (1949), who regarded it as a "defensive armour," an aggregate of characteristic psychological defenses (see Chapter 5) utilized so often by the person that it functioned as a "structure" always in a state of readiness, in preparation to react to anxiety-provoking situations. Seen in this special light, character has a primarily defensive function and can be regarded as one component of personality.

Concept of Personality Types in Patients

Although each person has his or her own unique personality, it is sometimes useful in the context of medical care to classify patients according to arbitrary but loosely defined "personality types." Identification of the personality type or style of a patient does not necessarily imply a need for changing it through treatment. Its value lies rather in the fact that assessment of the personality type helps us to understand the meaning of illness for the given patient and, in turn, its influence on sick-role performance. This understanding can then provide the rationale for developing optimal ways of interacting with the patient. Without it, an impassse can develop between a "difficult" patient and health-care personnel.

Habitual ways (e.g., neurotic styles) of dealing with stressful situations often become exaggerated when a person is ill, particularly when he finds himself in the strange environment of the hospital (see Chapter 18). Thus, a person who has a tendency to be precise and controlling may become even more precise and controlling in the hospital, even to the extent that his personality may come into conflict with the sick-role expectation of allowing himself to become dependent on the hospital personnel.

The meanings of illness, of hospitalization, and of medical procedures to a patient are congruent with his personality type, and the ways in which patients and physicians relate to one another are influenced by the interaction of their personalities. Kahana and Bibring (1964) described seven personality types often encountered in the general hospital and discussed how illnesses might have differential meaning according to differences in personality styles and how different management approaches might be used depending on the personality style of the patient. The nine personality types we present here are modifications and expansions based on their work.

Personality Types and the Sick Role

Patients may be categorized by personality type as follows*:
1. Dependent, demanding patients
2. Orderly, controlling (obsessive–compulsive) patients
3. Dramatizing, emotional (histrionic) patients
4. Long-suffering, self-sacrificing (masochistic) patients
5. Guarded, suspicious (paranoid) patients
6. Superior and special (narcissistic) patients
7. Seclusive, aloof (schizoid) patients
8. Impulsive patients with a tendency to act out
9. Patients with mood swings (cyclothymic)

Dependent, Demanding Patients

It is said that one can detect this type of personality by noting the amount of luggage the patient brings to the hospital. An exaggerated caricature form of this personality is indeed seen in the patient who comes into the hospital as though he were prepared to stay for months, if not years. Patients of this type have a need for a great deal of reassurance and often want special attention from health-care personnel. They tend to become dependent on the doctor and others who are involved in their care and often make frequent, inappropriately urgent calls to nurses and doctors. When their (excessive) demands are not met fully, they tend to feel angry and rejected.

---

* The terms in parantheses are "psychiatric" terms denoting extreme forms of the personality characteristics (patterns).

The underlying dynamic for this type of personality is considered to be a regressive wish to be cared for as though by an idealized, nurturant mother. The fear of being rejected, left out in the cold, and neglected tends to exaggerate the *need for reassurance and care.* The *sick role* may be considered to be a temptation for these patients to return to a state of infantile dependency, and they may consider the *illness* to be a result of a lack of protection and concern by others. This *regressive, dependent behavior* is congruent with some aspects of Parsons's description of the sick role except for the third and fourth elements, that is, considering the sick role as an undesirable state of being and cooperating with the doctor in becoming well again (Chapter 2).

The incessant demands of a patient of this type, coupled with relative comfort in the dependent position, may be regarded by others, especially doctors and nurses, as "enjoying" being sick. Hostile behavior, when excessive demands for attention are not met, provokes anger and conflict. The nurses, for example, may feel that the patient wants too much attention, while the patient feels that the nurses are cold and uncaring.

There is an opposite side to this coin as well. A patient of this type was referred to the psychiatrist by an alarmed surgeon because he too eagerly consented to an amputation the first time it was discussed as a possibility. In this instance, it was learned that the doctor had been overly indulgent with the patient, allowing special privileges and giving an inordinate amount of care and attention. The patient, before long, regarded the doctor as an omnipotent, mothering figure and wanted to go along with anything that the doctor suggested might be good for him.

Orderly, Controlling Patients

Such patients tend *not* to show feelings and generally experience illness without outward signals of emotional reaction. Their descriptions of symptoms are complete, precise, and dispassionate. [Isolation is often used as a defense mechanism by these patients (see Chapter 5).]

This personality style is motivated by a *desire to control* external as well as internal states. Behind the desire to control may be *fear of loss of control—of being helpless.*

The *sick role* is obviously a difficult pill for patients with these personality characteristics to swallow. Removal from normal responsibilities and daily routine may be experienced as disruptive. Being unable or not permitted to "help themselves" may be an alien experience for them.

Needing to seek advice and help from a professional may generate concerns about who will control whom, and they may feel deeply threatened by the control that doctors and nurses must assume over their lives and bodies in order to administer necessary medical care.

In response to these threats, they may become contentious, complaining, and accusatory. Usually quite conscious of time and details, for example, medication schedules, they may become incensed and critical if the nurse brings a pill a few minutes late.

Such patients do *not* respond favorably to blanket reassurances. They are likely to wonder if the physician is competent when reassurances are given without firm foundation in facts. The doctor's explanation of one hopeful laboratory finding may be far more reassuring to this type of patient than many impressionistic but unsupported optimistic statements.

A rule of thumb in dealing with this type of personality is to attempt to recruit the patient to be a part of a therapeutic team effort against the illness. This enables him to feel that the physician respects his autonomy enough to ask him to cooperate in the common endeavor. Detailed explanations of the diagnosis, the physical and laboratory findings, and treatment plans are helpful, especially for more educated patients. Sometimes it is useful to the patients to help the treatment team by keeping a diary of symptoms or by recording some of their clinical data, such as volume of water drunk and urine voided.

> A chemistry technician with diabetes mellitus was admitted for treatment of leg ulcers. Within days after admission, he complained of the "sloppiness" of the doctors and nurses, their lack of punctuality in bringing him medications, etc. Successful management involved the physician's acknowledging the patient as someone related to the medical profession. ("As a chemist, you would understand the mechanism of diabetes mellitus. Now, we want to treat this with diet and insulin, and we will follow the course with urine tests for sugar. It means . . . "etc.) In addition to giving the patient credit for his knowledge of chemistry, the doctor taught him to change his own medicated dressings (he could do it "much better than any nurse") and to keep track of his medications to be sure that they were taken on time.

Dramatizing, Emotional Patients

Patients with this personality type tend to come across as being rather charming and fun to talk with. They have a certain dramatic flair

when giving accounts of their lives and are often quite amusing. Their histories tend to be more *impressionistic* and diffuse than precise. They may be overtly *seductive*: female patients wearing provocative negligees and "parading around" in the hospital; male patients making sexually seductive comments to nurses and female physicians. There is a tendency for these patients to consider their relationship with the doctor as special, with sexual overtones. The medical staff often finds itself split around these patients, some liking them very much and others feeling angry with them. The patients themselves have usually unwittingly provoked these split reactions.

A major concern underlying such behavior is the need to be *attractive and desirable* to others, to prove their *"masculinity"* or *"femininity"* over and over again and to gain care and support. An *underlying fear* that they might not be found attractive and desirable is, of course, accentuated by illness, with its threat to the integrity of the body. As patients, persons of this type have an exaggerated need to be reassured that they are still attractive and will not be deserted.

The *sick role* may or may not be compatible with this type of personality. On the one hand, the dependency and social perquisites inherent in the sick role afford some of these patients an acceptable opportunity to exhibit and "flirt" with authority figures in a situation that sets limits. Patients with extreme forms of this personality, despite their overtly sexually provocative behavior, tend to be rather inhibited in actual sexual encounters. For them, the hospital and medical treatment may be exactly the type of setting they find most comfortable for seductive behavior without danger of actual sexual activity. On the other hand, some patients become extremely frustrated by the confinement and limitations of the sick role, especially if they had been accustomed to active, exhibitionistic, and gratifying life-styles. For example, a man who had been accustomed to a "Don Juan" life-style may find the restriction of sexual activity in the hospital most unbearable.

These patients respond best if the doctor responds, within set boundaries and limits, a certain amount to their needs to engage him. However, this should not be overdone, since these patients also tend to be frightened if their characterological seductiveness seems to lead to unexpected intimacy. Showing some *warmth and personal concern* is usually all that is needed. When there seems to be a split in staff feelings, these should be openly discussed and resolved in staff meetings. It may also be necessary to set *firm limits* with the patient, at the same time indicating concern and willingness to continue to take care of the patient. *Repeated reassurances* are often necessary. With this group of patients, unlike the orderly, controlling personalities, the doctor's personal man-

ner and attitudes are relatively more important in providing reassurance than factual content, that is, discussion of objective findings and test results.

Long-Suffering, Self-Sacrificing Patients

Some experienced physicians say that this personality type can be diagnosed by the pitch and tone of the patient's first utterance in the doctor's office. Such patients often speak in a wailing, complaining voice, and usually the history involves a long list of *hard luck and disasters*: surgical operations followed by complications, trusted persons turning out to be untrustworthy, promised cures for a symptom bringing on more symptoms and side effects than relief, and other complaints. They almost always have endured protracted pain and suffering, and this "present illness" represents an additonal suffering for a patient who seems to have been "born to suffer" (vignette 2).

When listening to patients with this type of personality, one usually finds that they have taken care of someone else despite their own suffering and misery. They take much pride in relating how this feat was achieved in the presence of so much suffering and so many misfortunes. Often, that someone else is a child, a spouse, or a parent.

A major underlying dynamic in these patients is considered to involve strong feelings of guilt that do not allow them to enjoy life for themselves. With a "need" to suffer in order to expiate the guilt feelings, *altruistic activities* (such as caring for others) in the presence of physical or emotional pain may allow them some covert gratification (claim to happiness). Thus, these patients appear as though they are "exhibiting" their misfortunes, sufferings, and altruistic acts.

Another underlying dynamic in such patients is the use of *pain and suffering as a life-style, as a means of maintaining interpersonal relationships*. These are patients who might be *"addicted" to the sick role*. The sick role is taken on from time to time throughout their lifetimes, although they also feel proud of having taken care of others despite the sick-role restrictions. A closer scrutiny reveals that the sick role is assumed as a way of meeting their needs indirectly through suffering and through ongoing contact with the physician. Many patients diagnosed as "hypochondriacs" have this personality type.

Patients with this type of personality often become severe problems for health-care personnel. Typically, they tend to *react negatively to reassurances*, totally frustrating the doctors. When the physician prescribes a medication with reassurance, for example, that it should relieve pain,

the patient is likely to return complaining of more rather than less pain, which may now be felt in areas that were previously free of pain! In addition, he may have nausea and dizziness. He may even overtly blame the physician for the added troubles, but most often this is attributed to bad luck. The physician, nevertheless, is often *made to feel guilty* by these patients. This frequently results in a *rejection* of the patient by the physician, which adds to the patient's feeling of being mistreated. Thus, these patients commonly have a history of repeated rejection or transfer from doctor to doctor. The long-suffering, self-sacrificing patients, although asserting belief in Parsons's third and fourth sick-role expectations, actually refute them in their behavior. Those who become addicted to the sick role fail to fulfill the sick-role expectations of "considering the ill state as being undesirable" and "attempting to get well as soon as possible by cooperating with the physician."

Patients with this personality type are best managed when the physician gives *"credit"* to their suffering and expresses appreciation for their courage and perseverance in the face of protracted pain and hardship. It is a mistake to promise such patients complete relief from pain and suffering. In fact, since they need to expiate guilt and maintain relationships, such a promise may provide a powerful reason for the patients' "refusal to improve." Taking away the symptoms and suffering would leave them exposed and helpless, without any means of relating to others.

Recognition of this pattern will also help the physician to recognize the necessity to accept and set limited goals for the treatment in order to avoid later frustration, feelings of helplessness, and reactive anger. This can prevent or postpone the development of disruptive tension in the relationship with the patient. It is often helpful for the physician to approach this type of patient with some degree of pessimism, such as, "Although we cannot take away the pain completely, this medication may take the edge off the pain somewhat."

Attempts to mobilize altruistic tendencies may also be helpful. For example, a female patient may be persuaded to seek proper treatment to alleviate crippling pain so that she might be better able to care for her children.

One has to differentiate this type of personality from patients who experience protracted suffering due to actual complications from treatment. Patients suffering from chronic illnesses without this character style do not show the self-sacrificing element, and although they may feel rather cynical about the prolonged illness, they do not show the tendency to "refuse to improve."

Guarded, Suspicious Patients

Patients with this personality type are always *watchful* and concerned about the possibility that harm might be done to them, intentionally or unintentionally. They are quite fearful of being exploited or taken advantage of. They are quite sensitive to the possibility of criticism. They are prone to wonder about ulterior motives or any suggestions or remarks made by the health-care personnel, especially if they are ambiguous. These patients are also likely to misinterpret statements and actions and read something ominous or threatening into them. This is especially true in the presence of great anxiety, as in being hospitalized, and in states of reduced cerebral function that impair the integration of sensory input.

Such patients also tend to *blame others for their illness.* For example, a patient may claim that he developed a heart attack because the employer did not provide air conditioning for his work area and "poisoned the air" with carbon dioxide exhaled by so many others.

These patients, obviously, do not enjoy being in the *sick role.* The dependency on the health-care personnel increases their feelings of vulnerability, and with that comes the fear that persons in positions will do things to them that might take advantage of them. Although they see the ill state as an undesirable one, they cannot *trust* the physician enough to cooperate fully.

A good strategy for *management* of these patients is to assume a relatively neutral attitude concerning their suspicions, criticisms, and other manifestations of hostility without becoming provoked by them or arguing with them. A helpful statement is, "I understand how you feel under the circumstances." Identifying their suspiciousness as "sensitivity" is also helpful, since patients like to feel that they are not "fools." Occasionally, agreeing with the patients about the inconveniences about which they are suspicious and then putting the blame on impersonal things like hospital regulations can diffuse their feelings of anger from being directed toward the health-care personnel.

Above all, it is important to provide as little cause for suspicion as possible. This involves *consistency* on the part of health-care personnel in terms of information imparted. It is also necessary to explain, in as much detail as feasible, the nature of the patient's illness and plans for treatment. This will tend not only to minimize the suspiciousness but also to reduce the likelihood of litigation in case of complications, since this type of patient is likely to be litigious as well. When a procedure is recommended to the patient, it is best to present it as objectively as

possible, so as not to arouse the suspicion that the doctor is trying to "manipulate" the patient for ulterior motives.

Superior and Special Patients

These are patients who behave like VIPs, whether or not they are. Such patients have a tendency to appear snobbish, self-confident, and sometimes grandiose (vignette 1). They are often quite *proud* of their bodies and their physical abilities. This basic style might be partially covered up by exaggerated, artificial humility. There is a sense of arrogance and disdain when they are in contact with other people. Though these patients may seek the most prestigious medical centers and the most eminent physicians when ill, there is often an air of tentativeness when the physician explains anything to them. They may display an arrogant attitude, especially toward persons on the lower strata of the hospital hierarchy, such as house officers, student clerks, and nurse's aides. They are likely to threaten to notify the chief of service or the director of the hospital of any inconveniences they suffer. They also use "name-dropping" to try to impress health-care personnel.

Many patients with this personality style have *idealized body images,* and *illness* represents a threat to the maintenance of this body image. Many neurotic patients with overvaluation of physical prowess, stamina, and fitness were found to have developed the neurosis after illness or injury, often of a minor nature [the "athlete's neurosis" (Little, 1969)].

The patients with superiority feelings naturally do not find the *sick role* agreeable. Their need to see themselves as being perfect and invulnerable is contradictory to the notion that they "cannot help themselves" and are in need of more competent help. Although they may submit to this unpleasant situation, they often attempt to find weaknesses and faults in the physicians, as though to cut them down to size in order to still feel superior to them.

Needless to say, health-care personnel often resent this type of attitude. The result is often a battle between the caretaking persons and the patient, each attempting to cut the other down!

Successful *management* of these patients involves a certain degree of magnanimity on the part of health-care personnel, allowing the patient to boast of his strengths. When this is done, the patient may feel secure enough to identify the caring persons with himself—as being almost perfect. It is, however, a mistake to be unnecessarily humble in relation to these patients. An attitude of security about one's profes-

sional competence, while recognizing the worth of the patient, is important to ward off insecure feelings on the patient's part that he might not be in the best hands, after all.

Seclusive, Aloof (Schizoid) Patients

This is the type of patient who seems to be remote, detached, and not in need of any interpersonal contact. They usually prefer to be in private rooms and seldom speak or relate to other patients or staff. They like to be involved in solitary activities, such as reading or listening to music. They appear shy and uninvolved. Nurses are sometimes so disturbed by the aloofness and lack of personal response that they suspect depression and bring the patient to the attention of the physician. Some patients with this personality might also appear to be eccentric, with affinities for activities associated with the counter-cultures, such as health foods and quasi-religious sects.

The main concern of these patients is a *desire not to be intruded on by others;* they wish to maintain a sense of tranquility by being absorbed in themselves and things familiar to them. Any attempt at socialization by others may be seen as an intrusion threatening their fragile tranquility.

Illness is seen as a threat to this self-absorption and tranquility. These patients therefore have difficulty in adjusting to the *sick role*, with its expectation of dependency on and cooperation with health-care personnel. The patients come to terms with the role expectations through *noninvolvement* at a personal level while allowing the medical process to go on. Thus, a patient with this personality may appear to be strikingly unconcerned about illnesses and procedures that would normally be expected to arouse much anxiety. Of course, many patients with this personality *delay seeking help* because of their aversion to the intrusion into their privacy that is necessary in receiving medical care. On the other hand, some patients with this personality may use the sick role as an excuse to find interpersonal relationships without true intimacy.

In *managing* such patients, it is important to recognize and respect the need for privacy. Although socialization and sharing are important to most people, these patients need to protect their privacy and tranquility. Some of these patients, however, may be able to form some relationship with one or two members of the hospital staff. These members can then serve as "translators" for these aloof patients.

Impulsive Patients with a Tendency to Act Out

These are the patients who keep on doing things they did not "mean" to do, usually on the basis of some impulse. These patients may appear to be rational and well controlled, until an impulsive action occurs. Usually, however, they have a history of being involved in interpersonal or legal difficulties because of some maladaptive acting-out behavior. The characteristic feature is a *lack of deliberation,* with decisions being reached on the spur of the moment. Patients with this character style seem to lack tolerance for sustained thinking and for frustration. They often say that they acted "without thinking" or "could not help" what they did and often are quite remorseful after the action. In the health-care system, these impulsive actions usually involve some aggressive acts against health-care personnel or ill-advised decisions such as *signing out against medical advice* despite having a serious illness.

These patients seem to feel an overwhelming sense of impotence in the presence of relatively minor frustrations and appear to be *unable to delay gratification* or to feel gratified by anticipatory cognitive processes such as planning.

The patient with an impulsive personality style is likely to seek help for relatively minor symptoms based on the immediate pain or discomfort experienced, and he is likely to demand *immediate relief* from the discomfort. If immediate relief is not produced, the person is prone to act out by such aggressive acts as cursing at the physician or kicking an article of equipment in the treatment room. Such patients, although wanting immediate relief from symptoms, often have difficulty in tolerating the treatment process, especially when it also involves some discomfort, such as swallowing a gastric tube. Although the patient may appear to have understood the necessity of such a procedure, he is as likely to curse and attempt to sign out in the midst of the procedure when discomfort occurs. Thus, cooperation with the physician (a *sick-role* expectation) is a difficult achievement for these patients.

Medical professionals, trained to be always deliberate and objective, tend to dislike patients with this personality type. They see these patients as being defective and childish. In fact, this style may be a manifestation of a *defect in the integrative functions of the brain* rather than a primarily developmental personality style. It is important, therefore, for health-care personnel to deal with it as a defect, just as they have to recognize and deal with a diabetic patient's metabolic defect. The *management* strategy, thus, would involve preventing situations in which the defect would be of major consequence and making compensations for it when it is avoidable.

For example, tranquilizers may be utilized more freely for these patients as a partial preventive measure against outbursts of aggression. Pain should be treated especially vigorously. Firm limit-setting is also necessary to establish some external control over their acting-out behavior. In fact, these patients feel reassured by firm limit-setting, which also gives them a sense of external control and caring. Whenever possible, persons familiar to the patient, such as friends and relatives, should be mobilized to support and control the patient.

Patients with Mood Swings

These are patients who characteristically have "ups and downs," that is, periods of relative euphoria and hyperactivity followed by periods of depressed feelings and lack of energy. Although most people have some periods characterized by euphoric or depressive moods, persons who have this personality trait exhibit such mood swings consistently. During the "up" periods, they feel optimistic, ambitious, and usually physically well. During the "down" periods, feelings of pessimism and a sense of malaise predominate. If these changes are exaggerated so as to cause major problems in function, the psychiatric diagnosis of bipolar depressive disorder may be made (see Chapter 6).

The importance in recognizing this personality trait lies in that depending on the period in which the person finds himself, the reaction to illness and to the medical treatment may be different. When an illness occurs during an up period, the patient may not even recognize the presence of the symptoms, or even if he recognizes them, he may brush them aside as being of no consequence. If he happens to be in a down phase, however, he may feel quite pessimistic about the symptoms and attach all kinds of grave implications to them. In fact, he may be convinced that he has a terminal illness for which there is no hope by the time he sees the physician. In addition, because of the feelings of malaise and lack of energy experienced during the down phase, these patients may experience exaggerated discomfort that may be caused by minor dysfunctions.

Patients with this personality trait might be more prone to develop severe depression in the presence of major stress such as a serious medical illness. If a patient who has this pattern develops evidence of serious depression, including feelings of hopelessness, guilt, and lowered self-esteem, coupled with weight loss, anorexia, sleep disturbances, and, perhaps, suicidal thoughts, referral to a psychiatrist should be made for

definitive treatment. This is also true for any other patients who show similar evidence of depression (see chapter 6).

From Types to Individuals

As should be clear from our discussion so far, the various characteristics of personality types are not mutually exclusive but tend to coexist in varying combinations. One of our most gratifying experiences is to hear our students complain to us, after a discussion of personality types, that they could not actually categorize a single living patient neatly into any single type. The personality types described here are like caricatures. In real life, it is the rule rather than the exception to see patients with characteristics belonging to several personality types. For example, one patient may be orderly and controlling *and* guarded and suspicious, or another may be dependent and demanding *and* also have mood swings. Once an individual is recognized as being unique, with certain characteristics from several different personality types, then the management of such a patient can be truly individualized.

Summary

How an individual reacts to illness, and the willingness with which he assumes the sick role, are determined by his personality. Nine personality types often encountered in medical settings are discussed: (1) dependent, demanding; (2) orderly, controlling; (3) dramatizing, emotional; (4) long-suffering, self-sacrificing; (5) guarded, suspicious; (6) superior and special; (7) seclusive, aloof; (8) impulsive; and (9) those with mood swings. *Illness* and *hospitalization* tend to have different *meanings* depending on the patient's personality type.

Management strategy should take into account the patient's personality since an effective approach with one type of patient may induce noncooperation in another type. For example, detailed explanation of contemplated procedures may win the confidence and cooperation of an orderly, controlling type of patient but may frighten a patient who has the dramatizing, emotional personality. Because of the heightened anxiety, such a patient may not be able to understand the explanation, and a general reassuring attitude with a general, undetailed explanation may be more helpful for him. An orderly, controlling person often has difficulty in accepting the *sick-role* expectations of dependency, while a

long-suffering, self-sacrificing type of person may be "addicted" to certain aspects of the sick role.

Although an understanding of the prominent features of a patient's personality can help health-care personnel in planning effective approaches, most patients do not fall into one or the other personality "type." Familiarity with the commonly encountered personality types and the meaning of illness to them should help health-care personnel *individualize* their approach to individual patients.

Implications

For the Patient

Illness interacts with the individual's personality system and determines his help-seeking behavior (including whether or not he will seek medical advice), adaptation to the expectations of being a patient (sick role), and performance as a patient. Depending on the personality characteristics, *illness, hospitalization, and medical care have different meanings to patients.* A patient is usually unaware of his personality characteristics and of the relationship of these characteristics to his feelings about being a patient. Thus, any conflict or contradiction arising between the personality needs and the sick-role expectations has a tendency to be seen by both the patient and the physician as a specific conflict arising from specific situations; that is, the patients have a tendency to blame a specific doctor or nurse for not understanding them or not caring for them (or for being incompetent, neglecting, or otherwise inadequate).

For the Physician

Understanding the patient's personality style can help the physician formulate an *optimal approach* to the patient. Physicians should also be aware that their own personalities influence how they treat their patients, and a particular mix of the physician's and patient's personalities may bring about a *conflict* in the medical setting. For example, an orderly, controlling type of physician may find himself feeling challenged and unrespected while treating a patient who also has an extremely orderly, controlling personality. Taking some distance from the situation emotionally when such conflicts arise and understanding the personality needs of patients can help the physician avoid becoming entangled in unnecessary personality conflicts (e.g., "power struggle") and meet pa-

tients' needs. Physicians should also be aware that conflicts arising between patients and other health-care personnel may reflect a lack of understanding on the part of the health-care personnel about the personality needs of the patients. The physician can then educate others in how to approach patients while taking into account their personality characteristics.

For the Community and the Health-Care System

Training for physicians and health-care personnel should include the skills for assessing patients' personality types and their interaction with illness and sick-role behavior. The health-care system should recognize that a patient brings into the medical setting an exaggeration of his premorbid personality characteristics in addition to being a person defined by the presence of the particular illness that made him a patient.

For preventive medicine to be effective, methods should be devised to induce persons with personality types that tend to avoid assuming the sick role to seek help when needed. Health-care personnel should be educated to recognize those patients whose main motivation for being patients is perpetuation of certain aspects of the sick role. The treatment for them can then be made more effective by avoiding the pitfalls of premature removal of the legitimacy of the sick role or unnecessary laboratory tests or surgical procedures.

Recommended Reading

Goldberg RJ: Personality types and personality disorders, in Leigh H (ed): *Psychiatry in the Practice of Medicine.* Menlo Park, California, Addison Wesley Publishing Co, 1983, pp 33–56. This article complements this chapter in extending the discussion of personality types to personality disorders, including a discussion of the borderline personality disorder.

Kahana R, Bibring G: Personality types in medical management, in Zinberg N (ed): *Psychiatry and Medical Practice in a General Hospital.* New York, International Universities Press, 1964, pp 108–123. This is the classic description of the personality types often seen in medical settings. The reader will find somewhat more detailed discussion of the psychodynamics and possible evolution of the personality types, as well as management approaches with examples.

Shapiro D: *Neurotic Styles.* New York, Basic Books, 1965. (Also available in paperback.) The author describes the cognitive characteristics of the major "neurotic" (or "personality," in less exaggerated form) styles, such as hysterical, obsessive–compulsive, and paranoid styles. An important book for understanding the influence of personality styles on how individuals perceive the world and how they think.

References

Kahana R, Bibring G: Personality types in medical management, in Zinberg N (ed): *Psychiatry and Medical Practice in a General Hospital.* New York, International Universities Press, 1964, pp 108–123.
Little CJ: The athlete's neurosis: A deprivation crisis. *Acta Psychiatr Scand* **45:**197, 1969.
Reich W: *Character Analysis.* New York, Orgone Institute Press, 1949.
Shapiro D: *Neurotic Styles.* New York, Basic Books, 1965.

The Hospitalized Patient

1. *A 42-year-old married woman was admitted to the hospital for an elective gallbladder operation. Her attending physician greeted her on her arrival at the hospital, and she was assigned a private room and a private-duty nurse. The hospitalization was not going to be as bad as she thought; the large donation her husband had made to the hospital last year had been very well advised.*

2. *The nurse phoned the intern on call at 3:00 A.M.*

Nurse: Doctor X, I am sorry to call you at this time, but Mrs. Smith's i.v. infiltrated, and she needs her i.v. antibiotic. . . .

Intern: Oh, hell! Why don't you go ahead and restart the i.v.? I know that you can do it as well as I.

Nurse: I am sorry, Dr. X. I'm not allowed to restart an i.v. unless I am in the ICU.

Intern: Come on! I saw you starting an i.v. on Mr. Jones only this morning.

Nurse: Yes, but that was in the ICU. Remember, I sometimes work two shifts. I will get into trouble if I start an i.v. on a regular ward. Rules are rules.

Intern: Oh, OK. I am wide awake now anyway. There should be a hospital rule that says i.v.'s are not allowed to infiltrate after midnight!

3. *During the first few days of hospitalization in a typical teaching hospital, a typical patient with low back pain has the privilege of meeting the following persons:*

> 1. *Receptionist and admitting office personnel*
> 2. *Ward secretary*

3. *Head nurse and one or two additional nurses (take vital signs)*
4. *Intern (does a history and physical exam)*
5. *Medical student doing clerkship (may be more than one) (history and physical exam)*
6. *Resident (history and physical exam)*
7. *Attending physician (brief history and physical exam)*
8. *Dietician*
9. *Maid*
10. *Nurse's aide*
11. *Lab technician—blood team*
12. *Radiologist and technician when X-rays (at least routine, probably more) are taken*
13. *Consultants: Usually, at least one or two consultations are given each patient. For example, in this case, the consultation may be with an orthopedic or neurosurgeon, with a psychiatrist if the patient seems to be anxious, and, finally, with an anesthesiologist if an operation is being considered.*
14. *Possibly a social worker or the clergy*
15. *Other patients on the same ward*

During his hospitalization, the patient may have to make the acquaintance of a whole new set of medical students, interns, residents, and even attendings as they rotate off the particular service. The same is true of the consulting teams, which, in turn, consist of residents, attendings, and students.

The hospital is a microcosm with which most of us have intimate contact. Most of us were born in a hospital, have some contact with one during our lifetime (if we do not actually work in one), and usually can expect to die in a hospital. Hospitals are seen by society in many different ways—as a necessity, a frightening place, a source of community pride or shame, an important industry, an employer, and in other ways.

Many of those who are ill either are already in the hospital or will be admitted to one sooner or later. We will consider the hospital as a social system and then the environment of the hospital as it pertains to the patient.

The Hospital as a Social System

Kinds of Hospitals

Hospitals can be classified on the basis of the length of stay of patients, the kinds of services provided, and the type of management.
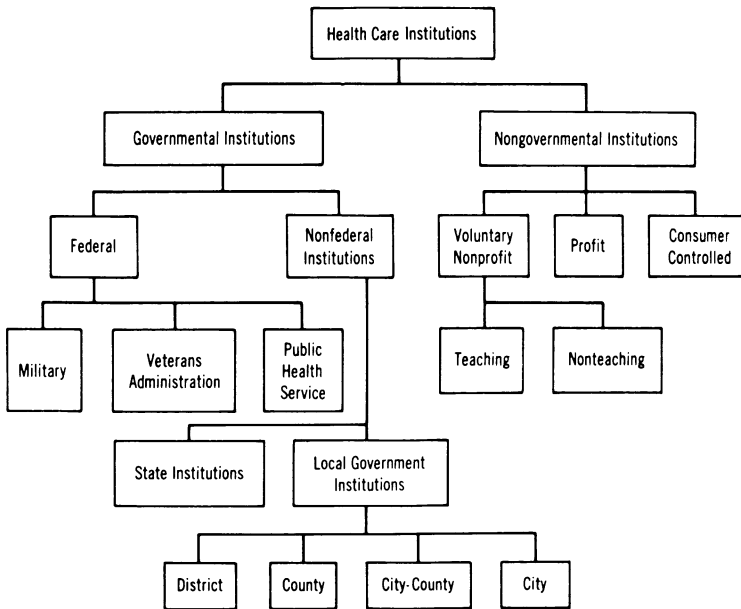
Figure 31. Classification of health-care institutions by control. (From Freeman *et al.*, 1972. Copyright 1972 by Prentice-Hall. Reproduced with permission.)

Thus, we can consider short-term vs. long-term, general vs. special, and community vs. noncommunity hospitals. The most common type of hospital in the United States is the short-term general hospital (see Figures 31 and 32).

The People in a Hospital

The hospital is an open system and is influenced by the culture of the greater society and community of which it is a part. The social status of the people in the hospital, to a large extent, reflects that of the larger society, although there is also a hierarchical social structure within the hospital. This is so because, to a great degree, the hierarchy in the hospital is parallel to that of the larger society. Those high up in the hospital hierarchy are also likely to have high status outside.

One possible exception here is the patient. In terms of the hospital hierarchy, the patients find themselves at the bottom rung—they are the least familiar with the ethos and culture of the hospital. And, as
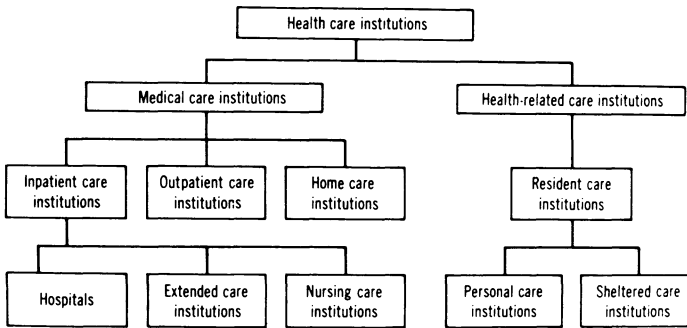
Figure 32. Classification of health-care insitutions by function. (From Freeman *et al.*, 1972. Copyright 1972 by Prentice-Hall. Reproduced with permission.)

transients, they are more likely to "put up with" inconveniences and occasional mistreatments than to make an issue of them. But here, again, the social status of the patient in the larger community plays a role. The VIPs are more likely to insist on, and receive, better personalized service than the "charity patients." Patients with high social status, patients who have professional ties to the health community, and those who had families who were willing and able to intercede for them with the health professionals have been shown to receive the kind of care that meets their expressed needs (Roth and Eddy, 1967; Scott, 1969) (vignette 1). The differentiation between the charity patient and the paying private patient is rapidly disappearing, thanks largely to the widespread family insurance coverage of the working population. Psychiatric patients, however, still tend to receive different diagnoses and treatments and assignments to different types of hospitals (state hospital vs. private hospital) depending on social class (Hollingshead and Redlich, 1958; Myers and Bean, 1968). Psychiatric patients from the higher classes have lower hospitalization rates and, once hospitalized, tend to be discharged earlier.

Patients in hospitals may also be informally classified according to their attractiveness and interest value for the staff. Thus, there are the "fascinomas," that is, the most interesting patients, on the one hand, and the "crocks," that is, the most undesirable patients, on the other. The degree of attention, sympathy, and support the patient receives often varies in accordance with this informal classification.

The personnel in the hospital are formally organized in a hierarchy. The general hospital's hierarchical structure is often very complex, with competing lines of authority and priority. Figure 33 shows a typical administrative organization. An important feature of the organization
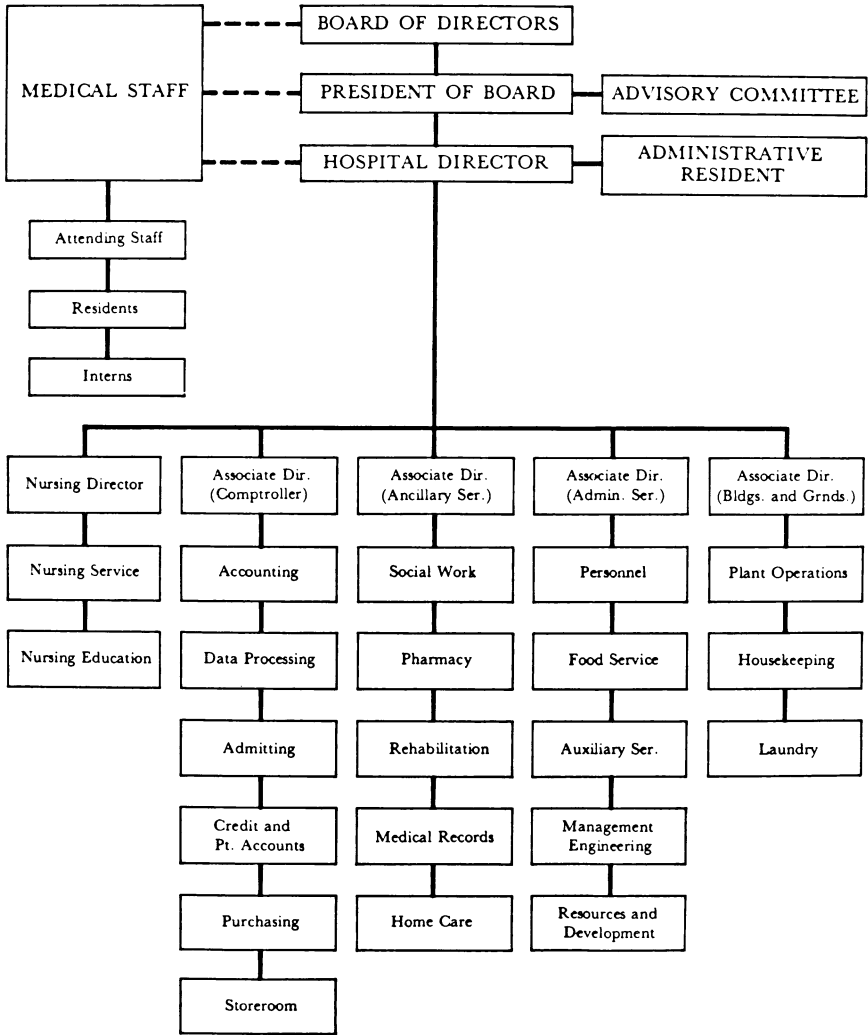
Figure 33. Administrative organization of a general hospital. (From Coe, 1970. Copyright 1970 by McGraw-Hill. Reproduced with permission.)

is that there are *two lines of authority*, as in Figure 33. The physicians are organized in a collegial fashion, although there is a stratification according to the specialty and status of the physicians into attendings, residents, interns, and students. This collegial organization of physicians is an outgrowth of the historical position of the physician as "vis-

itor'' or ''guest'' in the hospital (Coe, 1970). Of course, without these guests, the hospital cannot function. All other employees of the hospital are organized along another administrative line.

This dual line of authority is very different from almost any other kind of organization. In the customary single-line-authority organizations, like business, government, and the military, decision-making powers rest in the offices of the managers who direct the activities of the workers. In the hospital setting, however, the administrative managers have little authority concerning patient care, while the members of the medical staff, who are akin to consultants in single-line-authority systems, actually make decisions. The administrators' authority is often restricted to matters concerning the provision of means by which the doctors' orders may be successfully carried out (Coe, 1970). A potential conflict arises from the different sets of values: For the adminstrators, the primary concern is money, for the physicians, it is patient care and sometimes teaching and clinical research as well.

The dual-authority system can have major consequences in regard to *nursing*. Unlike physicians, nurses are directly responsible to the administrative structure of the hospital. In terms of professional activity, however, they are the recipients of the doctors' orders and share with the physicians a primary commitment to the patient. On occasion, this results in a ''double bind,'' a conflict between a nurse's professional role and her role as a hired employee of the hospital subject to the rules and regulations of an employee (vignette 2).

The hospital system is also characterized by an *extreme division of labor*. The professional, administrative, and nonprofessional staffs have clearly distinguishable and separate tasks, orientations, and loyalties. The stratification of occupational levels is rigid, and there is a ''*blocked mobility*'' in each level (Smith, 1955). Personnel cannot be promoted from one occupational level to another in the hospital without first resigning from their present position and obtaining further education. For example, without further schooling, a nurse's aide cannot be promoted to a registered nurse, and a janitor cannot be promoted to a hospital administration post. However, the physicians can and do become promoted to a higher rank, for example, from intern to resident to attending physician, without further formal schooling. A floor nurse can advance to being a head nurse, but not to being a physician.

This blocked mobility often results in the formation of cliques and special interest groups that are organized along occupational lines. Thus, nurses are more likely to talk with nurses most of the time, and physicians with physicians. One study showed, for example, that a doctor is three times more likely to speak with another physician than with a

nurse and that his interaction with personnel in other occupational groups is minimal. The frequency of conversational interaction of a nurse with other nurses was twice that of a nurse with other occupational workers (Wessen, 1958).

This same phenomenon is observed with respect to speciality lines even among physicians. Thus, a surgeon is more likely to talk with another surgeon than with a dermatologist, and a psychiatrist probably interacts with other psychiatrists most of the time. An obvious side effect of this *group formation* along occupational and specialty lines is the development of communicational barriers between occupations and specialties, and these can give rise to misunderstandings and mutual suspicions. This problem is especially acute if communication between nursing staff and physicians is blocked. It may take days for the nurse to communicate to the physician that his patient has been ruminating about depressive topics and not eating, even though other nurses may have known it all along. For example, in the case of the "sick Tarzan," the doctors were not informed directly by the nurse about his agitation and attempting to lift up the bed until the fifth day of hospitalization.

Another characteristic of the hospital organization is that it is *authoritarian* in nature. This is especially so concerning matters of patient care. The administrative authority is of the rational–legal type, based on the power residing in a particular office and concentrated at the top of the hierarchy. The nature of the physician's authority is more prestige-oriented and charismatic, related to the heavy responsibility of "saving people's lives," than rational–legal, related to the primary goal of the hospital—patient care. The dual line of authority, however, results in conflicts between the lines, the most common type being between physicians and administrators.

This type of conflict may become especially pronounced when planning for changes or renovations of a hospital. A study in England on four general hospitals asked the patients, staff, and administrators, "What is most important to alter in this hospital from a patient's point of view?" Each group had differing ideas about what was of primary importance. The administration emphasized physical change of the hospital, such as the provision of dayrooms; the doctors and nurses wanted to improve patient care, for example, by increasing the number of nurses; while the patients wanted to improve the quality of life in the hospital by improving the food, providing television sets, and in other ways (Raphael, 1965).

A more complicated authority relationship exists, especially among the medical staff, in a teaching hospital. The attending staff may be subdivided into the elite, university-affiliated professors and the private

practitioners in the community. Some hospitals have separate units, one for the academicians and another for the private physicians. Members of the academic attending staff are subject to dual roles, that of the clinician in the *hospital* and that of the teacher–researcher in the *university*, each of which has its own reward (promotion) systems based on different criteria. In some teaching hospitals, the house staff (interns and residents) may have conflicts in dealing simultaneously with the community attending physicians who are their patients' primary physicians and with the academic attending physicians to whom they are also responsible.

An interesting feature of the hospital as a hierarchical system is that somewhat as in the military, one can *identify* the *status* and *rank* of a person by *dress, speech*, and, to a lesser extent, *habits and manner*. Usually, the least neatly dressed, most disheveled· people are the patients, and the most well-dressed and well-groomed people are the attending physicians and hospital administrators. Physicians are often recognizable by the lab coats and uniforms (including the rank; e.g, attendings wear suits or long lab coats, while residents' and interns' costumes are usually different, as are the nurses'). Registered nurses wear uniforms different from those of practical nurses or nurse's aides. Some patients and outsiders take advantage of this and may "pass" for a doctor or nurse with appropriate dress and manner in the hospital, to leave without permission or even to steal drugs or equipment.

Organization of an Inpatient Unit

A typical inpatient unit of a general hospital consists of the following: beds, patients, nursing staff, ancillary staff, supporting staff, and physicians. Usually, all but the physicians and patients are more or less permanent; patients are transient, and the physicians generally rotate on and off inpatient units.

The number of beds usually determines the number of nursing staff to which the unit is entitled. Beds may be subdivided into private, semiprivate, and ward beds.

The *nursing staff* usually consists of a head nurse, an assistant head nurse, registered nurses, licensed practical nurses, and nurse's aides. Depending on the hospital, several nurses may be jointly responsible for the care of a number of patients ("team nursing"), or one nurse may primarily care for one patient ("primary nursing"), with the help of other nurses. The licensed practical nurses and nurse's aides assist the registered nurse. They are all responsible to the head nurse, who in

turn is responsible to the nursing supervisor of the division of nursing (e.g., medical or surgical) or to the director of nursing of the hospital. The head nurse and the registered nurses are also the recipients of the doctors' orders and are professionally required to carry them out competently. Nursing care, however, is not confined to carrying out the physicians' orders—it also includes interacting with the patients and providing comfort and evaluating distress when it occurs. It is obvious that good communication between the nurse and the physician is essential if the nursing care is to be coordinated with the therapeutic management plans of the physician.

The *ancillary staff* consists of the professionals related to patient care in a specialized area, such as dieticians, social workers, clergy, and the administrator (or the administrative intern) of the ward. They have liaison relationships with the nursing staff and sometimes with the physicians.

The *supporting staff* consists of the ward secretary or unit manager, who answers the telephones, makes sure that all the forms are in stock, and performs other duties, and the maintenance staff, including the janitors. Members of the supporting staff are usually responsible to a separate hierarchical administrative structure.

The physicians usually found on an inpatient service are the attending physician(s), the residents and interns (house staff), and the medical students. In teaching hospitals, the house staff is usually in charge of the patient, supervised by the attending and aided by the medical students. The physicians usually form a team around the patient, the team consisting of an attending physician, a resident, an intern, and a medical student. However, if the attending physician is not a full-time hospital or university physician, the house staff may have divided loyalties between the community-based attending and the full-time university-based physician supervisor to whom the house staff report as well.

Another socially complicating characteristic of the young physicians, particularly the house staff, is that they wield much power even though they are transient and relatively inexperienced members of the unit. Many members of the nursing staff on the unit may have been there for as long as 10 or 20 years and may be more knowledgeable about procedures and medical problems encountered on the ward than are the house staff, who change every few weeks or months. The mobility of the house staff may be envied by the less mobile staff members.

For a ward unit to function optimally, it is necessary that (1) each staff member has defined, unambiguous, and unchallenged roles, (2)

there is effective leadership and cooperation among disciplines, (3) there are smooth and open channels of communication between and among all the persons involved—including the patient, and (4) there is a spirit of commitment to cooperative efforts for good patient care.

To ensure the smooth running of this complex organization, many hospitals now assign *liaison psychiatrists* to inpatient services. The liaison psychiatrist, as a physician who is knowledgeable in psychosocial aspects of patient care and in organizational dynamics as well as biological aspects of illness, is often in a position to understand the dynamics of a ward and the complex interactive relationships between the medical and psychological aspects of patients and the ward problems. He may be in a position to improve and foster good communication between all concerned and to integrate multiple and fragmented observations for the primary physician. He is optimally situated to serve as a catalyst in opening up communications since, *unlike* everyone else on the ward, he does not report to any administrative authority who might take unfavorable action against a member of the ward, such as the nursing supervisor or the chairman of the department of medicine. The liaison psychiatrist may be an independent agent or may himself be a psychiatric resident, reporting to the department of psychiatry. The liaison psychiatrist is often assisted effectively by a *liaison nurse*, a psychiatric nurse with advanced training in liaison psychiatry, who may run liaison meetings with the nursing staff and act as a communicative link between the nurses and the physicians.

The liaison psychiatrist functions best when he forms a team with the primary physician (house staff or attending), the head nurse, and the social worker, in cooperation with the liaison nurse.

In most hospitals, the liaison psychiatrist also does psychiatric consultations on the patients in the unit. These involve evaluation and treatment of emotional or behavioral disorders of patients in the general hospital. Depression, suicide attempts, confusion, delirium, personality difficulties, drug-related problems, and intention to sign out against medical advice are common reasons for referral of patients to the psychiatrist. The liaison psychiatrist often finds that a patient's aberrant behavior reflects division and conflict among the staff. For example, a patient who receives conflicting information and advice concerning his illness from his attending physician and intern may be understandably confused and may wish to sign out. The liaison psychiatrist may arrange a meeting of the patient, the intern, and the attending to clarify the conflicting information.

The Hospital and Outside Organizations

Many organizations interdigitate with the hospital in personnel, patients, and material. Others have major impact on the policy decisions of a hospital. Finanical support for the hospital may come primarily from the government (Federal or local), charities, businesses, or, profits.

Laws and regulations at all levels directly influence hospital practices. Examples include the legislative acts requiring nondiscrimination because of race and the formation of professional standards review organizations (PSROs). Insurance companies' policies and practices, such as what procedures are covered, for what types of illness, and for how long, also influence hospitalization patterns and length of stay of patients.

Educational institutions, especially *medical schools*, have major direct influence on hospitals. Many full-time physicians in hospitals are also faculty members in medical schools and are influenced by the values and practices of the medical school. The prestige of the hospital is often directly related to the degree of affiliation with, and the prestige of, the medical school. This, in turn, affects the quality of the hospital personnel. (The more prestigious institutions can attract better-qualified staff.)

Religion and moral standards of the community have an effect on the hospital in influencing the types of patients admitted (e.g., Catholic hospitals have more Catholic patients and are unlikely to have many elective abortions).

The quality of patient care and physical and personnel adequacy of the hospital are scrutinized by such organizations as PSROs, the Joint Commission on Accreditation of Hospitals, and the American Medical Association.

Hospitals generally have intimate relationships with other hospitals, and each may refer patients to the others. Chronic care hospitals and psychiatric hospitals often receive patients from general hospitals. Clinics and the offices of private practitioners in the community often refer patients to the general hospital for admission.

The Hospital Environment

When a patient is admitted to a hospital, he finds himself in a *strange environment* and an *alien culture*. He is usually apprehensive, because he has a condition considered too serious to be treated in the doctor's office.

The intensity of his apprehension increases with his uncertainty about what to expect in the hospital. Much of what will happen in the hospital depends on tests and procedures to be done there; for example, laboratory test results may indicate the need for an exploratory operation. Depending on what is found at operation, there may be a need for radical surgery, chemotherapy, or no treatment—possibly the difference between life and death. *Uncertainty reigns.*

The *physical environment* of the hospital is not of much help in alleviating admission apprehension and anxiety. Cold, impersonal architectural styles, dark, gloomy rooms, and busy, businesslike, and forbidding staff characterize many traditional, especially older, hospitals. Patients often feel like unwelcome guests who nonetheless have to stay for, and in fear of, their lives.

Once in a hospital room, particularly a multibed room, the patient may be confronted with almost total *lack of privacy*. Conversations can be overheard, and many of the most private functions such as elimination now become public—often patients do not have *control* over when to bathe, urinate, or defecate—sponge baths are given by nurses, intimate parts of the body can be poked, observed, photographed, X-rayed, opened, or even cut with little advance warning and, it seems to the patient, by almost anyone wearing a uniform. There is even little control over what the patient can eat or drink.

The *language* spoken by the "people who count" in the hospital, the doctors and nurses, often sounds foreign to the patient—somehow distant and professional. The patient feels too stupid to ask questions—besides, the staff members seem to be so busy. The patient may also be in terror of offending the doctors and nurses—after all, his life *literally* depends on them.

An interesting situation occurs at the time of *doctors' rounds.* The patient's history, often including rather personal and confidential material, is presented to the attending while other patients, their visitors, and other staff (possibly the janitor and the maid) are looking on. Then, the patient may be undressed to varying degrees to be examined by a number of persons including the medical students. Heated debates concerning the diagnosis and treatment plans may take place among the house staff members and the medical students. The intern or resident who had the closest relationship with the patient may be humiliated by the attending for his lack of knowledge or for (horrors!) stupid proposals about treatment. Occasionally, discussion concerning a patient whom the doctors have already finished seeing continues in front of the next patient's bed. Of course, the patient before whose bed the discussion takes place assumes that the discussion is about him! Thus, sometimes

patients get entirely erroneous ideas about their conditions and treatment plans at the time of medical rounds.

Most patients' personal habit patterns must undergo change in the hospital. Their activities change and so do their habits concerning food, routine, and personal hygiene. Patients who like to play golf or work every day may instead have to read or watch television. Patients who are accustomed to sleeping alone may find it hard to fall asleep in a multibed room and annoying to be awakened in the middle of the night for the check on vital signs (blood pressure, pulse rate, temperature, and respiration). One patient's groaning, snoring, or moaning may keep other patients awake. Sedating medications, on the other hand, may keep patients constantly in a daze.

Many patients lose track of time in the hospital, and those who have the vulnerability become confused. Even if elderly patients with cerebral arteriosclerosis have not been confused at home in their usual habitats, in the absence of normal cues to help orient themsleves, many such patients become disoriented, confused, and agitated in the hospital, especially toward the evening when fewer cues are available (sundowning syndrome).

It should be understood that patients may become very *self-centered* in the hospital, partially as a function of the anxiety concerning the self and also partially due to the regression fostered by the hospital environment (see Chapter 5). When this happens, a patient may develop a tendency to think that almost anything he sees or hears is somehow related to him. Patients commonly think that conversation overheard between the doctors or nurses in the corridor concerns them; even conversations between other patients may be misinterpreted. The implication of this is that the staff should refrain from saying things within hearing of any patient (especially in the corridors or in the same room as the patient behind drawn curtains) that might be misconstrued in any way.

Certain specialized units in the general hospital present particular problems due to the physical and interpersonal environment. They include the intensive care unit, recovery room, operating room, hemodialysis unit, isolation unit, and cancer ward.

The *intensive care unit*, including the coronary care unit, has been thought to be contributory to a psychiatric syndrome called the "ICU psychosis." This is usually characterized by confusion, agitation, and a florid paranoid psychosis. ICU psychosis is most often considered to be an organic psychosis due to sensory deprivation (monotony), electrolyte imbalance, and multiple medications that are often administered in the ICU. Drug-induced psychosis, especially by meperidine (Demerol) and

by antiarrhythmic agents such as atropine, lidocaine, and procaine, is also common in the ICU.

The physical setting of the coronary care unit seems to have an impact on the general feeling tone of patients. Leigh *et al.* (1972) studied two types of coronary care units in the same hospital: an open, four-bed type and closed private cubicles. They found that patients were freer to express hostility, were concerned about possible shame, and manifested more anxiety concerning mutilation in the "open" unit, whereas patients in the cubicles were unable to express hostility directly but tended to deny and displace hostility or direct it inward and were more concerned about loneliness and separation. In the coronary care units studied, patients who showed high levels of hostility directed inward, low levels of overt hostility, and high levels of separation anxiety had a higher risk of developing cardiac arrhythmias.

Hackett *et al.* (1968) showed that patients who were able to deny anxiety effectively in the coronary care unit had a better prognosis than those who were unable to deny their anxiety. Witnessing other patients' distress and death is another hazard of being in an ICU. Most patients are able to deny these events effectively, but the next time, they tend to state a preference to be in single rooms (Hackett *et al.*, 1968). Although most patients deny being apprehensive in the coronary care unit, they nonetheless think about rather sad events, such as the death of loved ones in the past. An implication of this is that the physician need not feel constrained to inquire of these patients whether they are thinking upsetting or sad thoughts, lest he "suggest" such thoughts to them.

Psychological preparation is important in transferring patients out of the coronary care unit. Klein *et al.* (1968) found that patients had emotional reactions and more frequent cardiovascular complications (accompanied by an increase in urinary catecholamines) if they were transferred out of a coronary care unit without preparation. On the other hand, when the patients were psychologically prepared beforehand about the transfer, and when the primary nurse actually accompanied the patients to the new ward and introduced them to the new nurse, there were fewer complications after the transfer and less increase in urinary catecholamines.

The (surgical) *recovery room*, like the ICU, is a very confusing environment. Here, there is a curious and intense mixture of sensory overload and sensory monotony. There is always activity and usually no night–day difference because of artificial lighting and absence of windows. In addition, the patients are, as a rule, heavily sedated and in the process of recovering from anesthesia. Organic brain syndrome due to central nervous system depression brought on by anoxia (operative),

medications, electrolyte imbalance, and other disruptions, is common. Severe pain can contribute to the disorientation.

Some patients in the surgical recovery room are also unable to talk because of tracheostomies or soreness due to endotracheal tubes. This often results in severe frustration and fear on the patients' part, as well as agitation. For these patients, provision of a pencil and paper is often extremely welcome.

A rather frequent cause of agitation and psychosis during the post-operative phase is withdrawal from alcohol and other central nervous system depressants (see vignette 4 in Chapter 11). Patients' drug and alcohol histories should be carefully obtained at the time of hospitalization to prevent such withdrawal reactions from complicating the postoperative picture. Many alcoholic patients secretly bring alcoholic beverages into the hospital. Thus, delirium tremens can occur three days postoperatively, even though the patient may have been in the hospital for many days prior to the surgery.

A special form of psychosis, called *postcardiotomy delirium*, has been reported after open-heart surgery. Typically, the picture, consisting of illusions, visual hallucinations, and paranoid delusions, occurs after a lucid period of three to four days following the operation. The sensory monotony, sensory overload, amount of time on the artificial pump, and sleep deprivation in the surgical recovery unit have been suspected as contributing factors. The personality of the patient also seems to contribute to the development of postcardiotomy delirium; patients who score high on the dominance scale on Cattel and Weber's 16 Personality Factor Questionnaire have a greater tendency to develop it (Kornfeld *et al.*, 1972). The delirium usually clears within 24–48 hr after the patient is transferred to a standard hospital environment (Kornfeld *et al.*, 1972). This postcardiotomy delirium can be distinguished from the organic brain syndrome some patients have after open-heart surgery in that in the latter, the confusion is demonstrable immediately after the surgery, without any intervening lucid period.

The *operating room* is an environment in which patients often hear more than they see. In the course of the induction of anesthesia, hearing is the last sensory modality to become anesthetized and the first one to return. Even when the patient seems to be unconscious, he may still be able to hear. Conversations before an "unconscious" anesthetized patient, therefore, should be cautious and judicious. Thoughtless joking about the patient or discussions about the serious nature of his disease should be avoided. Special psychotherapeutic measures such as ventilation under hypnosis may sometimes be necessary to work through

anxiety and depression resulting from the effects of conversation overheard during anesthesia (Cheek, 1959).

The *hemodialysis unit* provides life support for many patients with chronic renal failure. Being dependent on the machine for life creates a number of psychological problems, including a sense of lack of control and of being dependent on external control. Some patients do not comply with the restriction in salt and fluid intake, and the suicide rate is greatly increased. Electrolyte imbalance and uremia often produce organic brain syndromes and depression. Here, again, successful use of denial has been associated with good prognosis.

Isolation rooms are provided in the hospital for patients with infectious diseases or when reverse isolation (guarding against exposure to infection) is necessary, such as in immunodeficiency diseases. The patients are in individual rooms, and everyone entering may have to wear a mask and a gown. There is a reduction in the number of visitors. In extreme cases, the patient may be in a plastic "bubble," totally insulated from the outside, and touched only through plastic gloves at the end of plastic arms built into the walls. Sometimes, individuals dressed in "space suits," wearing gloves, of course, may touch the patient. In this type of environment, the most significant complaint by patients is their inability to touch or be touched by another human being (Holland *et al.*, 1970). The significance of touching in development has already been discussed in Chapter 14.

The *cancer ward* increases anxiety and defensiveness on the part of both patients and staff. Patients are especially attuned to the doctors' attitudes—for signs of hope or evidence of rejection. Although patients usually do not ask the physicians directly about the prognosis of advancing cancer, they tend to consider the physicians' continuing interest almost as a commitment to cure. The physicians, on the other hand, tend to use the defense mechanisms of denial, isolation, and intellectualization vis-à-vis the patients, concentrating on the laboratory results and tissue findings. It is easier for the physician not to confront the patient's approaching death and his own impotence in the face of this inevitability (Leigh, 1973).

Doctors therefore have a tendency to minimize their contact with patients, leaving this up to the nurses. The nurses often feel unhappy and angry about having to be the only persons left to deal with the patients at a personal, emotional level. Opening up communications between the nurses and doctors, and showing doctors that their continuing interest is essential for the patients' maintenance of hope, can alleviate this situation. A liaison psychiatrist can contribute to this by being the catalyst for such communication (Klagsburn, 1970).

The hospital environment, in general, is a strange and anxiety-provoking environment for the patient. In the face of such anxiety, the patient responds with an exaggeration of his personality style, habitual coping mechanisms, and defenses. In the case of the "sick Tarzan" (Chapter 15), we see that he was in the coronary care unit, where his anxiety level was increased by the sensory monotony and the need for lying in bed. During the interview, his personality style of exaggerated showing off, dramatizing tendencies, and need to be in control was clearly demonstrated. One also sees evidence of major denial ("Nothing bothers me"), which augurs well for the patient in terms of prognosis, if only it were modulated and he were to stay in bed.

Summary

The hospital is a *complex social system,* influenced by various factors in the larger society. The formal hierarchical organization of the hospital personnel has *two lines of authority*: the physicians and all the other employees. Nurses, unlike physicians, are directly responsible to the hospital administration and at the same time are responsible to physicians in terms of patient care. Across occupations in the general hospital, there is a *"blocked mobility"*; that is, one cannot be promoted from one occupational level to another without further schooling.

Physicians often have *dual loyalties*: patient care and academic, especially in teaching institutions.

The typical *inpatient unit* in a general hospital is the functional unit in patient care, consisting of the beds, patients, nursing staff, ancillary staff, supporting staff, and physicians. Liaison psychiatrists and liaison nurses can play an important role in the smooth function of such a unit.

The hospital environment, in general, is characterized by *uncertainty* and lack of control from the patient's point of view. Certain special areas of the hospital, such as the intensive care unit, have their own special environments and attendant problems.

Implications

For the Patient

The hospital environment is a highly anxiety-provoking one in which the patient is threatened with the loss of autonomy and, possibly, life. *Privacy* is often disregarded, and patients feel too stupid to ask

questions or make requests. There are particular problems associated with special areas of the hospital. Patients should be *prepared*, if possible, about what to expect in the hospital, including whom to ask for information and other matters.

For the Physician

For obvious reasons, physicians feel quite at home in the hospital. They therefore tend to feel that patients should share their feeling of ease and familiarity in the hospital. Physicians should recognize how frightening and strange the hospital environment is for the patients. Physicians should make deliberate attempts to *ask* patients about their reactions to the physical and interpersonal environments of the hospital and encourage them to *communicate* their thoughts and feelings. Physicians should also recognize that cooperation among the professions and occupations in the hospital, including nurses and other workers, is essential for good patient care. Doctors should encourage the *nurses* to *report* any new observations concerning patients and to communicate openly about difficulties or dissatisfactions. Doctors should also recognize the *"double bind"* nurses often feel in having dual lines of authority to which they are responsible.

For the Community and the Health-Care System

Hospitals should be constructed *aesthetically* and *functionally*. Every room should have paintings, calendars, and television or radio. Educational programs should be designed for patients and potential patients about where to get information in the hospital and where to "gripe," and the hospital administration and the medical establishment should encourage communication and cooperation among different occupational groups in the hospital. Medical education should emphasize respect for a patient's *privacy* and the importance of not embarrassing patients (or the medical student, for that matter) on walk rounds. Hospitals should provide *liaison psychiatrists* for patient-care units to foster cooperation and communication among different groups and also to prevent and manage behavioral and emotional problems of patients.

Recommended Reading

Coe RM: *Sociology of Medicine*. New York, McGraw-Hill, 1970. A lucid and concise textbook of medical sociology. Has a good chapter on the hospital as a social system, as well as on the development of the modern hospital.

Kornfeld DS: The hospital environment: Its impact on the patient. *Adv Psychosom Med* **8**:252–270, 1972. A good review paper on the hospital environment's impact on the patient. Good list of references for interested readers.

References

Cheek DS: Unconscious perception of meaningful sounds during surgical anesthesia as revealed under hypnosis. *Am J Clin Med* **1**:101, 1959.

Coe RM: *Sociology of Medicine*. New York, McGraw-Hill, 1970, pp 270–271.

Freeman, HE, Levine S, Reeder LG: *Handbook of Medical Sociology*. Englewood Cliffs, New Jersey, Prentice-Hall, 1972, pp 276–277.

Hackett TP, Cassem NY, Wishnie HA: The coronary care unit: An appraisal of its psychological hazards. *N Engl J Med* **279**:1365–1370, 1968.

Holland J, Harris S, Plumb M, *et al.*: Psychological aspects of physical barrier isolation: Observation of acute leukemia patients in germ-free units. *Proc Int Congr Hematol*, 1970.

Hollingshead AB, Redlich FC: *Social Class and Mental Illness*. New York, John Wiley & Sons, 1958.

Klagsburn S: Cancer, emotions and nurses, *Am J Psychiatry* **126**:1227–1244, 1970.

Klein RF, Kliner VS, Zipes DP, *et al.*: Transfer from a coronary care unit. *Arch Intern Med* **122**:104–108, 1968.

Kornfeld DS, Heller SS, Frank KA, *et al.*: Personality and psychological factors in postcardiotomy delirium. *Arch Gen Psychiatry* **31**:249–253, 1974.

Leigh H: Psychiatric liaison on a neoplastic inpatient service. *Int J Psychiatry Med* **4**:147–154, 1973.

Leigh H, Hofer M, Cooper J, *et al*: A psychological comparison of patients in "open" and "closed" coronary care units. *J Psychosom Res* **16**:449–458, 1972.

Myers JK, Bean LL: *A Decade Later: A Follow-Up of Social Class and Mental Illness*. New York, John Wiley & Sons, 1968.

Raphael W: If I could alter one thing. *Ment Health (London)*, pp 1–5, 1965.

Roth JA, Eddy EM: *Rehabilitation for the Unwanted*. New York, Atherton Press, 1967.

Scott RA: *The Making of Blind Men: A Study of Adult Socialization*. New York, Russell Sage Foundation, 1969.

Smith H: Two lines of authority are one too many. *Modern Hospital* **85**:48–52, 1955.

Wessen AF: Hospital ideology and communication between ward personnel, in Jaco E (ed): *Patients, Physicians and Illness*. New York, The Free Press, 1958, pp 448–468.

Therapeutic Dimensions

*A 42-year-old married white man was admitted to the hospital with high
fever. Subsequent workup showed that he had aspiration pneumonia (pneu-
monia due to aspiration of a foreign substance such as food into the lungs,
usually in a state of intoxication or coma). History revealed and highlighted
the fact that the patient had increased his alcohol intake considerably in the
last two years. He had been divorced four years ago and remarried two years
ago. His second wife had turned out to be an alcoholic.*

*In addition to aspiration pneumonia with secondary staphylococcal in-
fection, liver function tests showed abnormalities indicating early liver dis-
ease, probably due to excessive alcohol consumption. The pneumonia was
treated with antibiotics. The high fever was treated with aspirin and alcohol
rubs. His wife was interviewed by the doctor and the social worker. As the
pneumonia subsided, the doctor had a serious talk with the patient and his
wife concerning the role of alcohol in the patient's medical condition. It was
decided that both the patient and his wife should join Alcoholics Anonymous.
Also, arrangements were made for them to see the social worker on a regular
basis for couples therapy. They were also directed to take multivitamins.*

Management plans must be directed at all three dimensions—the bio-
logical, the personal, and the environmental. In the foregoing vignette,
the main biological treatment for the disease, pneumonia, was anti-
biotics. To prevent the vitamin deficiency syndrome that often occurs
in alcoholism, multivitamins were also prescribed.

Alcohol rub for the fever was a treatment directed at the heat in-
terchange between the patient and the physical environment. Air con-
ditioning and humidification of the room are also examples of therapy
in this dimension.

385

The physician was able to diagnose a disorder at the personal level in this patient—alcoholism (or the pattern of excessive alcohol intake). Further, he found that this disorder was shared, and probably encouraged, by the patient's wife. The management plans thus included treatment for alcoholism in the environmental and personal dimensions through Alcoholics Anonymous, the social worker, and couples therapy.

We will now briefly consider some common therapeutic modalities as they are directed primarily at each of the dimensions of the patient system. It will be clear in each section, however, that the actual therapeutic effects interact extensively across dimensions.

Approaches in the Biological Dimension

Broadly considered, any therapeutic intervention works through the biological system, including interpersonal interventions and those directed at the personal dimension, since even symbolic transactions occur through perceptual and cognitive processes the substrates of which are physicochemical. In the discussion that follows, treatment at the biological level is considered in a narrower sense, that is, biological treatment of the disease, such as treatment directed at eradicating its etiological cause (e.g., the use of antibiotics, potassium administration to correct hypokalemia, and surgical correction of tetralogy of Fallot). Most medications are used with the expectation that the biological effect will result in cure, reversal, or amelioration of the disease process. Despite such expectations of biological efficacy, physicians should recognize that many drugs also work through the expectancy effect (personal dimension). Furthermore, medications with proven biological effects may, in addition, have *side effects in the personal dimension* (Chapter 20). The side effects on the personality may be mediated through the drug's biological effect on the central nervous system or through its symbolic meaning. For example, although methadone is a potent analgesic biologically, some patients for whom it is prescribed may become anxious because they identify it with drug addiction, and they may experience little pain relief, especially if it is administered without adequate explanation and reassurance. Many drugs cause psychological effects. (For example, depression is a relatively common side effect of certain medications, such as reserpine, a drug used in management of hypertension.)

Surgery is a definitive intervention in the biological dimension. However, it is important to recognize that surgery may also have placebo effects. For a while many years ago, ligation of the internal thoracic

artery was considered to be effective in the treatment of angina pectoris, and, in fact, many patients seemed to benefit from it. Eventually, however, the procedure turned out to be without long-term benefit. The beneficial effects experienced by patients were due to the placebo effect.

Some medications and surgical procedures have major interpersonal side effects (side effects in the environmental dimension). For example, foul-smelling ointments, loss of hair due to chemotherapy, and mutilating operative procedures may restrict the patient's social contact. With some biological treatments, such as a plaster cast, the patient's mobility is greatly compromised.

Surgical procedures are often associated with personal fantasies unrelated to their biological effects. For example, the frequent confusion between the kidney and genital organ functions may lead some patients to have fears concerning sexual potency after nephrectomy. Many women have the notion that their sexual enjoyment and libido will cease following simple hysterectomy. In fact, because of this expectation in some patients, there may be changes in sexual behavior and experience after hysterectomy even though the external genitals and ovaries are intact.

Physicians should *explore* the patient's fantasies and ideas concerning any proposed biological intervention and *clarify* them with adequate and lucid information.

Approaches in the Personal Dimension

Therapy in the personal dimension is geared toward the *patient's illness behavior, general health* (including habits), and the *meaning of illness* and psychological reactions to it. Reduction of the suffering aspect of the disease (illness) is essential for good collaborative work between the doctor and the patient. As we examined in Chapter 3, the immediate expectations of the patient in the consulting room are primarily concerned with reduction of suffering (personal dimension).

Biological treatment modalities that may be used for treatment in the personal dimension include *drugs, physical therapy,* and *electroshock treatment,* as well as psychotherapy and psychosocial interventions. Unlike biological treatment in the biological dimension, in which etiological factors are the main target, biological treatment modalities in the personal dimension are primarily directed at modifying *feelings* and at the general functional capacities and well-being of the patient. Accordingly, drugs used in symptomatic treatment, for example, to relieve pain and

anxiety or to reduce fever and inflammation, are included in this category.

As discussed in Chapter 16, the *doctor–patient relationship* is a powerful therapeutic tool operating in the personal dimension. The placebo aspects of being treated by a doctor are often powerful, at least initially, no matter what the doctor actually does to treat the disease. Newly "discovered" medications and procedures often seem especially efficacious as a result of this effect—thus, the admonition, "You should treat as many patients as possible with new drugs while they still have the power to heal," which has been attributed to Trousseau, Osler, and Lewis (Shapiro, 1960). Physicians should be aware of this temptation and weigh the possible expectancy effect (beneficial) of a new medication against possible, yet unreported, adverse effects. The potency of the healing effect of the doctor–patient relationship can be increased by the physician's interest, empathy, and competence. It can be neutralized by a lack of interest in attempting to understand where the patient's distress and concerns are focused.

Psychotherapy in Medical Settings

Psychotherapy conducted by the physician consists of the application of psychological techniques within the context of the doctor–patient relationship, usually with specified and limited goals in mind. A more general and detailed discussion of psychotherapy is beyond the scope of this book; the interested reader should refer to standard psychiatric texts. The brief discussion that follows deals with psychotherapeutic principles and techniques that nonpsychiatric physicians should understand and be able to use effectively in clinical practice.

The doctor–patient relationship renders any and all actions and ministrations of the physician psychologically influential, whether they are explicitly intended to be or not. In turn, their psychological impact can and often does influence the course of illness and the patient's response to treatment. The physician who understands the psychological meaning of his work with patients can wittingly, judiciously, and deliberately utilize the psychotherapeutic potential inherent in the doctor–patient relationship to promote healing and maximize the beneficial effects of his medical treatment. Consider, for example, the psychological aspects of history-taking, physical examination, ordering of diagnostic tests, prescription of medication, and scheduling and conduct of follow-up visits.

The friendly and nonjudgmental, but at the same time professional, interest and concern that are manifested toward the patient by the physician in the process of skillful *medical history-taking* provide an interpersonal base for reassurance and for the relief of tension that occurs in the patient when he can share with the doctor personal worries, problems, guilts, and anxieties that he had not shared with anyone previously and so had carried as private personal burdens. And, as the patient is encouraged to review possible contributing factors to disease, important facts concerning personal habits (e.g., smoking, alcohol), occupational stresses, interpersonal tensions, and other problems may emerge that will be relevant to formulating the nature of the clinical problem and the therapeutic approaches that will be required to deal with it.

Similarly, it is easy, on reflection, to appreciate the anxiety-relieving value of a thorough *physical examination*, skillfully and systematically conducted in an appropriate, dignified, and comfortable setting. This is important in setting the basic tone for the developing doctor–patient relationship. Readers might think back on some of their own personal experiences as patients undergoing physical examination.

It is important for the physician not only to plan a program of *diagnostic procedures* in advance but also to share its rationale, strategy, and meanings with the patient. The physician's medical knowledge and skill are often crucially tested when he has to decide when and whether to dispense with further diagnostic tests. The way in which differential diagnosis is pursued can reinforce previous good developments in the relationship and can be particularly important in imparting to the patient the belief that the doctor is competent, concerned, and trustworthy.

When these initial steps have gone well, the patient's trust and confidence are based on reality as well as on positive transference phenomena that, of course, reinforce his trust and confidence.

At this juncture, the issue of *sharing information* with the patient and of *reassurance* warrants discussion. Reassurance should never be "hollow" or insincere; that is, the physician should not make explicit assertions that he knows to be unfounded. When it is necessary to spare the patient undue worry and anxiety, it is usually better for the doctor to carry the burden of leaving some things unsaid until such time as the patient may be ready to cope with them. Most often, the patient's fears are much worse than reality—providing the physician with considerable room for positive, explicit, factual explanations that will be reassuring. As we have noted earlier, sharing information is important, and in doing this, the doctor should take into consideration the patient's personality type, habitual defenses, and ways of coping in order to pro-

vide the information in a way that will be most helpful and useful to the patient (Chapter 17).

Hospitalization may be psychotherapeutic in several ways. Despite the *anxiety and adjustment process* inherent in this major environmental change for the patient (see Chapter 18), hospitalization (if prepared for thoughtfully) means a definitive step for the patient in his commitment to fight the disease process. The hospital environment, once its disturbing features have been dealt with, provides the patient with a safe place where competent professionals will help him to get well. In fact, for some patients, the hospital may provide a needed temporary respite ("vacation") from pathogenic stressful life situations with which they have not yet been able to cope successfully. Complex and imposing hospital equipment may be seen as reassuring. Without trust in the physician and the hospital (based on careful preparation or a preexisting good relationship or both), the same equipment may be frightening. Hospitalization may provide legitimate opportunities for gratifying dependency needs, and this may relieve a chronic state of frustration or deprivation that may even have contributed to predisposition or vulnerability to disease. Some patients may, for the first time, gain a perspective on their lives and learn better ways of coping or relaxing during the course of hospitalization.

Follow-up appointments manifest that (1) the physician is interested in the patient's course and recovery and (2) the physician feels some hope about the patient's future and, in any case, will not abandon him. The latter is especially reassuring to seriously ill patients, including terminal cancer patients (Abrams, 1966). *Physical therapy* may provide a sense of mastery and hope for paralyzed patients. *Drugs*, as we have seen, are potent psychotherapeutic as well as pharmacotherapeutic agents, and not only because of placebo effects. In addition to the fact that it is reassuring for the patient to know that there is a drug for his illness, the doctor's "giving" of the medication often gratifies basic dependency needs and is received in the context of the patient's trusting relationship with the physician. On the other hand, if drugs are given casually and haphazardly, the patient may consider the drug as a poor substitute for the physician's interest—an antipsychotherapeutic effect (see Chapter 20).

Psychotherapeutic *techniques* that may be used by the general physician in the context of medical management of patients include the following:

Reassurance: This consists of a general optimistic and hopeful attitude and specific statements, based on data or experience or both, de-

signed to allay exaggerated or unfounded fears of the patient. For reassurance to be effective, the physician should know the sources of the patient's fears. They are often based on an incorrect understanding of the disease or proposed procedure.

Sharing life's problems: The doctor is a person in whom many patients can confide and to whom they can talk about things that they may feel embarrassed or fearful to discuss with anyone else. The simple act of verbalizing an emotionally taxing thought or situation can relieve chronic anxiety (and physiological activation that accompanies it). After such verbalization, the patient may achieve a fresh outlook and be able to cope with the situation. The process of talking about an emotionally charged situation or thought, with expression of the emotion, has been called "ventilation"; we consider the term "sharing life's problems" to convey its nature more correctly.

Guidance and advice: Judicious advice and guidance based on sound medical knowledge carry authority and often achieve readier acceptance by the patient than if they had been offered by a parent or employer. This is especially true when the advice is directed toward a possible contributing factor to disease, such as alcohol. For example, an alcoholic patient who has just recovered from an exquisitely painful episode of pancreatitis is more likely to heed the physician's advice (rather than a relative's) to join Alcoholics Anonymous.

Education: Through an educative process involving questions and answers and, possibly, reading material suggested by the doctor (bibliotherapy), the patient can learn about his medical condition and the doctor's strategy in treating it. This can reduce unnecessary anxiety and reinforce positive aspects of the doctor–patient relationship. This may also foster the adaptive defense mechanisms of sublimation and intellectualization (Chapter 5). Education may also be directed toward behavioral changes to reduce contributing factors to disease, for example, the dangers of smoking and the early warning signs of cancer requiring checkup.

Environmental manipulations (see also the section on Approaches in the Environmental Dimension): When it becomes clear that certain stressful factors in the environment (e.g., occupational, interpersonal) are participating as serious vectors by precipitating or accelerating the disease process, it may be wise for the physician to suggest changes in the environment if such factors can be avoided (e.g., change of job and avoidance of mother-in-law's visits).

Limited interpretations: This consists of judicious sharing with the patient the observation that certain stressful life situations are regularly associated with flare-up of disease or with incidence of symptoms. For

example, the physician may note that many of a patient's attacks of migraine headache have regularly followed periods of intense, pressured work to meet deadlines. The patient may be unaware of this fact and, if made aware, may be able to rearrange work schedules to avoid repetitions of this situation or develop better ways of coping with the stress, for example, by finding tension outlets at such times in exercise or the practice of relaxation techniques. Obviously, the physician must know his patient quite well and have a good basic relationship in order to apply his own "insight" into the patient's problems in this way. The same holds true for environmental manipulation.

What may all the foregoing accomplish, particularly for patients for whom psychosocial factors may play relatively minor roles as etiological, pathogenic, or influencing factors? For many patients, the doctor's expectations of realizing direct positive benefits for the patient may be quite limited. Still, some very worthwhile limited goals may be realized:

1. Varying degrees of symptom relief
2. Giving up of secondary gain
3. Improvement in morale and overall quality of adjustment to illness and maintenance of hope
4. Improvement in general life adjustment, interpersonal relationships, and quality of life
5. Achievement of good compliance with the medical regimen

Noncompliance is a major problem in medical care (Sackett and Haynes, 1976), particularly in chronic diseases that are relatively asymptomatic for long periods, but nonetheless dangerous if untreated, for example, essential hypertension. Perhaps one of the major benefits of good psychologically oriented medical management is realized in prevention of serious noncompliance.

For patients for whom psychosocial stresses may play contributing etiological, pathogenic, or influencing roles, some additional benefits may be realized to the degree that the measures discussed above enable the patients to manage their lives more effectively (i.e., to avoid stressful situations, where possible, and to understand, defend, or cope better); the rate of progression of the disease process may be slowed and complications or exacerbations delayed and possibly, in some cases, avoided.

Often, the physician who works in this way with a patient will find that problems persist to the degree that the patient needs more advanced or specialized psychotherapy. This may be either because his life-adjustment problems are serious enough in their own right to require it

or because the doctor judges that they are seriously aggravating the disease. Here is where another benefit of working psychotherapeutically is realized—the patient can be expected to react positively to the suggestion that he have a consultation with a psychiatrist. The physician is in a position to make an effective and comfortable referral. For example:

> Mrs. Jones, it is clear from our discussions that your ulcer flared up when things got tense at home between you and your husband. The problems in your marriage that you are telling me about touch on issues that may be beyond my technical competence as an internist. But since they, like the foods you eat, and so on, play an important part in healing or failure to heal, I think it would be a good idea for you to see Dr. Smith, a specialist in psychiatry, for a consultation. He has been helpful to me before in evaluating such situations and in helping to reach an opinion on the advisability of undertaking a course of psychotherapy. In any case, I will continue with your medical treatment and follow-up

Such a referral does *not*: (1) say to the patient, "It is all in your head"; (2) reject the patient; (3) leave the patient without hope.

Formal Psychotherapy

Formal psychotherapy may be classified into individual, couple, family, and group psychotherapies on the basis of the number and type of patients treated. On the basis of theoretical orientation, it may be psychodynamically oriented (which may be subclassified into Freudian, Jungian, Adlerian, Sullivanian, Honeyian, and other types), behavior-therapy-oriented (Skinnerian), "client-centered" (Rogerian), or "eclectic" (flexible combinations of the aforementioned types). Medications, when needed, may be combined with any of these approaches. Classified on the basis of special techniques, there are psychoanalysis, psychoanalytically oriented psychotherapy, hypnosis, sodium amytal interview, behavior therapy (techniques such as desensitization, flooding, biofeedback), sex therapy, and others such as psychodrama and art therapy.

Most psychodynamically oriented psychotherapies can be classified along a spectrum from mainly "insight-oriented" to mainly "supportive" psychotherapies, although, almost invariably, they coexist with differing emphasis on one or the other aspect. The insight-oriented psychotherapies, as represented by psychoanalysis, aim at helping the patient develop understanding—explanatory concepts regarding the un-

conscious conflictual origin of his difficulties—that will aid in resolving conflict and permitting personality growth and more satisfactory life adjustment. Supportive psychotherapies (aspects) aim primarily at increasing the patient's defensive and coping abilities.

Self-Regulatory Techniques (Biofeedback, Relaxation Response, Self-Hypnosis). Biofeedback, relaxation and meditative techniques, and self-hypnosis are geared toward "self-regulation," that is, the maintenance of adaptive changes in the body through self-generated behaviors (Leigh, 1978). Through these techniques, the patient can control maladaptive or potentially maladaptive physiological activation, produce a state of relaxation, and increase his coping abilities.

Biofeedback is a technique by which the patient learns to acquire control over bodily functions that are usually not controlled voluntarily, such as skin temperature, heart rate, blood pressure, and tones of certain muscles as recorded by EMG. This usually involves the use of electronic equipment that monitors the organ system to be controlled, the activity of which is "fed back" to the patient in the form of signals indicating the success or failure of the attempted control. For example, blinking green lights from a polygraph may indicate a decrease in heart rate. The subject may then attempt to increase the frequency of the green light if he is to learn to decrease his heart rate. Biofeedback is based on operant conditioning principles.

Operant conditioning (sometimes called "instrumental" conditioning) was first described by B. F. Skinner in the 1930s (Skinner, 1938). Learning by operant conditioning, unlike Pavlov's classic conditioning (see Chapter 4), involves behaviors that are learned in response to *reward* or *punishment.* For example, a hungry rat in a cage equipped with a lever will, at some point, lean on the lever (spontaneously emitted behavior, or "operant behavior"). If food is given to the rat each time it leans on the lever, the animal will increase the frequency of lever-pressing. The food in this case is a *positive reinforcer*, a stimulus that increases the probability of the operant behavior if the stimulus is given after it. A *negative reinforcer* is a stimulus the *removal* of which following an operant behavior increases the probability of that behavior. For example, if a rat can turn off continuous electric shock by pressing a lever, the shock is a negative reinforcer for the operant response of pressing the lever.

Operant conditioning was initially considered to be a higher type of learning than classic conditioning, and the autonomic nervous system, being more primitive phylogenetically than the voluntary somatosensory nervous system, was considered to be incapable of operant conditioning. The pioneering work of Miller (1969) and his colleagues

showed, however, that the autonomic nervous system could indeed "learn" from reward and punishment. For example, Miller was able to show that animals could, among other things, increase or decrease blood pressure and pulse rate, and increase or decrease glomerular filtration rate in response to reward. Biofeedback is the clinical application of this principle. Biofeedback has been shown to be effective in a variety of stress-related disorders such as migraine headaches, Raynaud's disease, certain cardiac arrhythmias, and tension headaches (Blanchard and Young, 1974; Task Force Reports of the Biofeedback Society of America, 1978).

The *relaxation response* is defined by Benson (1977) as a set of integrated physiological changes that may be elicited when a subject assumes a relaxed position and engages in a repetitive mental action, passively ignoring distracting thoughts. The physiological changes include decreases in oxygen consumption, heart rate, respiratory rate, and blood lactate; slight increases in skeletal muscle blood flow; and an increse in EEG alpha waves. This response is presumed to be an integrated hypothalamic response that functions as a protective mechanism against overstress and as a counteraction against the fight–flight response (see Chapter 4).

The relaxation response was identified by Benson (1975) when he was studying the physiological changes accompanying meditative techniques. Thus, the self-regulatory physiological effects of meditative techniques are considered to be mediated by the relaxation response. The relaxation response seems to be a useful adjunct in the management of essential hypertension (Benson, 1977; Jacob *et al.*, 1977; Blanchard and Miller, 1977).

Self-hypnosis may also be used to bring about muscular relaxation and emotional tranquility. Hypnosis seems to elicit beneficial physiological effects through vivid imagery and, in highly susceptible individuals, through a change in perception (Hilgard, 1965, 1975; McGlashan *et al.*, 1969). Self-hypnosis is also useful in pain control, habit control (such as smoking and overeating), and anxiety control (Spiegel and Spiegel, 1978). Interestingly, hypnoanesthesia is not reversed by naloxone, unlike acupuncture anesthesia or placebo anesthesia (Goldstein, 1976).

Approaches in the Environmental Dimension

Any treatment modality involves some environmental change, including the contact with the health-care system for the patient. As noted above, simply being in the hospital is often helpful for the patient, be-

cause of both the expectations of forthcoming help and a sense of being "in good hands." Beneficial effects may also arise from the distancing of the patient from possibly pathogenic physical or interpersonal situations by admission to the hospital.

A change in the environment other than that of hospitalization may be indicated in some patients. Until recently, rest cure was an important treatment modality very commonly prescribed by physicians. We are all familiar with the revitalizing effects of a vacation away from home.

Environmental change may be necessary specifically to avoid pathogens, as in the case of severe allergies to endemic allergens. This may involve, for some patients, giving up pets, and for others, moving to a different climate.

Interpersonal changes may involve family (e.g., divorce, marriage, family planning), school, occupation, or type of work. For example, a patient with serious heart disease may have to be transferred from a physically strenuous job to a less strenuous one.

Another interpersonal change may involve *changes in approach* to the patient. The hospital staff's learning how to approach a patient while taking into account his personality needs can result in a therapeutic environmental change. In the case of the "sick Tarzan," this played a very important role in the management strategy (see Chapter 21).

Often, strains and tensions among the *staff* contribute to behavioral and psychological problems of patients. For example, when excessive staff turnover and low morale have generated interpersonal difficulties between members of the hospital staff, measures taken to improve morale and communication between and among the different groups (Chapter 18) will often, if successful, produce a better environment for patient care and amelioration of what appeared to be patient problems.

The *physical environment of the hospital* room can be therapeutic. A bright and cheerful physical environment contributes to a hopeful and optimistic psychological set. Respecting the patient's wishes concerning a multibed or private room can increase his sense of control and so lessen anxiety (Leigh *et al.*, 1972).

It is essential that the hospital environment provide patients who have a tendency to be confused and irascible (Chapter 8), especially those with chronic organic brain syndrome, with cues for orientation: a large calendar, a clock, and a radio or television. Staff members should also introduce themselves each time they touch the patient or perform procedures, since the patient may have forgotten that he is in the hospital and may develop delusions about what the staff is doing. Night lights are also helpful for these patients, since they may become completely confused and agitated on waking up at night in strange sur-

roundings. Familiar objects from home (e.g., framed photographs, books) should be brought to the hospital and kept next to the patient's bed. Family and friends should be encouraged to visit. This will reduce the patient's anxiety by providing a sense of security and familiarity.

Summary

Management of a patient involves intervention in three dimensions: the biological, the personal, and the environmental. Therapeutic modalities geared primarily toward the biological system often have additional or side effects at the personal and interpersonal levels. The *doctor–patient relationship* is a powerful tool in therapeutic management in the *personal* dimension. Insight-oriented psychotherapy, in general, aims at helping the patient develop an explanatory perspective concerning his sufferings. Supportive psychotherapy is aimed at increasing the patient's coping ability here and now. Simply providing an opportunity for the patient to talk about his concerns and problems with the physician has major therapeutic impact. Many drugs and surgical procedures are used to alleviate discomfort and promote general health— measures directed at the personal dimension. *Environmental intervention* is *inherent* in any contact of the patient with the health-care system, but especially in the case of hospitalization. Changes in approach to the patient can play an important role. For patients with a tendency to confusion, provision of a stable and orienting environment is an important aspect of treatment.

Implications

For the Patient

Therapeutic modalities geared toward the environmental and personal dimensions may have the most obvious impact for the patient. Therapeutic modalities geared toward the biological dimension, if the results are not immediately obvious, may not be adequately appreciated by the patient unless sufficient information is given. The patient's fantasies and ideas concerning all therapeutic modalities are determined by his unique experiences and cultural expectations. His ideas concerning the nature and efficacy of the proposed modality may be entirely at variance with the physician's (e.g., the efficacy of laetrile, of electroshock therapy, or of a particular school of psychoanalysis).

For the Physician

The physician should determine rational management approaches to the patient directed at the *three dimensions* by using the systems–contextual framework and the Patient Evaluation Grid (PEG). The *priority of intervention* is determined on the basis of the gravity of the treatment objective and the immediacy of the problem. Physicians should be aware that the *doctor–patient relationship* is an important therapeutic tool in the personal dimension. We should also think about possible side effects of any proposed therapeutic modality in all three dimensions, for example, the side effects of a surgical procedure at the personal and interpersonal levels.

Psychotherapy is *not* a technique exclusively in the domain of the psychiatrist or psychotherapist. Whenever a physician talks with a patient, there is an element of psychotherapy.

For the Community and the Health-Care System

Medical education should emphasize the three dimensions of intervention for optimal treatment of patients. Physicians should be educated to play a central role in the three-dimensional management of patients. There has been a tendency for physicians to concentrate on the treatment of disesae, while nurses and social workers have been concerned primarily with problems in the personal dimension and their interpersonal aspects. This is less than optimal, since medical knowledge and psychiatric skills taught to physicians are necessary in assessing the therapeutic needs of the patient in all these dimensions. The *primary evaluator* and *manager* of the patient should be the responsible physician, who can bring (and collaborate with) members of other disciplines and specialties into the specialized care of the patient.

Recommended Reading

Levine M: *Psychotherapy in Medical Practice.* New York, Macmillan, 1945. Although somewhat dated, this classic book shows the psychotherapeutic aspects of various ministrations by the general physician, as well as some techniques that can be used by more psychiatrically oriented physicians and psychiatrists.

Shapiro AK: A contribution to a history of the placebo effect. *Behav Sci* 5:109–135, 1960. This is an excellent review of the prescientific doctor–patient relationship and the placebo effect in general. Highly recommended reading for any student of medicine.

Strain JJ, Grossman S: *Psychological Care of the Medically Ill: A Primer in Liaison Psychiatry.* New York, Appleton-Century-Crofts, 1975. A concise and useful book on the psy-

chological management of medically ill patients. Certain specific clinical issues, such as hypochondriasis, the dying patient, and the surgical patient, are discussed, as well as the function of the liaison psychiatrist in the management of medically ill patients. Good supplementary reading for the student interested in comprehensive care.

References

Abrams R: The patient with cancer: His changing patterns of communication. *N Engl J Med* **274:**317–322, 1966.

Benson H: *The Relaxation Response.* New York, William Morrow & Co, 1975.

Benson H: Systemic hypertension and the relaxation response. *N Engl J Med* **296:**1152–1156, 1977.

Blanchard EB, Miller ST: Psychological treatment of cardiovascular disease. *Arch Gen Psychiatry* **34:**1402–1413, 1977.

Blanchard EB, Young LD: Clinical applications of biofeedback training: A review of evidence. *Arch Gen Psychiatry* **30:**573–589, 1974.

Goldstein AL: Opioid peptides (endorphins) in pituitary and brain. *Science* **193:**1081–1086, 1976.

Hilgard ER: *Hypnotic Susceptibility.* New York, Harcourt Brace & World, 1965.

Hilgard ER: Hypnosis. *Annu Rev Psychol* **26:**19–44, 1975.

Jacob RG, Kraemer HC, Agras WS: Relaxation therapy in the treatment of hypertension: A review. *Arch Gen Psychiatry* **24:**1417–1427, 1977.

Leigh H: Self-control, biofeedback, and change in "psychosomatic" approach. *Psychother Psychosom* **30:**130–136, 1978.

Leigh H, Hofer M, Cooper J, *et al.*: A psychological comparison of patients in "open" and "closed" coronary care units. *J Psychosom Res* **16:**449–458, 1972.

McGlashan TH, Evans FJ, Orne MT: The nature of hypnotic analgesia and placebo response to experimental pain. *Psychosom Med* **31:**227–245, 1969.

Miller NE: Learning of visceral and glandular responses. *Science* **163:**434–445, 1969.

Sackett DL, Haynes R (eds): *Compliance with Therapeutic Regimens.* Baltimore, The Johns Hopkins University Press, 1976.

Shapiro AK: A contribution to a history of the placebo effect. *Behav Sci* **5:**109–135, 1960.

Skinner BF: *The Behavior of Organisms: An Experimental Analysis.* New York, Appleton-Century-Crofts, 1938.

Spiegel H, Spiegel D: *Trance and Treatment: Clinical Uses of Hypnosis.* New York, Basic Books, 1978.

Task Force Reports of the Biofeedback Society of America, *Biofeedback Self Regul* **3**(4), 1978.

Drugs That Affect Behavior

1. A 20-year-old single woman who works as a secretary complained of a severe headache. Another secretary working in the same office gave her two aspirin tablets. When her headaches returned in two hours, she bought a bottle of aspirin and took three tablets. She did not have lunch because she felt sick, but drank cup after cup of black coffee. Later, her supervisor advised her to go home and rest when she saw her taking another three aspirin tablets. At home, she continued to feel sick with both headache and nausea, but she could not rest because she had an important date with a new boyfriend. She took four more aspirins. She was brought to the hospital emergency room by her boyfriend when she developed severe nausea, abdominal pains, and dizziness in the evening.

2. "A man with advanced lymphosarcoma was included in an experimental study of the since-discredited drug Krebiozen. After one administration, his tumors disappeared. When reports came out that the drug was ineffective, the doctor told the patient not to believe what he read and treated him with 'double strength' Krebiozen—actually an injection of water. The patient again experienced rapid remission. Then the AMA and FDA pronounced the drug worthless. The patient died within a few days."*

3. A 40-year-old housewife had been complaining for several years of shortness of breath and easy fatigability. She would not consult a doctor, however, and would demand attention from her husband and children. Eventually, her family treated her like an invalid, with a certain amount of contempt. She gained weight progressively and reduced her activity level. A few weeks ago, her dyspnea became so severe that she agreed to consult a physician.

---

* From Holden (1978). Quoted with permission. The vignette is based on a case history reported by Klopfer (1957).

401

Rheumatic mitral valve disease was diagnosed, and she was placed on a regimen of digitalis. Her dyspnea disappeared, and she lost weight dramatically. (The weight gain was largely edema—retention of fluids.) She again felt energetic and resumed her normal activities as an efficient housewife. Her relationship with family members improved and so did her sex life with her husband. She also became socially active again.

4. A 19-year-old single man was admitted to the psychiatric ward of the hospital. For the last several weeks, he had become increasingly withdrawn; he would stay up all night in his room, ostensibly listening to music, and sleep all day long. On the night of admission, his parents found him in his room, screaming at an imaginary person. He was quite incoherent and would not respond to his parents. On the psychiatric unit, he was given an intramuscular injection of chlorpromazine, after which he fell asleep. The following morning, the staff was able to converse with him rationally, although he would lapse into incoherent language from time to time. He was placed on a regular regimen of chlorpromazine.

5. A 60-year-old widow was admitted to the hospital because of severe low back pain. She appeared quite depressed and tearful on admission. History revealed that she had been diagnosed as having a depressive syndrome (see Chapter 6) three years ago, and antidepressant medication had been prescribed. She was "continued" on the antidepressant drug in the hospital. Within ten days, she was much brighter, her tearfulness and hopeless feelings abated, and she slept better. Subsequently, it was found that she had not been taking the antidepressant medication regularly at home because she felt that she needed to take the medication only when she felt extremely depressed. Thus, after receiving again a regular regimen of antidepressants in the hospital, she showed dramatic improvement.

These vignettes illustrate the multiple effects of drugs in the three major dimensions. For example, aspirin taken for the relief of headache (vignette 1) was probably associated with the following sequence of events:

1. Salicylate, by inhibiting prostaglandin synthesis, reduces stimulation of pain receptors and, in addition, acts directly on certain brain areas involved in the processing of pain sensation. These actions affect biological components of the pain and lead to . . .
2. Relief from the experience of headache (personal dimension), which leads to . . .
3. The patient's buying a full bottle of aspirin (environmental–interpersonal interaction) when the headache returns. The association of aspirin with relief from headaches is a learned behavior (personal dimension).
4. The patient takes more aspirin, which results in irritation and possible ulceration of the gastroduodenal mucosa (biological).

5. Increased consumption of coffee (personal), perhaps related to anxiety and discomfort associated with interpersonal stress (personal, environmental), contributes further to the gastrointestinal irritation (biological).
6. The observation by her supervisor of her illness behavior (personal) and the aspirin-taking leads to her going home early (environmental change).
7. The date with her boyfriend increases her anxiety (personal), perhaps aggravating the headache, which again leads to increased drug-taking, ultimately resulting in . . .
8. Acute gastrointestinal disturbance and mild salicylate toxicity (biological), leading to hospitalization (environmental).

Drugs Affect All Dimensions of the Patient

Drugs are usually administered with the intent of producing a particular effect in the biological or personal dimension. We may forget that drugs also have effects in dimensions other than those we have in mind. As illustrated in vignette 1, drug effects occur in many subsystems and in all dimensions: behavior of the person, of intracellular processes in neurons, and of other persons and systems in the environment.

The *pharmacological action* of a drug is usually mediated by effects in the biological subsystem, and these often lead to changes that are perceived in the personal dimension. The analgesic effect of aspirin is an example. Sometimes a drug may simultaneously cause desirable changes in the biological dimension and undesirable changes in the personal dimension. Such undesirable side effects may be due to the pharmacological effects of the drug on the central nervous system or to psychological responses to the drug's *symbolic meaning*. Patients with pain who do not respond to methadone, a potent analgesic, may ascribe conflictual symbolic meaning to this drug, which is also used for the management of addiction.

Most drugs have some pharmacological central nervous system action. The physician must keep informed about the central nervous system effects of medications that he prescribes; patients are often concerned about how a drug will make them feel.

As would be expected, there may be complex interactions between drug actions, drug-taking behavior, and the patient's personality (see Chapter 17). Certain patients (e.g., with dependent, demanding personalities) may consider drugs as tangible evidence of the doctor's caring, while others (e.g., with orderly, controlling personalities) may view

the taking of drugs as a threat to their autonomy. Guarded, suspicious patients may become distrustful of prescribed drugs, suspecting that they might be harmful—even poisonous—especially if unexpected side effects occur and if the doctor–patient relationship is less than optimal. Sedating drugs may be especially threatening to patients who have concerns about autonomy. Patients with long-suffering, self-sacrificing personalities who are "addicted" to the sick role may feel threatened by drugs that promise "cure." Patients with impulsive personalities may be erratic in drug-taking and may unexpectedly abuse or overdose on drugs. Patients with chronic memory deficits may not be able to self-administer drugs because they forget the dosage and schedule and when the medication was last taken. Serious accidental overdosage may occur in these patients.

It is important to remember the *placebo effect* in prescribing any drug. Placebos may act as powerful agents, exerting their effects through as yet incompletely understood central nervous system mechanisms associated with the psychological phenomenon of expectancy. The effects of placebos attest to the powerful influence that the brain can sometimes exert on disease processes and the experience of illness (see vignette 2; see also Chapters 9 and 16 for further discussion of the placebo effect).

Drug effects that may be observed in the *environmental dimension* of the patient may be mediated in several ways. Some drugs cause changes in the *appearance* and *social attractiveness* of the patient, for example, staining of skin by tar ointments used in some dermatological conditions, hair loss caused by cytotoxic agents, and the smell of paraldehyde. The social relationships of patients receiving these drugs may become restricted. Some drugs are used primarily to change the *physical environment,* such as antiseptics in the operating room and insecticides in the home. Antibiotics used indiscriminately may foster the development of drug-resistant strains of bacteria that are dispersed into and contaminate the environment.

In addition to direct effects on the environment, social attitudes toward certain drugs may indirectly influence the behavior of the patient's family and friends. For example, a patient who requires methadone for pain may be stigmatized because of the social symbolic association of methadone with addiction. A patient under "chemotherapy" may be treated by his friends or relatives in accordance with their ideas of how a *cancer patient* should be treated (whether or not the chemotherapy is in fact being given for cancer).

Other indirect effects of drugs in the environmental dimension can be mediated by changes in the *patient's behavior.* A medication causing a depressive syndrome as a side effect may indirectly lead to isolation

of the patient from environmental supports because of social withdrawal; an antidepressant medication, on the other hand, may increase the patient's interaction with the environment.

To reiterate, any drug, no matter what its intended pharmacological action might be, can exert effects in all dimensions—directly, through specific pharmacological actions or symbolic meaning, and, indirectly, through induced changes in behavior.

Another important consideration to bear in mind is that *prescription* (or ordering) of a drug does *not* ensure the taking of the drug. One quarter to one half of all outpatients do not take the prescribed medication at all (Blackwell, 1973)! Factors that influence patient compliance with a medication regimen include the patient's understanding of the need for the drug, the duration and schedule of administration, perceived effects and side effects, the symbolic meaning of the medication, the patient's personality, and the status of the doctor–patient relationship (see Chapter 16). In the presence of a good doctor–patient relationship, patients are willing to continue drugs even if they do not seem to have an immediate beneficial effect or despite uncomfortable side effects. Open, comfortable communication between the patient and the doctor will enable the patient to talk to the physician about side effects of the medication rather than to discontinue it without telling the doctor.

Even the *shape, size,* and *color* of medications have important effects on the likelihood of their being taken and on the safety of their use (Mazzullo, 1972). For example, pills of similar size and color can be confused, resulting in an overdosage of one of them. A larger pill often seems stronger to a patient than a smaller pill, regardless of its actual strength.

In the hospital, drugs are usually administered by a nurse in response to a physician's written order, and patients are generally not informed about the medication being given by the nurse. Even when the physician writes an order to continue the same medication the patient had been taking prior to admission, the hospital pharmacy may stock a different brand with a different shape and color. Difficulties and confusion can be avoided by discussing medications with the patient—those to be continued, whether the shape and color will be different, and whether new medications will be added.

Psychotropic Drugs

Some drugs are used primarily to change the patient's mood, thought processes, or behavior. Those medications that have their pri-

mary pharmacological action on the central nervous system and that are used to produce changes in mood, thought processes, or behavior are called *psychotropic* drugs. It should already be obvious from the preceding section that psychotropic drugs are *not* the only medications that may have such effects. The psychotropic drugs, however, may be narrowly defined as a subset of drugs that act primarily and selectively on the central nervous system to produce relatively specific effects rather than general central nervous system stimulation or depression.

Psychotropic drugs may be broadly classified into (1) antianxiety drugs, (2) drugs that affect mood (antidepressants and lithium salts), (3) antipsychotic drugs, and (4) psychotomimetic drugs (those that cause psychosis-like syndromes).

We will discuss very briefly general principles concerning the use of the first three classes of psychotropic drugs in clinical management. For detailed information, the reader should consult standard textbooks of pharmacology and psychiatry.

Antianxiety Drugs (Minor Tranquilizers)

As discussed in Chapter 4, anxiety is usually a signal of impending danger that may either be external or originate within the personality system of the patient. Ideally, the best treatment of anxiety is removing or avoiding the danger situation rather than decreasing the danger signal. Many anxiety situations are not amenable to relief by immediate intervention, but may subside or undergo resolution given adequate time. It may be necessary and desirable in such cases to reduce overwhelming anxiety and so reduce also the strength of associated physiological reactions that may themselves have pathogenic effects (see Chapter 4). Especially in hospitalized patients, excessive anxiety may interfere with recovery (e.g., in patients with myocardial infarction). As noted earlier, hospitalization and medical procedures in and of themselves almost always induce and aggravate anxiety.

Drugs commonly prescribed for their antianxiety effects include benzodiazepines, barbiturates, propanediol carbamates, antihistaminics, and antipsychotic medications in small doses.

Benzodiazepines such as chlordiazepoxide (Librium), diazepam (Valium), and alprazolam (Xanax) (see also p. 409) are the most commonly used and abused. Benzodiazepines bind to specific benzodiazepine receptors, which are functionally linked to the γ-aminobutyric acid (GABA) recognition sites, and form a GABA–benzodiazepine–chloride ionophore complex (Squires and Braestrup, 1977; Snyder *et al.*, 1977;

Table 18. Commonly Prescribed Benzodiazepines in the United States[a]

Drug	Approximate dose equivalence (mg)	Relative rapidity of effect	Active metabolites	Half-life[13] (hr)
Chlordiazepoxide (Librium)	10	Intermediate	Yes	5–30
Clorazepate (Tranxene)	7.5	Fast	Yes	30–200
Diazepam (Valium)	5	Fastest	Yes	20–100
Lorazepam (Ativan)	1	Intermediate	No	10–20
Oxazepam (Serax)	15	Slower	No	5–15
Prazepam (Centrax)	10	Slowest	Yes	30–200

[a] From Rosenbaum (1982). Reprinted with permission.

Stein *et al.*, 1977; Hoehn-Saric, 1982). Benzodiazepines potentiate GABA, which in turn decreases neuronal activity by opening chloride channels that are directly linked to the GABA receptors. The resultant influx of chloride ions hyperpolarizes the cell and renders it less excitable.

Benzodiazepines are effective in reducing anxiety but may be habit-forming and, in some individuals, may produce paradoxical agitation, confusion, or fatigue. Benzodiazepines can also be used as hypnotics (to induce sleep); they do not affect REM but decrease stage 4 sleep. They also increase the seizure threshold. See Table 18 for a list of commonly used benzodiazepines.

Antipsychotic drugs (discussed below) also have antianxiety action and can be used in small doses (about one tenth of the antipsychotic dose) to treat anxiety in nonpsychotic patients. One should be aware of infrequent but serious side effects of antipsychotic agents, such as tardive dyskinesia, when one considers using them (see below).

Barbiturates and *propanediol carbamates* (e.g., tybamate and meprobamate) are similar in action to benzodiazepines, but perhaps more sedating and less specifically "anxiolytic." *Antihistaminics* are primarily sedating.

In addition to the drugs mentioned above, *narcotic analgesics* such as morphine are anxiolytic, especially in patients with severe pain or other physical distress such as shortness of breath. For such patients, adequate analgesia is also adequate treatment of anxiety.

A general principle in the use of antianxiety agents is that they should be used intermittently and for short periods of time because of their addictive potential. Concurrent comprehensive evaluation of the danger situation or a medical condition that may cause anxiety symptoms (Chapter 4) should be carried out, and the possibility of referral

for counseling or psychotherapy should be considered. Self-control techniques of anxiety reduction, such as self-hypnosis, relaxation exercises, and pursuing hobbies, are sometimes helpful and should also be considered.

Drugs That Affect Mood

Antidepressants. Drugs used in the treatment of the depressive syndrome are called antidepressants. When a depressed patient also shows signs of psychotic thought disorder (such as delusions), antipsychotic agents are often used in addition to antidepressants.

The antidepressants are not necessarily central nervous system stimulants. When an antidepresant is given to a normal person, the effect is usually sedation and drowsiness rather than euphoria. When it is administered to a patient with the depressive syndrome (see Chapter 6), in 80–90% of cases, there will be clear improvement, usually manifest in about two weeks' time, and often dramatic improvement by four to six weeks.

There are two classes of antidepressants: the monoamine oxidase (MAO) inhibitors and the tricyclic antidepressants. Both classes of drugs ultimately seem to cause an increase in the functional levels of biogenic amines in the brain, especially norepinephrine and serotonin. The MAO inhibitors are believed to act by reducing the intraneuronal breakdown of biogenic amines by MAO.

The tricyclic antidepressants were initially thought to work primarily by inhibiting the reuptake of norepinephrine or serotonin or both at the nerve terminals, thus increasing their functional availability at the synapse. More recently, however, the modulating effect of tricyclic and other antidepressant drugs on the biogenic amine *receptor sensitivity* has received much attention. Long-term treatment with tricyclic antidepressants, MAO inhibitors, and electroshock therapy has consistently resulted in an enhancement of responses to serotonergic and α_1-adrenergic stimulation through development of postsynaptic receptor supersensitivity and a reduction in the sensitivity of the β-adrenergic receptors (Charney *et al.*, 1981a). Charney *et al.* (1981b) proposed, on the basis of these findings, that the changes in postsynaptic receptor sensitivity may be the "final common pathway" of antidepressant action. It is generally believed currently that the ultimate effects of antidepressant therapy involve a normalization of the dysregulation of the various biogenic amine systems involved in mood modulation [see Figures 10 and 12 (Chapter 6)].

Tricyclic antidepressants: Imipramine, desipramine, amitriptyline, protriptyline, and doxepin are examples of tricyclic antidepressants. Imipramine (Tofranil) seems to have a greater effect on norepinephrine-containing neurons, while amitriptyline (Elavil) has a greater effect on serotonergic neurons in usual therapeutic doses (Maas, 1978).

Tricyclic antidepressants have a number of *side effects,* especially due to their *anticholinergic* effects. They include dry mouth, blurry vision, urinary retention, tachycardia, and orthostatic hypotension.

Tricyclic antidepressants do not seem to increase the risk of cardiac arrhythmias except in overdoses (Boston Collaborative Drug Surveillance Program, 1972). In fact, tricyclics seem to have quinidinelike antiarrhythmic effect (Kantor *et al.*, 1978). Amitriptyline has the most anticholinergic effect, desipramine the least.

Tricyclic antidepressants are also *sedating,* so that a hypnotic drug may not be necessary if the antidepressant is given at night. Tricyclics increase delta sleep and decrease REM. Amitriptyline appears to be more sedating than imipramine. Protriptyline seems to have little sedative property.

Tricyclic antidepressants interact with a number of other medications. For example, they reduce the potency of antihypertensive agents such as guanethidine by interfering with their uptake by the peripheral adrenergic neurons. Tricyclics should not be given with MAO inhibitors because of possible severe hypertensive crisis.

Tricyclic antidepressants are generally nonaddicting, and tolerance to the antidepressant effect does not develop, although the anticholinergic effects do become better tolerated.

Tricyclic antidepressants are toxic in large doses, primarily due to the anticholinergic effects and the cardiotoxic effect of an increase in the catecholamines in cardiac tissue. In prescribing tricyclic antidepressants, the possibility of overdose with suicidal intent should be considered.

In addition to treatment of depression, tricyclic antidepressants may be used to treat enuresis in childhood, severe obsessive–compulsive disorders, as well as phobic anxiety and panic states.

Maprotiline (Ludiomil) is a relatively newly marketed tetracyclic compound and Trazodone (Desyrel) is a phenylpiperazine derivative. Both seem to have antidepressant actions and side effects similar to those of the tricyclics, except that their anticholinergic action seems to be less prominent (Hollister, 1981).

Alprazolam (Xanax) is a benzodiazepine that has both antianxiety and mild antidepressant action.

MAO inhibitors: Phenelzine (Nardil), tranylcypromine (Parnate), and isocarboxazid (Marplan) are examples of MAO inhibitors. Unlike

tricyclic antidepressants, MAO inhibitors, especially tranylcypromine, may have amphetaminelike central-nervous-system-stimulating properties in addition to their antidepressant effect. The antidepressant effect of MAO inhibitors is considered to be related to the drug's entering into stable combination with the enzyme MAO, thereby irreversibly inhibiting its action.

MAO inhibitors are potent REM suppressants. They can also lower blood pressure, although their anticholinergic effects are much less potent than those of the tricyclic antidepressants (Snyder, 1977).

A major consideration in the use of MAO inhibitors is that they *interact* with a number of *foods* and *beverages*, as well as with other medications. In general, any sympathomimetic drugs and foods containing sympathomimetic amines, particularly *tyramine*, can cause a *hypertensive crisis* in conjunction with an MAO inhibitor. Cerebrovascular accidents may occur during the hypertensive crises. Foods containing tyramine include cheese, beer, wine, pickled herring, chicken liver, aged meats, sausage, broad-bean pods, coffee, yeast, and canned figs. Many nonprescription drugs contain sympathomimetic agents. MAO inhibitors potentiate the effects of biogenic amine precursors like L-dopa. *Meperidine* is *contraindicated* and can cause marked hyperpyrexia. MAO inhibitors can interfere with the detoxification mechanisms of many drugs, including barbiturates, alcohol, anticholinergics, and tricyclic antidepressants. Patients on MAO inhibitors should be cautioned not to take any other medication without consulting the physician and should be given a *list of foods and medications* that should not be taken concurrently.

There is some evidence that "atypical" depressives who do not show a classic depressive syndrome but whose depression is severe enough to warrant drug treatment may benefit from MAO inhibitors more than from tricyclic antidepressants. This population includes patients with depressive and hypochondriacal symptoms (Robinson *et al.*, 1978). In view of the myriad interactions with foods and other drugs, however, treatment with MAO inhibitors should be considered with extreme caution and used only when the physician is satisfied that there will be full compliance with the instructions.

In addition to treatment of depression, tricyclics and MAO inhibitors have been used to treat panic disorders, bulimia, and narcolepsy (utilizing the REM-suppressant action).

Lithium Salts. Lithium salts were used in the 1940s as a salt substitute for cardiac patients. Deaths occurred due to lithium poisoning, and the use of lithium salts was discontinued until the reintroduction of lithium as an antimanic drug in the 1960s. Lithium is currently used as a car-

bonate (Li$_2$CO$_3$). Lithium carbonate is effective in the *treatment of acute mania* (improvement in 70–80% of patients within 10–14 days) and as a maintenance drug in the *prevention* of manic attacks (Goodwin and Ebert, 1973). Lithium is also effective as a maintenance drug in reducing the number and intensity of recurrent unipolar depressive episodes (Baldessarini and Lipinski, 1975). This may be especially helpful in patients who have already shown recurrent episodes of depression (see Chapter 6).

Lithium carbonate may also be used to augment the antidepressant effect of a tricyclic or an MAO inhibitor drug in patients with refractory depression (De Montigny *et al.*, 1981). Dramatic improvements have been reported within two to three days of the initiation of lithium in patients who had been receiving an antidepressant drug without much effect.

The mechanism of action of lithium seems to be attributable to its being an "imperfect substitute" for other cations such as sodium and potassium and to its alteration of the intracellular microenvironment necessary for hormone action (Singer and Rotenberg, 1973). The latter involves interference with the hormone activation of adenyl cyclase or the action of cyclic AMP.

How lithium reverses and prevents mania and how it prevents depressive episodes are not completely known. Lithium may correct the tendency toward increased intracellular sodium concentration in affective disorders (see Chapter 6). There is evidence that lithium may exert antagonistic actions at catecholamine-mediated synapses in the brain. On a short-term basis, lithium increases serotonin turnover in the brain. With chronic administration, however, the turnover rate becomes normalized. Lithium initially increases norepinephrine turnover in rat brain, but on a long-term basis, the norepinephrine synthesis is normal. Lithium may increase the intraneuronal release of norepinephrine to be metabolized by MAO and decrease its extraneuronal release. Lithium inhibits basal and norepinephrine-activated adenyl cyclase (Gerbino *et al.*, 1978).

Lithium may also decrease the synthesis and release of acetylcholine and affect the metabolism of GABA and glutamates in the brain (Baldessarini and Lipinski, 1975). The overall clinical effect of lithium seems to be stabilization of moods.

In using lithium carbonate, it is necessary to *monitor* the plasma *lithium concentration* on a regular basis. The therapeutic range is 1.0–1.5 mEq/liter initially, and for maintenance, 0.6–1.0 mEq/liter. The plasma level of lithium should *never* exceed 2 mEq/liter! The blood level should be drawn preferably 12 hr after ingestion of the last dose. Lithium should

be administered in divided doses to maintain, as much as possible, steady blood levels.

Lithium can cause fatigue and muscular weakness. Toxic signs include nausea, vomiting, diarrhea, slurred speech, and ataxia. Other side effects include nephrogenic diabetes insipidus, goiter with hypothyroidism, and EEG changes.

Lithium should be administered *with extreme caution* to patients on a sodium-restricted diet, because lithium excretion is slow and severe toxicity may occur. Lithium decreases the pressor effect of norepinephrine.

Antipsychotic Drugs (Major Tranquilizers, Neuroleptics)

A number of drugs are now available for the treatment of psychotic states, including *schizophrenia.* They generally fall into one of the following categories: phenothiazines, butyrophenones, thioxanthines, dibenzodiazepines, and rauwolfia alkaloids.

The phenothiazines are representative of the antipsychotics. Synthesized in 1883, phenothiazine was first used in the 1930s as an antihelminthic, urinary antiseptic, and insecticide. Promethazine, a phenothiazine, was used as an antihistamine, sedative, and, in the early 1950s, as premedication for anesthesia. The introduction of chlorpromazine as an antipsychotic agent in the late 1950s and its wide acceptance in the 1960s revolutionized psychiatric treatment for schizophrenia.

All drugs that belong to the category of antipsychotic agents have, to a greater or lesser extent, certain features in common, as represented by chlorpromazine.

Central Nervous System Effects. All antipsychotics except rauwolfia alkaloids *block dopamine receptors* in the brain [see Figure 11 (Chapter 6)]. The dopamine-receptor blockade in the dopaminergic mesolimbic system by the antipsychotics is considered to be the main mechanism of the antipsychotic effect. Dopaminergic blockade in the nigrostriatal tract by the antipsychotic agents can result in extrapyramidal symptoms, including parkinsonian features, dystonias, and the serious side effect of *tardive dyskinesia.* Tardive dyskinesia is a syndrome that is marked by characteristic involuntary movements of the mouth, tongue, and other parts of the body. It is considered to be due to the development of "receptor hypersensitivity" of the postsynaptic neurons (see the section on Behavioral Effects). Dopaminergic blockade is also probably respon-

sible for the increase in prolactin levels in patients receiving antipsychotics, since dopamine normally inhibits prolactin release from the pituitary. Increased prolactin levels may cause gynecomastia and lactation. Rauwolfia alkaloids such as reserpine probably exert antipsychotic action through depletion of biogenic amines in the neurons, including dopamine, norepinephrine, and serotonin. Antipsychotic agents also block the reuptake of norepinephrine and serotonin in the brain, but do not affect GABA.

At the hypothalamic level, antipsychotic agents inhibit the release of growth hormone and may antagonize the release of the hypothalamic prolactin-release-inhibiting hormone. Hypothalamic temperature regulation may be interfered with by antipsychotics, and *hypothermia* may ensue.

Antipsychotics seem to increase the "filtering activity" of the reticular activating system in the brain stem, reducing the inflow of stimuli in a selective fashion [see Figure 7 (Chapter 4)]. Antipsychotics have varying degrees of antiemetic action through an action on the chemoreceptor trigger zone. Chlorpromazine (Thorazine) and prochlorperazine (Compazine) have relatively high antiemetic action.

Antipsychotic agents slow the EEG pattern but also *lower the seizure threshold*. Drug-induced seizures are common among patients with preexisting seizure disorders or with a tendency to seizures.

Autonomic Nervous System Effects. Antipsychotic agents have varying degrees of *anticholinergic* activity, *anti-α-adrenergic* activity (direct receptor block), *adrenergic* activity (through blockade of reuptake of catecholamines), and *antihistaminic* activity. Chlorpromazine may reverse the pressor effects of epinephrine. Postural hypotension occurs more commonly with chlorpromazine than with more potent (and thus used in small doses) piperazine-ring phenothiazines such as fluphenazine or butyrophenones. Thioridazine (Mellaril) may cause ejaculatory incompetence in male patients. In high doses, thioridazine may also cause retinitis pigmentosa.

Behavioral Effects. Antipsychotic drugs have a *specific effect* on psychotic individuals in reversing or reducing the psychotic symptomatology, including the thought disorder, autism, hallucinations, and paranoid ideations. Agitation or belligerence is also reduced. These effects tend to occur over the course of several weeks in schizophrenia. Psychotic states due to any disease process, including organic psychosis as well as schizophrenia, often respond to antipsychotics.

In addition to the specific antipsychotic effect, the antipsychotic agents also have varying degrees of *sedative action*. Characteristically, the sedation caused by antipsychotic agents is accompanied by a feeling of indifference to environmental events and psychomotor slowing. This phenomenon has been called the "neuroleptic syndrome," and the antipsychotics, *"neuroleptic drugs."* Antipsychotic drugs are also called "major tranquilizers," in comparison to the antianxiety agents like benzodiazepines, which are called "minor tranquilizers."

In experimental animals, antipsychotic agents impair *conditioned avoidance learning*. This test is often used for screening drugs that might possess antipsychotic effects. Minor tranquilizers do not have this effect. Sleep caused by a sedating antipsychotic drug such as chlorpromazine is easily arousable.

All antipsychotic agents reduce spontaneous *motor activity* in humans and animals. In large doses, this blends into the *parkinsonian* picture of rigidity, loss of associated movements, and tremors. Some patients may develop *catalepsy* ("waxy flexibility") with the administration of large doses of antipsychotics. *Akathisia* is an extrapyramidal symptom caused by some antipsychotics. It is characterized by a marked increase in motor activity and restlessness and a literal inability to sit still. This as well as other extrapyramidal symptoms can be effectively reversed with anticholinergic agents or antihistaminic agents such as benztropine (Cogentin) or diphenhydramine (Benadryl). A serious side effect with prolonged use is *tardive dyskinesia*. This syndrome is often irreversible.

The side effects of antipsychotics, in addition to those already mentioned, include hepatic microsomal enzyme induction, obstructive jaundice, cardiac arrhythmias, EEG changes, blood dyscrasias, photosensitivity, and skin rash.

Indications and Precautions. In the presence of a psychotic state, antipsychotic agents are indicated to control the psychotic symptoms. In psychosis associated with an underlying medical disease [(see Tables 6 (Chapter 7) and 16 (Chapter 12)], treatment of the underlying disease should accompany symptomatic treatment of the psychosis with antipsychotic agents. The choice of drug depends, to a large extent, on the side effects to be avoided or taken advantage of. For example, an agitated patient might receive a more sedating phenothiazine such as chlorpromazine, while a psychomotor-retarded, depressed, psychotic patient might do better with a less sedating drug. For details concerning the side effects of individual antipsychotic drugs, the reader, as noted earlier, is referred to standard textbooks of psychiatry and pharmacology.

Summary

Although drugs are usually administered to effect a particular change in the biological or personal dimension, all three dimensions, including the environmental–social dimension, are affected by drugs.

The pharmacological action of a drug is usually mediated by changes in the *biological dimension,* which may eventually cause changes in the *personal dimension* (e.g., analgesia). The personal dimension may be affected, additionally, by the *symbolic meaning* of the drug. The *placebo effect* is a potent and often beneficial effect that accompanies the use of any drug and may augment its action. The *environmental dimension* of the patient may be affected by drugs directly (e.g., foul odor of the breath of a patient taking paraldehyde) or indirectly (through the symbolic meaning of the drug to others or through the behavioral change of the patient).

Prescribing a drug is *not* equivalent to its actual administration. Up to one half of outpatients do not take prescribed drugs. The factors that affect *compliance* with a drug regimen include (1) the quality of the doctor–patient relationship, (2) the shape, size, and color of the drug, (3) the patient's information about the drug, and (4) the personality of the patient.

Psychotropic medications are drugs the primary pharmacological action of which is on the central nervous system to produce relatively selective changes in mood, thought processes, or behavior. They include *antianxiety drugs, drugs that affect mood, antipsychotic drugs,* and *psychotomimetic drugs.*

The *benzodiazepines* are the most commonly used *antianxiety* agents. While they are usually effective, they may produce paradoxical agitation in some individuals. They increase seizure threshold and reduce delta sleep. They are also sedating and may be used as hypnotics (especially flurazepam). The benzodiazepines are habit-forming with prolonged use. Antianxiety agents should be used judiciously and, generally, temporarily.

The *tricyclic* antidepressants *imipramine* and *amitriptyline* are the most commonly used antidepressants. The tricyclics seem to block the reuptake by the presynaptic membrane of released norepinephrine or serotonin or both, which causes an increase in the functional levels of these amines at the synapse. In addition, their long-term effects include an enhanced sensitivity of postsynaptic serotonin and α_1-receptors and reduced sensitivity of β-receptors.

The antidepressants require time to work fully; usually, at least 10–14 days are necessary before the effects are manifested. The most common side effects of tricyclics are sedation and anticholinergic effects.

There are many drug interactions with *monoamine oxidase inhibitors*, and many foods, especially those containing the pressor amine tyramine, interact with the MAO inhibitors, causing serious hypertension. Meperidine is contraindicated in a patient receiving an MAO inhibitor.

Lithium carbonate is effective as an *antimanic* agent and also in preventing recurrent unipolar depressions. The plasma lithium level must be monitored in patients receiving lithium. The therapeutic range is 0.6–1.5 mEq/liter. Lithium should be administered with extreme caution to patients on sodium-restricted diets.

The *phenothiazines* and *butyrophenones* are representative *antipsychotic* agents. Their action seems to be the blockade of dopamine receptors, and they appear to increase the filtering activity of the reticular activating system. They tend to be sedating, and some have antiemetic action. They lower the seizure threshold and impair conditioned learning in animals. Autonomic side effects include orthostatic hypotension and anticholinergic effects. Neurological side effects include pseudoparkinsonism, akathisia, and tardive dyskinesia.

Implications

For the Patient

Properties other than the specific pharmacological action of a drug may be of primary importance to the patient, for example, the size, color, shape, and name of the drug as well as the price. The symbolic meaning of the medication also plays an important role in the patient's perception and expectations of a drug. Many patients do not comply with a drug regimen. This may be due to a number of reasons, the most important being the lack of a trusting doctor–patient relationship and associated lack of communication. The side effects of medications, although not alarming to the doctor, may be quite frightening to the patient if unexpected. Some patients attach extraordinary significance and importance to taking drugs and may become dependent on them.

For the Physician

Physicians should recognize that drugs affect all dimensions of the patient, regardless of the desired specific pharmacological action. In dis-

cussing and prescribing a medication, the physician should take into account the patient's personality type, previous experience with drugs, and expectations and symbolic meanings of the proposed drug. Specific questions must be asked to obtain such information. Good compliance with a drug regimen rests on a good doctor–patient relationship and an open channel of communication. The significance of the nonpharmacological aspects of drugs, such as the size, shape, and color of a pill, should be recognized and understood by the physician. Explanation concerning possible side effects of a medication is essential in reassuring the patient and maintaining a good doctor–patient relationship. The physician should be familiar with the effects of any planned medication in all dimensions of the patient and also with its interaction with other drugs and substances (e.g., tyramine-containing foods and MAO inhibitors).

For the Community and the Health-Care System

The community should be educated about the fact that drugs have many actions, some of which may be dangerous in themselves or in interaction with other drugs and substances. The public should learn to use medications only in consultation with a physician. Medical education should emphasize that any drug affects all three dimensions of the patient. Emphasis should also be placed on the nonpharmacological aspects of drugs, such as the shape, size, color, name, and symbolic meaning. Unrealistic and exaggerated claims by some pharmaceutical companies concerning their products should be regulated.

Recommended Reading

Goodman LS, Gilman A: *The Pharmacological Basis of Therapeutics.* New York, Macmillan, 1980. This is the standard textbook of pharmacology. We would especially suggest the chapter on drugs and the treatment of psychiatric disorders for a more detailed treatment of the subject. An excellent reference for all questions concerning drugs.

Hansten PD: *Drug Interactions.* Philadelphia, Lea & Febiger, 1976. (Paperback.) A very useful reference. Various drug interactions are presented in a tabular form.

Jarvik ME (ed): *Psychopharmacology in the Practice of Medicine.* New York, Appleton-Century-Crofts, 1977. A multiauthor volume. Unlike that edited by Lipton *et al.* (see below), this is oriented more toward the primary physician. Concise but comprehensive.

Lipton MA, DiMascio A, Killam KF (ed): *Psychopharmacology: A Generation of Progress.* New York, Raven Press, 1978. An up-to-date, comprehensive, and authoritative multiauthor book on various aspects of psychopharmacology, including basic biochemistry,

neurophysiology, neurotransmitters, animal behavior pharmacology, and clinical psychopharmacology. An excellent reference book for the relatively advanced reader. The book is huge (1731 pages, including index).

References

Boston Collaborative Drug Surveillance Program report: Adverse reactions to the tricyclic antidepressant drugs. *Lancet* 1:529–31, 1972.

Baldessarini RJ, Lipinski JF: Lithium salts: 1970–1975. *Ann Intern Med* 83:527–533, 1975.

Blackwell B: Drug therapy: Patient compliance. *N Engl J Med* 289:249–252, 1973.

Charney DS, Menkes, DB, Heninger GR: Receptor sensitivity and the mechanism of action of antidepressant treatment. *Arch Gen Psychiatry* 38:1160–1180, 1981a.

Charney DS, Heninger GR, Sternberg DE, *et al.*: Presynaptic adrenergic receptor sensitivity in depression: The effect of long-term desipramine treatment. *Arch Gen Psychiatry* 38:1334–1340, 1981b.

De Montigny C, Grunberg F, Mayer A, *et al.*: Lithium induces rapid relief of depression in tricyclic antidepressant drug nonresponders. *Br J Psychiatry* 138:252–256, 1981.

Gerbino L, Oleshansky M, Gershon S: Clinical use and mode of action of lithium, in Lipton MA, DiMascio A, Killam KF (eds): *Psychopharmacology: A Generation of Progress.* New York, Raven Press, 1978, pp 1261–1275.

Goodwin FK, Ebert MH: Lithium in mania, in Gershon S, Shopsin B (eds): *Lithium: Its Role in Psychiatric Research and Treatment.* New York, Plenum Press, 1973, pp 237–252.

Hoehn-Saric R: Neurotransmitters in anxiety. *Arch Gen Psychiatry* 39:735–742, 1982.

Holden C: Cancer and the mind: How are they connected? *Science* 200:1369, 1978.

Hollister LE: Second generation antidepressant drugs. *Psychosomatics* 22:872–879, 1981.

Kantor SJ, Glassman AH, Bigger JT, *et al.*: The cardiac effects of therapeutic plasma concentrations of imipramine. *Am J Psychiatry* 135:534–538, 1978.

Klopfer B: Psychological variables in human cancer. *J Project Tech* 21:331–334, 1957.

Maas JW: Clinical implications of pharmacological differences among antidepressants, in Lipton MA, DiMascio A, Killam KF (eds): *Psychopharmacology: A Generation of Progress.* New York, Raven Press, 1978, pp 955–960.

Mazzullo J: The nonpharmacologic basis of therapeutics. *Clin Pharmacol Ther* 13:157–158, 1972.

Robinson DS, Nies A, Ravaris CL, *et al*: Clinical pharmacology of phenelzine. *Arch Gen Psychiatry* 35:629–635, 1978.

Rosenbaum J: The drug treatment of anxiety. *N Engl J Med* 306:401–404, 1982.

Singer I, Rotenberg D: Mechanisms of lithium action. *N Engl J Med* 289:254–260, 1973.

Snyder SH: Antidepressants and the muscarinic acetylcholine receptor. *Arch Gen Psychiatry* 34:236–239, 1977.

Snyder SH, Enna SJ, Young AB: Brain mechanisms associated with therapeutic actions of benzodiazepines: Focus on neurotransmitters. *Am J Psychiatry* 134:662–665, 1977.

Squires RF, Braestrup C: Benzodiazepine receptors in rat brain. *Nature (London)* 266:732–734, 1977.

Stein L, Belluzzi JD, Wise CD: Benzodiazepines: Behavioral and neurochemical mechanisms. *Am J Psychiatry* 134:665–669, 1977.

Some Illustrative Patients

In this chapter, we will present the evaluation and management of three illustrative patients, beginning with the case of the "sick Tarzan," whose history and interview were presented in Chapter 15. All the cases presented in this chapter, including the "sick Tarzan," are actual case histories. Of course, some identifying data have been changed for reasons of confidentiality.

Management of the "Sick Tarzan"*

We are now prepared to formulate a comprehensive three-dimensional management plan for our patient, armed with information and insights from all our previous chapters. In rereading the case history, it should become clear that there is a discrepancy in the model of illness between the patient and the doctor. For example, "My doctor thinks I had a heart attack. All I need is to get back my strength, which I lost from being in here too long." In fact, the patient believes, "Once you start laying in bed without moving and letting your blood move, you might as well bury yourself." Once we understand the patient's own unique theory concerning illness and ways of recovery, we can understand why it is so important for the patient to become active and why he may feel compelled to try to lift his bed.

His commitment to activity, however, is not necessarily unamenable to reason. He in fact believes that being too active and not "resting a

---

* Please reread Chapter 15.

minute" after sexual intercourse with his wife might have caused his disease ("I didn't give my heart a chance to rest").

The doctor, then, might consider attempting either to change the patient's model of illness to correspond with his own or to modify it just enough to allow optimal medical treatment to proceed. To make this decision, the physician has to assess why and how strongly the patient's model is held. In Tarzan's case, the importance of activity was emphasized over and over again, as though it were a prophylactic against death. It becomes clear during the interview that the patient had used activity as his most characteristic life-style and that he felt very proud of his strength and "bigness": "I've got a *big* chest so I can take in a lot of air"; "I don't drink small amounts of anything"; "In my youth, I was so strong that . . . I had to wrestle with four or five fellows at one time"; "I was what they called a natural strong man"; and so on. To a man with this kind of self-image, feeling weak and ill must be a terribly uncomfortable state. And this was exactly what he felt: "But today I feel sorta weak. . . . I think it's because they don't let me move my blood in the hospital." The patient's need for activity now is not simply a logical outcome of his general belief concerning health maintenance, but it also has a defensive quality—he needs to overcome his feeling of weakness from being inactive.

The physicians, with the help of the psychiatrist, decided to attempt to modify the patient's model rather than to change it completely. Strongly held beliefs that serve defensive functions are not easily given up, and the emergency nature of the medical condition militated against an attempt that might possibly have resulted in a rupture of the doctor–patient relationship. To attempt to change his activity orientation would have been to attempt to change his long-term (background-context) personality and coping style.

The patient's apparent denial of heart disease itself ("My doctor thinks I had a heart attack") was not complete: "I didn't give my heart a chance to rest." If he does not deny the presence of disease, he does deny the emotional upset that one might expect to be associated with it: "*Nothing bothers me. . . .* I don't let nothing bother me." Behind this denial, we get a glimpse of great suffering in the past—he apparently had thought that he had cancer in his 30s but was told that "it was all from nerves." Since then, he adopted an attitude of "nothing bothers me." Although this attitude may not be conducive to the patient's preparing for danger situations in a deliberate fashion, the ability to deny anxiety and fear may be helpful, if used in moderation, once he is on the coronary care unit (see Chapter 18). So long as the patient's behavior

is not maladaptive due to the denial, denial can be a protective mechanism. The question in this case might be how to change the behavior (e.g., lifting up the bed) without frightening the patient excessively, as would happen if one confronted him with the consequences of extending the heart lesion. If his denial were not functioning, we might again have a patient who is, as he described he had been when he thought he had cancer, worried, with pains in his head and diarrhea lasting for years.

The patient gives a good indication that an important person to recruit as a collaborator in the management may be his wife: "She wants me to stay home with her on Saturdays, I stay home with her." We notice that the patient is rather boastful, especially concerning his strength and, perhaps, masculine prowess. He did not respond well to nurses' reproaches concerning his activity. Perhaps an authoritarian approach threatens his sense of masculinity and need to be in control. His need for control, which goes along well with his activity orientation, is pretty clear throughout the interview itself, including, "And, someday, you fellas are going to come to the same conclusion—that moving the blood is the most important thing of all."

The patient came into conflict over authority with the nursing staff about who could give orders to whom. This resulted in his increased anxiety and the need for more activity to reassure himself that he did have control after all. In part, the authoritarian attitude of the nursing staff may be related to the rigidly authoritarian nursing hierarchy generally found in hospitals and the "double bind" in which nurses often find themselves (Chapter 18). To the nurses, it is unthinkable that a patient should wish to violate orders deliberately!

Actually, there were indications that the patient was having difficulties adjusting to the coronary care unit as early as the second day of admission. For example, he was found sitting up in a chair and was agitated at night and "arrogant." He asked for large doses of Valium. Because of a communication gap, however, the nurses' observations were not known to the doctors, who might have attempted to understand the patient's source of increasing anxiety.

To summarize, then, we have a patient who has an activity-oriented, controlling personality and who feels threatened about being immobile in bed in the coronary care unit. He has difficulties with nurses over authority but has a supportive wife, for whom he would do "anything." He has documented serious heart disease and is in pain and danger of his life. He has a strong belief that he has to "move his blood" to survive.

On the basis of the discussion above and the PEG of the "sick Tarzan" (Figure 34), the three-dimensional management plans outlined below were worked out.

Biological Dimension

1. Adequate pain relief with narcotic analgesics (meperidine, 50 mg intramuscularly every three hours): Pain relief is necessary for myocardial rest and gives the patient a sense of control.
2. Decrease diazepam, since large doses of tranquilizers may make the patient feel weak and drowsy.
3. Other necessary treatment of the MI and PVCs.

Personal Dimension

1. Treatment of pain with narcotic analgesics as above.
2. Nausea ceased to be problematic spontaneously.
3. Allow "moving blood" without compromising treatment: A schedule of token toe exercise was devised so that the patient could "move the blood" without having to strain the heart trying to lift up the bed. Taking into account the active, controlling, and taking-pride-in-being-a-strong-man personality, this plan was presented as follows:

> DOCTOR: I think we can suggest a good exercise for this purpose. However, I wonder if I can ask you to do something that's very difficult for a strong and big man like you—in fact, this may be the most difficult thing that anyone can ask of you—but, since you have a lot of strength, you might be able to do this.

> PATIENT: Well, maybe I can try. I can do anything, you know. What is it, Doc?

> DOCTOR: For a very strong and active man like you, staying in bed for a few days is one of the most difficult things for anyone to ask you to do. But your heart needs the rest so that it can heal. While you are in bed, you can still move your blood by doing an exercise—you can move your toes up and down, which will pump the blood all the way through the toes. But I am not sure that you have the patience and strength to do this difficult thing. . . .

> PATIENT: Well, Doc, I think I can try. I am an impatient man, but when I know that I am moving my blood, I can become the most patient man on earth.

59 yrs, ♂, married, president of a small company, 2 daughters, ages 26 and 24

DIMENSIONS	CONTEXTS		
	CURRENT (Current States)	RECENT (Recent Events and Changes)	BACKGROUND (Culture, Traits, Constitution)
BIOLOGICAL	Chest pain Nausea Relevant physical data Dx MI Diazepam, procainamide Meperidine	Borderline hypertension (7-8 years, no meds)	Hx of heart disease and cancer in family
PERSONAL	Nausea Pain Thinks pain due to indigestion Believes exercise essential for survival	Increased smoking (3-4 yr) Increased drinking (3-4 yr)	No previous hospitalization Has a "strong man" image Uses denial as a defense Hx of "nerves"—thought he had cancer, decided to deny— "nothing bothers me" Active, "take charge" type of personality boastful, dramatic Believes in exercise
ENVIRONMENTAL	ICU Bedrest Nurses Wife Very attached to wife; will "do anything for her" Being in hospital means business associates will be in charge	Father d. of MI 6 yr ago Mother d. of pulmonary edema (3 yr ago) 2 daughters married within last 4 yr, live in another state Sex immediately before MI hospitalization	Middle class Italian, Catholic backround High school education Many relatives died of cancer Father was very authoritarian executive of a small company for 25 yr

Figure 34. PEG of the "Sick Tarzan."

In the long run, the patient was able to give up smoking and reduce drinking when these were presented to him as challenges to his will-power and strength to promote his health.

Environmental–Interpersonal Dimension

Perhaps the most important intervention occurred in this dimension, because it involved changing the approach of the medical and nursing staff to the patient and his wife. The psychiatrist consultant coordinated this educational task.

1. The primary physician was instructed how to be able to present the approach he used concerning "moving blood" (see above). The patient was offered pain medication regularly, but he could decline to take it if he had no pain (reverse p.r.n. schedule).

2. The patient's wife was mobilized to be a collaborator in management. For this reason, she was invited to sit down with the doctor and nurses and jointly discuss the patient's progress—and compliance with the regimen. If the patient seemed to become too active, such as tending to sit up or get out of bed, the wife would go to him and say, "You know that it's important for me that you are strong and well. It worries me a lot when the doctors get worried about you and your getting out of bed; please stay in bed for a few more days—for me." She turned out to be a good collaborator, not only in ensuring compliance with the treatment, but also in reporting to the doctors and nurses about any change in the patient's state, level of anxiety, and sedation, so that medications could be adjusted on the basis of her observations.

3. Taking into account the talkative, boastful aspect of the patient, the members of the nursing staff were encouraged to listen to him and communicate that they were impressed by his strength. The nurses were able to understand that he needed to feel extra strong because otherwise he would feel so weak and afraid. As the nurses listened to him, they found him to be a rather charming "tall-story" teller, with whom it was rather enjoyable to converse. When the nurses had to be firm with him, they asked him to comply because otherwise they would "get into trouble." He usually heeded this appeal, because it gave him a sense of masculine chivalry—to do things so that a nurse would not get into trouble.

As his medical condition improved, he was given as much control as possible over his activity, including setting his own schedule for shaving and bathing. He was allowed out of bed as soon as possible and transferred out of the coronary care unit as soon as his physical condition

allowed. On transfer from the coronary care unit, the nurses on the medical ward were alerted to his unique needs, and he was prepared for the transfer carefully.

The concerted efforts by everyone concerned—doctors, nurses, and wife—were successful, and the remainder of his hospital stay was uneventful.

The Case of the Catatonic Patient with Enlarged Ventricles*

History and Course

This 27-year-old white married woman was admitted to the medical service for treatment of severe ulcerative colitis. She was a high-school graduate and the mother of two children. On arrival at the hospital, she was mute and immobile.

The family stated that the patient had complained of "racing thoughts" and a feeling that she was going "crazy" beginning several weeks prior to admission. In fact, the patient had stopped taking prednisone (a synthetic cortisol-like drug) that was given her for the colitis, because she felt it was making her "crazy." It was restarted, however, and she was receiving relatively high doses (60 mg/day) of prednisone for three weeks prior to hospitalization. She had no previous psychiatric history.

In the hospital, she was mute and unresponsive and showed waxy flexibility; that is, her limbs would remain indefinitely in any position in which they were put. Her eyes were open, and she appeared vigilant, occasionally grimacing at people in her room. The physical examination on admission revealed an emaciated and diaphoretic young woman with labile hypertension (170–130/120–90 mm Hg), tachycardia (90–125/min), and fever (rectal temperature, 100–101°F). Laboratory findings showed anemia (hematocrit, 29–27%), hypokalemia (2.2 mEq/liter), and hypomagnesemia (1l6 mEq/liter). All other values, including thyroid studies, were within normal limits.

On admission, she was placed on a regimen of hydrocortisone, 100 mg intravenously four times a day. Serum potassium and magnesium abnormalities were corrected by administration of potassium and mag-

---

nesium without visible improvement in the patient's mental status. An automatic computerized tomographic axial scan (ACTA), which visualizes the brain structures on X-ray, was performed on the second day of hospitalization and revealed "grossly enlarged lateral, third, and fourth ventricles with no evidence of cortical atrophy"; that is, a structural abnormality of the brain was found. A repeat ACTA scan three days later confirmed enlarged ventricles with no evidence of a tumor that might have caused this abnormality. An EEG revealed moderate, generalized, bilateral rhythmic slow-wave activity with no focal or epileptic features—an indication of generalized slowing of brain function. At this time, the most likely diagnosis was considered to be subacute sclerosing panencephalitis, a very grave condition for which there is no specific treatment.

She would occasionally manifest severe tremors of all her extremities that were sometimes mistaken for convulsions by the nursing staff. She was placed on small doses (2 mg) of perphenazine, an antipsychotic phenothiazine, three times a day, with some improvement. She would occasionally "wake up" from her stuporous, immobile state and converse with the staff, but would then lapse back into the catatonic state. During the lucid intervals, she was apparently responding to visual and, occasionally, auditory hallucinations.

Throughout her hospital course, she had stools positive for blood and developed episodes of frank gastrointestinal bleeding with marked evidence of toxicity. An emergency total colectomy was performed in the sixth week of hospitalization.

After the colectomy, all vital signs and laboratory findings returned to normal, and the hydrocortisone was gradually tapered off. Her mental status improved gradually such that five weeks after the colectomy, she was completely oriented and showed good recent and remote memory. She did serial 7s well, and her calculations and abstractions were within normal limits (see Chapter 12 for a discussion of the mental-status examination). Her judgment was good. Her affect was normal, and she related with the staff quite well. Tremors, waxy flexibility, and hallucinations were no longer present. She was eager to go home and return to her family and stated that she had no recollection of the events that had transpired during the period she was in the hospital until the time she recovered from the surgery. She did not remember any of the conversations she had with the physicians during her lucid periods, nor did she remember any of the hallucinations she had before surgery. A repeat ACTA scan performed at the time of discharge from the hospital, two and one half months after surgery, still showed enlarged ventricles of the same magnitude.

Comments

The PEG constructed shortly after admission is presented in Figure 35. The management plan for this patient involved discussing all aspects of the case with her husband, the important supportive person. This was especially important, because the patient, being mute and immobile, could not sign any forms for hospitalization or surgery and was not in any state to discuss management plans with the physicians. Treatment of the catatonic syndrome involves first an evaluation and treatment of the possible causes [see Table 6 (Chapter 7)]. Symptomatic treatment with antipsychotic medications (such as perphenazine) may be necessary. Possible causes of the catatonia included, in the biological dimension, hypokalemia, hypomagnesemia, steroids (hydrocortisone, prednisone), and the structural abnormality of the brain (enlarged ventricles on the ACTA scan). The toxicity from ulcerative colitis and fever may also contribute to this picture. The electrolytes (e.g., potassium, magnesium) were corrected, and the ulcerative colitis was initially treated with high doses of steroids without much change in the mental status. This left the possibility that (1) the steroids given to treat the colitis might be contributing to the mental state; (2) the structural abnormality of the brain on ACTA was indicative of a brain pathology that was causing the catatonic state, such as subacute sclerosing panencephalitis; or (3) the catatonic syndrome was primarily a psychiatric disorder, for example, schizophrenia, that might be only secondarily related to the biological abnormalities found.

In view of the negative family and past history of psychiatric disorder, and the outgoing, sociable personality of the patient, it seemed less likely that the patient had schizophrenia. Although the encephalitis was a possibility, no specific measures could be applied to treat it. In retrospect, and in view of the good outcome, this was probably not the cause of catatonia.

Following colectomy for the ulcerative colitis, the patient was withdrawn from the steroids. Simultaneously, her mental status improved. Thus, most likely, the steroid medications given to treat her medical disease contributed significantly to her abnormal mental status.

The patient's limited response to antipsychotic medication illustrates the nonspecific nature of antipsychotic treatment; that is, regardless of the etiology, psychosis can be symptomatically treated with these medications. In certain situations in which steroid medications are lifesaving, the psychiatric side effects may have to be treated with antipsychotic drugs while the steroid medications are maintained.

27 yrs, ♀, white, married, housewife, 2 children

CONTEXTS

DIMENSIONS	CURRENT (Current States)	RECENT (Recent Events and Changes)	BACKGROUND (Culture, Traits, Constitution)
BIOLOGICAL	Labile hypertension Tachycardia Fever Anemia Electrolyte imbalance Hypokalemia Hypomagnesemia Steroids ACTA – enlarged ventricles Ulcerative colitis	Ulcerative colitis for 3 yr Prednisone 60 mg (3 wk)	No family history of psychiatric illness
PERSONAL	Catatonic syndrome Mute	Racing thoughts (several weeks) Felt she was "going crazy" for a few weeks – stopped Prednisone Prednisone restarted (3 wk ago)	No past psychiatric history Pleasant, sociable person
ENVIRONMENTAL	Came in ambulance accompa- nied by husband	Married five years Two children Husband supportive	High school graduate Middle class Protestant Two siblings well Parents living and well No psychiatric history in family

Figure 35. PEG of the catatonic patient with enlarged ventricles.

Many physicians felt initially that this patient had an irreversible process in the brain. Such thinking led some clinicians to despair of effective therapy and even to suggest the futility of colectomy in "such an obviously brain-damaged patient." The subsequent course of this patient indicates, however, the value of correcting any treatable source of a multifactorial problem.

While the structural brain abnormality might have been associated with the brain's vulnerability to catatonia, it could not have been the major "cause" of the problem. This case draws our attention to the fact that every effort should be made to identify and treat *all* possible etiological and contributing factors in a seriously ill patient.

The Case of the Suicidal Terminal Cancer Patient*

History

This 35-year-old white woman, a physician's wife and the mother of three children, was admitted to the intensive care unit of the hospital. She was found comatose in her bed by her husband when he came home from work late at night. An empty bottle that contained secobarbital, a sleeping medication, was found next to her bed. Prior to the suicide attempt, the patient was being treated with radiation and chemotherapy for a cancer of the breast with widespread metastases.

Four years prior to the present admission, a lump in her left breast was detected on a routine physical examination and was subsequently diagnosed as malignant. Two years following the radical mastectomy, another cancer was detected in the right breast, necessitating another radical mastectomy. Approximately one year prior to the current admission, there was evidence of spreading of the cancer to the bone and behind the eyes. The spreading cancer was treated with chemotherapy, radiation therapy, and removal of both ovaries, since breast cancer tends to spread less when female hormones are absent. She also received steroids and pain medications for the severe pain she developed in her back and legs due to the spreading of cancer to the bones. The side effects of these treatments were grogginess, baldness, and facial puffiness, and there was growth of a moustache due to the absence of female hormones and the masculinizing effect of steroid hormones. The steroids also made her feel "high" and euphoric sometimes.

* Originally published in a somewhat different form in Leigh H.: Psychotherapy of a suicidal, terminal cancer patient. *Int J Psychiatry Med* 5:173–182, 1974. Copyright 1974 by Baywood Publishing Co. Portions reprinted with permission from the publisher.

She was a young woman who was intelligent and attractive (despite the bodily changes of late), but felt defeated and depressed. With the appearance of signs of spread of the cancer, she progressively withdrew from her social activities and her work as a social worker. She was feeling guilty about being a burden to her family and frightened about the possibility of being a "vegetable." For fear of burdening her family, she did not discuss her feelings of frustration and sadness. She wrote in her diary, which was found by her husband when she attempted suicide, "In order to maintain my equilibrium and not burden others, I'll try to be my own therapist and write down my feelings and thoughts about myself." In it, she expressed feelings of being "out of battle" and of not being involved. She had feelings of depression whenever she had pain, but occasional euphoria due to steroids and pain medications: "It is crazy to feel okay with what I have, but I am ready to take my exit pills tomorrow, if necessary."

Evaluation of the environmental–interpersonal dimension through interviews with her and her husband confirmed the initial impression that she had a supportive and concerned family. Her husband, however, had guilty and conflictual feelings because he could not spend as much time as he felt he needed to with his wife because of his busy schedule as a cardiologist. He was also uncertain about how to prepare his children for the eventual death of their mother. The suicide attempt was a great shock to him and increased his guilt feelings. The patient's mother lived in a city some distance away. The patient saw her occasionally, but had a very ambivalent relationship with her. Her father, described as an ineffectual person, died as the result of an accident when the patient was in her teens. She had a younger sister who was a housewife in another town.

Her oncologists were highly competent and empathic, but tended to expect their patients to deny the serious nature of the disease. They were very proud, for example, that all their patients with terminal cancer had a bright emotional outlook and "smiled at everybody." Of course, this patient's suicidal attempt belied this notion and came as a great shock to them.

The patient characterized herself as having been a fighter, for whom activity and mastery were very important. She had a full-time job and was a skillful tennis player. Pain and prolonged suffering, however, made her feel exhausted, weak, and defeated. She seemed to have used the defenses of activity and intellectualization successfully in the past, but of late, they seemed to be less effective in the face of pain and continuing progression of disease. She found that trying to smile and deny the presence of the serious illness, as her doctors seemed to want

her to do, was more depressing to her. She wished that she could talk about her pessimistic thoughts with her doctor, but felt that this would be a burden to the doctor.

Evaluation

On the basis of information available at the time of evaluation, a PEG was formulated (Figure 36). The immediate need of the patient was, of course, treatment of the overdose during the hospitalization, evaluation of the patient's suicide potential and prevention of suicide, and evaluation of the family situation and mobilization of support.

In terms of relatively long-term management, two problems had to be considered: the metastatic cancer and the depression and suffering of the patient and family. In the environmental dimension, the husband's guilt feelings about not spending enough time with his wife (recent context) had to be dealt with. In view of her ambivalent feelings about her mother and sister, these family members were not considered to be a great resource for interpersonal support. She was not a religious person and did not wish the involvement of clergy.

In the personal dimension, encouraging activity and intellectualization within the limits of physical capacity would be useful in view of her personality style, coping style, and defense mechanisms. A plan of collaborative treatment of her illness would be more likely to succeed than blanket reassurances with a "trust me" attitude. Her occupation as a social worker and her diary indicated that she would probably use and benefit from psychotherapy, although she had not yet sought it. In the recent context, the presence of pain was an important factor in making her feel out of control, defeated, and discouraged. A regimen of adequate pain medication was planned. If the disfiguring side effects of treatment were put in the context of tangible signs of a fight against cancer, they might be less depressing given this patient's personality, somewhat like the combat scars of old and proud veterans. Also, antidepressant medication should be considered for this patient.

In the biological dimension, treatment of the cancer should, of course, continue. As much information as possible about the expected effect of the treatments and the progress should be shared with the patient.

Course of Management

The patient was initially managed in the hospital until she recovered fully from the effects of the overdose. During this period, her suicide

35 yrs, ♀, married (to an M.D.), social worker, 3 children

DIMENSIONS	CONTEXTS		
	CURRENT (Current States)	RECENT (Recent Events and Changes)	BACKGROUND (Culture, Traits, Constitution)
BIOLOGICAL	Massive overdose of barbiturates; Terminal metastic breast cancer; Steroids; Pain medications	Lump in breast 4 yr ago – radical mastectomy; Recurrence in remaining breast – radiation, then mastectomy 2 yr ago; Metastases to orbit & bones (1 yr); Chemotherapy, radiation, steroids, oophorectomy – (within last yr)	Family history of cancer – aunt
PERSONAL	Suicidal attempt; Fear of immobilization	Pain ↑ 1 yr; Baldness, appearance change, jaundice (about 6 mo); Feeling defeated in recent mos; Futility over recurrent disease; Fear of burdening family and doctor – recent mos	Social worker; Attractive, intelligent; A fighter, activity & mastery important; Orderly, controlling type; Intellectualization; Counterphobic maneuvers
ENVIRONMENTAL	Husband & 3 children are supportive; No suicide note; Oncologists	Husband – busy physician feels guilty for not spending more time; Oncologists expect patient to be cheerful	Father died when patient in teens; Controlling and ambivalently experienced mother; Mother preferred sister (who lives in another town); Jewish, middle class

Figure 36. PEG of the suicidal terminal cancer patient.

potential and depression were evaluated by the psychiatrist in collaboration with the primary physician. It was decided that although the suicide attempt was serious, it was a reaction to the patient's feeling out of control and at an impasse with the oncologists about communicating her concerns. Thus, with improved communications and psychotherapy, she might cease to be suicidal. A considerable amount of depression was present, and amitriptyline therapy was instituted. Her family was evaluated by the psychiatrist, and it was decided that the psychiatrist would see the patient regularly—daily while in the hospital and once a week after discharge—and that her husband would also be seen by the psychiatrist on a biweekly basis to discuss his own feelings of guilt. This would also provide him with the opportunity to discuss various concerns of his including the preparation of their children for the eventual loss of their mother.

As these plans were set in motion, the patient was discharged from the hospital as the acute medical condition resolved. She kept her appointments with the psychiatrist diligently. The psychiatrist maintained close contact with the primary physician and the oncologist, discussed with the patient any new symptoms and plans of therapy, and encouraged her to communicate directly with the oncologists. She no longer felt that she had to always smile at them.

The psychiatrist, with the support of her primary physician and oncologist, encouraged her to return to work as much as she could tolerate (which was about half-time) and also to write down her experiences in fighting cancer. Writing this chronicle was a substitute activity for a more strenuous one, like playing tennis, and also served the function of being a record of her valiant fight against this serious disease.

She wanted to discontinue the amitriptyline soon after it was begun because of its side effects, especially sedation—she felt more out of control when she was groggy. It was discontinued, and the patient's depression lifted without medication as she gained a greater sense of control.

Psychotherapy was initially aimed at increasing her coping ability through discussing how she might cope with possible stressful events, such as increasing side effects from treatment (e.g., baldness). The idea that the side effects might be seen as something like battle scars was accepted by the patient with relief and determination. Soon, however, she wanted to explore her unconscious conflicts and meanings concerning her disease, cancer. She discussed in detail her ambivalent relationship with her mother, who had always been inconsistent and inconsiderate of her. She remembered experiences of being reprimanded by her mother for any independent activity and her mother's attempts to control her every activity, including what time in the morning she

could get up and what she should eat. She sought relief from this un-happy relationship by going to college away from home. Her father was ineffectual and did not interfere with her mother's controlling attitude.

In the course of psychotherapy, the meaning of the cancer became clear to her—it represented an alien, evil force that was attempting to control her life, change it, and, ultimately, extinguish it. In many ways, she saw her cancer as a symbol of the kind of overwhelming force that, like her mother, seemed to want to control her and subjugate her com-pletely. As she began to see cancer as an alien object, she was able to feel that she might be able to win over it, as she had been able to gain her independence from her mother. The process of gaining independ-ence from her mother was a protracted one in which, at times, she had to give in, but, at other times, she was able to achieve greater inde-pendence. She began to see her struggle with the cancer in a similar vein. She resolved not to feel completely defeated by one or two set-backs.

She decided to live and plan for relatively short and discrete pe-riods—a few months at a time. She felt a certain degree of mastery in *willing* herself to live for the discrete intervals.

During the seven-month period of outpatient psychotherapy, she seldom mentioned pain or physical discomfort and stayed away from prolonged discussions concerning her medical treatment. In fact, she seemed to want to believe that whatever physical symptoms she felt had a psychogenic origin and could be dealt with in psychotherapy. However, she recognized this as wishful thinking and did not neglect to take medications and treatments for the disease. The psychiatrist was in close contact with the oncologists throughout this period and made sure that the patient was complying with the medical regimen. Although she had free use of narcotic analgesics, she used them very sparingly, because she did not like their sedating side effects.

The disease progressed relentlessly despite the combined efforts of the primary physician, surgeon, oncologists, and psychiatrist. As spread of the cancer to the brain was discovered, she began to feel dizzy and had constant nausea. She still continued psychotherapy and was able to talk about her sadness over her disappearing youth and mourn for her unfinished plans, but then she hoped that her husband would re-marry soon after her death—almost as though to continue her happy marriage.

As she became too ill to come to the psychiatrist's office, her care was left primarily to her family. She chose not to be hospitalized during the final phase of the illness. The psychiatrist talked with her regularly on the telephone, and she would tell him about the amount of work

she was able to do at home despite the symptoms. During the final weeks of her life, her husband was almost constantly with her, having taken partial leave from his work. He continued to see the psychiatrist regularly and several times after her death, for support, discussion of plans, and sharing his thoughts and feelings.

Comments

This case illustrates how a terminal cancer patient found herself unable to communicate with her caretakers. As a result of complex interactions among her personality, her disease, its symbolic meaning, physical and emotional pain, and the effects and side effects of treatment modalities, she was driven to a suicide attempt.

Management plans based on a systematic evaluation of the patient were three-dimensional and effective. The patient's life was prolonged, and she died of her disease, not as a suicide—but above all, the eight and one half months of life she lived after the suicide attempt were *gratifying* to her. She died like the fighter that she had always been, and she died feeling that she had fought well to the very end, fighting as a team with her doctors and her family.

CHAPTER 22

Summary and Perspectives

Staying alive is something we do automatically. Health is usually taken for granted; ordinarily, we become aware of it only when something goes wrong, that is, when we experience signals of bodily malfunction, symptoms and signs of disease. While the nature of life itself remains a mystery, a great deal is known about the processes and mechanisms involved in living systems (Miller, 1978).

General systems theory proposes that the universe is composed of a hierarchy of concrete systems (Von Bertalanffy, 1968; Miller, 1978). These systems are accumulations of matter and energy organized into interacting, interrelated subsystems existing in a common space–time continuum. A patient is a *living system* composed, in turn, of biological subsystems. He is also part of a surrounding suprasystem (ecosystem). The living system is an *open system*; matter–energy and information pass back and forth and across the system. The general systems approach avoids the dichotomy between mind and matter. What have been called "mental" and psychological are conceptualized as manifestations of the information-processing subsystems, while what have been called "somatic" and physiological are conceptualized as manifestations of the matter–energy-processing subsystems of the living system. Of course, both systems are made of matter–energy. The health-care system is also an open system, into which patients enter, in which they are treated, and which they ultimately leave.

Living systems are amazingly complex. For example, an individual person in his life space can be thought of as a virtually infinite series of mutually interactive systems arranged in such a way that (1) one single

437

system may function individually as a *unit* or as a part (subsystem) of a larger system (suprasystem); (2) there are two-way interactions between and among adjacent and related unit systems and suprasystems and subsystems; in these transactions, both interactants are affected and changed; and (3) change in any unit or group of units eventually may affect functioning in all other parts of the aggregate. It's a good thing we do it automatically!

Each of us constitutes an individual or unit living system, existing in an environmental system. Both the individual person and the surrounding environment can be regarded as subsystems of a biopsychosocial suprasystem. Although energy and information can flow freely in both directions between the person and his environment, the person nonetheless maintains a separateness that is evidenced by the fact that the total energy level in the living person is higher than that of the surrounding environment (Schrodinger, 1962). This fact, in one sense, defines what it means biologically to be alive, that is, to maintain a dynamic equilibrium that sustains differential energy levels between the organism and the surrounding environment in an open energy system (negative entropy). When life ceases, the energy differential disappears.

Physical integrity of the living person is maintained by a series of interacting boundaries that begin at the interface between person and environment (e.g., skin, mucosal lining of body orifices, epithelial lining of lung, and sensory appartus of the nervous system) and extend progressively deeper and deeper within the individual's interior and specialized subsystem (see Figure 37). Energy transactions with the physical components of the environment consist of a chainlike series of physicochemical reactions starting at or relatively near the surface of the body and extending inward all the way to intracellular processes.

Psychosocial integrity of the person is maintained by a series of articulations with the interpersonal and social environment. In this sphere, transactional exchanges of information or meaning are mediated by symbols, including language. Patterns of sensory stimuli that are conducted into the individual via the peripheral nerves or special sensory organs are processed by the brain into *meanings* against the background matrix of memory and other cognitive processes. These as yet poorly understood brain processes and functions are grouped together as belonging with the realm of the "mind" (psychological processes).

Emotions participate in both of the foregoing series of articulations— the energy transactions ("physiological") and the meaning transactions ("mental"). Thus, emotions bridge the physical and symbolic aspects of environment with the physiological and mental aspects of the person.
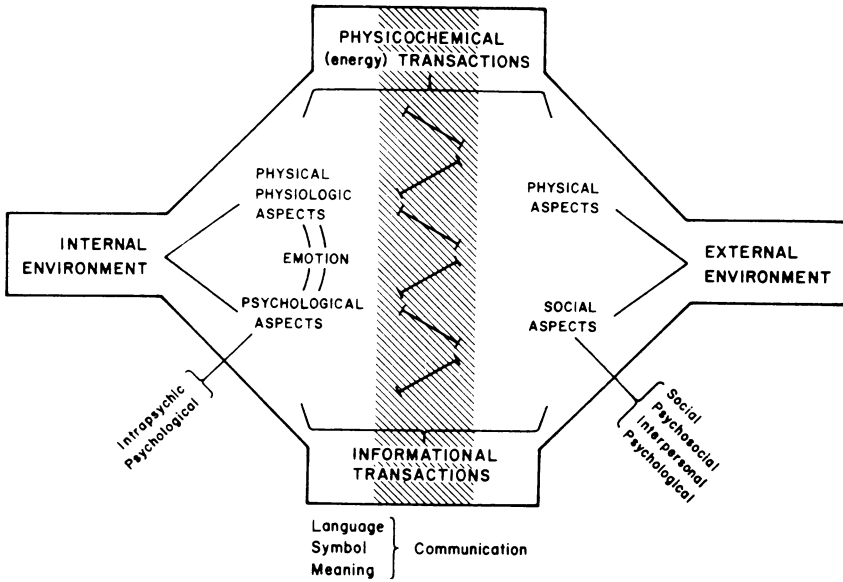
Figure 37. Interacting boundaries between the person and the environment.

The famous French physiologist Claude Bernard pointed out that the internal environment of the body must be maintained in a constant, steady state for optimal life processes to occur. The myriad elaborate and intricate chemical and physiological regulatory mechanisms that are involved in stabilizing the *milieu intérieur* are referred to collectively as *homeostasis,* a term coined by the American physiologist Walter Cannon (1932). The collective functioning of dynamic physiological processes that maintain the constant internal environment (which supports health and the accompanying sense of well-being that we take so much for granted) in the face of challenging inconstancy of the external environment is called *adaptation. Health* can be regarded as a state of *successful and effective adaptation* and *illness* as *maladaptation.* Adaptation to both physical and psychosocial aspects of the environment encompasses transactions between the person and the environment and among the components within the person. The latter may serve either or both of two purposes: (1) to maintain internal constancy despite environmental challenge; (2) to initiate and implement action that would change or accommodate the environment. All this is integrated, coordinated, and mediated by the nervous system and the neuroendocrine apparatus. Psychological and social challenges in the environment are perceived,

evaluated, and reacted to by the organism through the multiple operations of the brain, which we call by the collective term "mind."

So, with respect to both the physical and psychosocial dimensions of the environment, we can see that the brain serves as the control center for physiological and psychological mechanisms. The environment, the person as a unit, and the components of the person are, so to speak, connected by and in the brain, which serves to regulate the person's physiological and behavioral adaptation to environmental challenge.

Environmental challenges can be thought of as *stresses* that create a demand for adaptive work on the part of the organism; they may be physical or symbolic in nature. Their detection and processing as well as the ultimate responses to them in the form of internal readjustments or behavioral output into the environment are mediated by neural and endocrine structures. The extent and vigor of response reflect the *strain* that was created within the organism in reaction to stress. Some of these responses are primarily physical or physiological, for example, regulation of the pH of the blood; others are primarily psychological, for example, writing poetry. There is also a vast spectrum of life processes that express themselves (and are mediated) in both the physiological and the psychological spheres. The most prominent of these, of course, are the emotions.

The intriguing "intimate-but-separate" nature of the relationships between the inside and outside of living systems has stimulated interesting philosophical reflections about reciprocating influences of each on the other in evolution and development. In his essays, Lewis Thomas (1974) discusses in a highly imaginative and stimulating way some implications of resemblances between the cytochemistry of cells and the oceanic saline environment in which living matter had its origin. For example, he speculates that the mitochondria in our cells might be the descendants of some primitive bacteria that entered the primordial cells eons ago and stayed there in a symbiotic relationship with the cells. On the other hand, the advent of photosynthetic cells changed the course of evolution of the earth through the production of oxygen, which in turn determined the course of evolution of living systems on earth. He also finds striking similarities between the single cell and the earth as an entity.

Similar analogies can be and have been drawn between the nature of social structures and institutions on the one hand and some aspects of personality structure on the other, for example, those based on similarities between (external) moral and ethical standards of behavior as reflected in law and religion and internal psychological "structures" such as conscience (superego) and the emotion of guilt. It may not be

too farfetched to speculate that these internal and external analogues evolved from an interactive process, that is, psychological processes influencing and shaping developing social conventions and in turn being influenced and shaped by society. Some students of semiotics postulate similar reciprocal relationships between structural characteristics of the central nervous system and structures of the environment that determine patterns of sensory input. Noam Chomsky (1978), a linguist, postulates that meanings as expressed in language are dependent on functional substructures inherent in the organizational nature of the central nervous system. All these ideas do seem to converge in suggesting a mutually interactive developmental lineage between the inside and the outside of living organisms.

To summarize, a person is comprised of, and is a part of, a virtually infinite series of systems and subsystems. These systems and subsystems can be broadly grouped into two organizations—the person and the environment. The *physical environment* includes terrain, climate, fauna, flora, and environmental toxins. The *social environment* consists of significant individuals, families, small groups, communities, cities, states, and nations, all embedded in and influenced by a variety of cultures and subcultures—present and past.

The person can be viewed as subdivisible into organ systems, individual organs, cells, and intracellular subsystems. The *meaning system*, a function of the brain, defines the person's psychosocial integrity. Transactions or changes in any part of the personal system ultimately can be expected to affect all other parts of the system. For example, a change in the salt content of drinking water in the environment will ultimately affect hormones regulating electrolyte and fluid balance, function of cells in the kidney, function of the cardiovascular–renal system, and, finally, thirst and water-seeking behavior.

All the systems we have discussed exist in *time* as well as in space. The environment and the person have a past, present, and future. Progress through time is a continuing evolutionary process. At any given moment, the systems express what has gone before, and what transpires in the present will affect the state of the systems in the future. To understand the current state (including maladaptation) of any of these systems, knowledge of the past is helpful and often necessary. People undergo continuing development and maturation. The life cycle, the process of individuals' change over time, can be divided into phases, each of which has its unique adaptive tasks (Erikson, 1963; Levinson *et al.*, 1978). At any given time, the person exhibits remnants of past patterns of function as well as current patterns that have been built on the older ones that have been modified in the course of development—a

process called epigenetic development. To understand the environmental past, we turn to history, geology, archaeology, and cultural anthropology. To understand the person's past, we look to his or her genetic endowment and developmental life history, that is, a review of life experiences and their impact on developmental processes and maturation. This historical perspective has relevance in clinical diagnosis—the current or immediate state of balance between pathogenic vectors and the resistance of the individual to the pathogenic challenge will reflect and be influenced by what has transpired before in relevant subsystems. This is the reason that the medical history starts with the current chief complaint and proceeds to the history of the present illness (a chronicle of developing symptoms and signs) and, from there, to past medical history, review of systems, and family history. In this volume, we have expanded this theme to propose a clinical diagnostic method for tracing biological, personal, and environmental dimensions in terms of the present, the recent past, and the distant past (background).

States of adaptation or maladaptation (health or disease) have biological, psychological, and social aspects, and to be fully understood, they must be described with respect to all three systems: (1) the biological system, (2) the personal (psychological) system, and (3) the environmental system.

Keeping all this in mind, review what actually occurs in a routine medical encounter, for example, the first time a patient and physician meet in the consulting room. Each constitutes a biopsychosocial system. When the two come together, a therapeutic doctor–patient relationship is established, and around this dyadic relationship a whole series of subsystems forms and evolves in support of the care of the patient. This new complex system will include individuals and groups who are significant members of both the patient's and the physician's psychosocial spheres. This means family and friends of the patient and the myriad individuals and organizations drawn from the health-care system, including other physicians, nurses, social workers, and allied professionals (e.g., physical therapists, aides). The interactions will occur in clinics, offices, hospitals, wards, and special treatment units in the hospital and will require activities of administrative and financial organizations such as hospital administration, health plans, insurance, and government subsidiaries. Since transactions at all interfaces affect all parts of the system, the physician will have to deal with relevant biological, psychological, and social vectors as he carries out his job. Because of this, the behavioral sciences provide important and critical facts and concepts that are basic and necessary for satisfactory understanding and management of these complexities of medical practice. It has been our goal

in this volume to introduce and sensitize the readers to what we think may be the most immediately relevant information from the behavioral sciences and to show how it may be integrated with relevant biological and physiological principles in understanding health, disease, and clinical practice. Of course, such a goal cannot be fully achieved in an introductory textbook. We hope that we have stimulated the readers' thoughts, whetted their appetites, and started them on the road toward developing their own approaches.

References

Cannon WB: *The Wisdom of the Body.* New York, WW Norton & Co, 1932.

Chomsky N: Language and unconscious knowledge, in Smith JH (ed): *Psychoanalysis and Language.* New Haven, Yale Universities Press, 1978, pp 3–45.

Erikson EH: *Childhood and Society.* New York, WW Norton & Co, 1963.

Levinson DJ, Darrow CN, Klein CB, *et al.: Seasons of a Man's Life.* New York, Alfred A Knopf, 1978.

Miller JG: *The Living Systems.* New York, McGraw-Hill, 1978.

Schrodinger E: *What is Life? The Physical Aspect of the Living Cell.* Cambridge, Cambridge Universities Press, 1962.

Thomas L: *The Lives of a Cell: Notes of a Biology Watcher.* New York, The Viking Press, 1974.

Von Bertalanffy L: *General System Theory.* New York, George Braziller, 1968.

Index

445