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## **PREFACE**

The goal of this book is to help you, the reader, to obtain a high score on the Behavioral Science Section of Step 1 of the United States Medical Licensure Examination (U.S.M.L.E.). I believe that this book includes practically everything you need to know about behavioral science for the Step 1 examination. Much of the book's contents will also be useful in U.S.M.L.E. Step 2 and for your future practice of medicine, but these goals are secondary.

Michael Alan Taylor, M.D., co-authored two of the chapters with me. I thank Georgette Pfeiffer and Deborah Rubenstein for their help in preparing the manuscript, and Nutan Atre-Vaidya, M.D., and Stephen Goldberg, M.D., for their helpful suggestions about the book.

At many points in the book, to save space, I used the abbreviation MD instead of physician or doctor. I know that many readers have MBBS or DO degrees, which are functionally the same as MD in the U.S.

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# SECTION 1. ETHICAL, LEGAL AND SOCIOECONOMIC ASPECTS OF MEDICINE

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## CHAPTER 1. MEDICAL ETHICS

### UTILITARIANISM VS. DEONTOLOGY

Two fundamental topics in medical ethics are 1) *utilitarianism vs. deontology* and 2) *autonomy vs. beneficence*.

#### *Utilitarianism*

*Utilitarianism's* goal is the greatest good for the greatest number. An example is Medicaid in Oregon not paying for transplants because of the huge expense and, instead, allocating this money for prenatal and child care, which is more cost-effective. Several Oregonians died for want of transplants, but many moved to states which funded transplants.

#### *Deontology*

*Deontology* is decision-making based on principle. An example is the 1984 federal *Baby Doe Regulations*, which required aggressive life-sustaining care of newborns with severe birth defects, including infants with minimal chances of surviving. Utilitarian and deontologic positions sometimes synchronize and sometimes conflict.

### AUTONOMY VS. BENEFICENCE

#### *Autonomy*

*Autonomy* refers to a person being the final authority of what happens to him or her, including what medical care he or she receives. Examples include informed consent, right to refuse treatment, advance directives, and honoring religious beliefs.

#### **Informed Consent**

Consent for medical care should be *informed*. The MD should tell the patient about 1) the diagnosis and prognosis, 2) the rationale for a diagnostic procedure

or treatment, 3) the expected outcome of the care, 4) the precautions for and side effects of the intervention and 5) the alternatives. Standards for how well the patient must understand are not well defined, except that the patient should know what a reasonable person should know under the circumstances. A patient could ask to be spared the above details, but some information must be conveyed.

### **Right to Refuse Treatment**

Patients have a *right to refuse treatment*. You can offer a correct diagnosis and treatment for a serious, curable condition, and the patient can say no. Ascertain the reason for the refusal, decide if the decision is rational, and tell the patient that refusal carries serious risks. However, unless you could demonstrate in court that the patient cannot give an informed consent or refusal, or is suicidal or homicidal, you must “let the patient walk.” Ask the patient to sign a form refusing care *against medical advice* (AMA), but the patient can refuse to sign.

### **Advance Directives**

*Advance directives*, including the living will and durable power of attorney, assert the person’s current wishes about the medical treatment he or she wants or doesn’t want in the future if he or she cannot decide autonomously due to coma, delirium or other condition that affects decision-making. The *living will* is an easily-obtained, easy-to-understand, inexpensive document in which you write tersely what are your wishes about care if you cannot decide what you want.

In addition to or instead of a living will, you can assign *durable power of attorney*, designating someone who is familiar with your wishes to make decisions when you cannot do so, or if your living will is ambiguous. If you, the reader, have not made a living will and assigned durable power of attorney, do it.

### **Religious Beliefs**

Physicians must honor an adult patient’s *religious beliefs*. The classic example is a Jehovah’s Witness. Jehovah’s Witnesses are Christians who do not believe in the Trinity, revere religious symbols, or salute national flags. They will not accept transfusions of blood or blood products (exceptions: open-heart surgery or dialysis where blood is recirculated). They may ask that family members not receive transfusions either, even if the family member is not a Witness. They carry cards stating their beliefs. When they need surgery, they seek surgeons willing to operate by using blood substitutes like lactated Ringer’s. Some hypothetical cases:

- 1) An adult Jehovah’s Witness comes to your ER and needs a transfusion to save her life. You explain this, and she states “With all due respect, I’m a Jehovah’s Witness, and I cannot accept a transfusion.” She displays her ID card. You believe her. What should you do?
- 2) A Jehovah’s Witness brings her nine year old son to your ER. He needs a transfusion to save his life. You explain this, and she states “With all due respect, I’m a Jehovah’s Witness and so is my son, and he cannot accept a transfusion. What should you do?”

- 3) A comatose woman is accompanied to your ER by her husband. She needs a transfusion to save her life. You explain this, and he states “She’s a Jehovah’s Witness and cannot be transfused.” What to do?

My answers: Case 1: Unless you think a blood substitute can save her life, let her refuse and leave. Case 2: A parent cannot make a martyr of a child because of religious beliefs. Because this is life-threatening child neglect, you should hospitalize the child against the mother’s wishes, transfuse him, report the case to the department of child and family services, and inform your hospital’s administration about the situation. Case 3: Ask the husband to “prove” she is a Jehovah’s Witness, either by her card or a call to a relative or her minister. Once persuaded, unless you think a blood substitute can save her life, you must let the man refuse the treatment for his wife.

### ***Beneficence***

*Beneficence* is your obligation to help patients based on your medical knowledge. *Beneficence* and *autonomy* usually fit well, but sometimes they compete, as when a person requires but refuses a transfusion. In the U.S., when *autonomy* and *beneficence* conflict, *autonomy* usually wins. *Beneficence* guides decisions in some emergencies when we invoke 1) implied consent or 2) therapeutic privilege.

### **Implied Consent**

Faced with an unfamiliar comatose patient and no means of obtaining history or consent, you use the principle of *implied consent*: A reasonable person would want good, prompt medical care in this emergency—and you proceed to diagnose and treat.

### **Therapeutic Privilege**

Occasionally, such as when a combative patient threatens others’ safety, you treat without explaining the rationale in detail to the patient. This *therapeutic privilege* suggests that we forego detailed explanations when it is unsafe to take time to explain. Although using therapeutic privilege is not an iron-clad protection, in many circumstances it is the right thing to do.

### ***Nonmaleficence***

The converse of *beneficence* is *nonmaleficence*, summarized by the Latin phrase *Primum non nocere* (first, do no harm). MDs should not provide treatments that will hurt or be useless for the patient, even if the patient requests such a treatment.

### **Physician Assisted Suicide: Beneficence or Maleficence?**

Many persons assert that *physician-assisted suicide* is maleficent. Most US states have laws against it, and the other states have no laws about it. In June, 1997, the US Supreme Court confirmed the legality of state laws against MD-assisted

suicide. However, no US physician, including Kevorkian, was ever successfully prosecuted for homicide for assisting a suicide.

Most patients seeking MD-assisted suicide have depression responsive to treatment, after which they no longer want to commit suicide. But, some MDs think that, under certain circumstances, MD-assisted suicide is ethical and should be legal, as summarized in an Oregon legislative proposal: 1) The patient has less than six months to live, is likely to suffer intractably, and repeatedly requests assisted suicide. 2) The patient is not clinically depressed and has normal judgment, verified by independent MDs. 3) The MD must report assisting the suicide, cannot be prosecuted for assisting, and cannot be required to assist.

## **SUPREME COURT ABORTION DECISIONS**

In 1972, the US Supreme Court ruled in *Roe v. Wade* that therapeutic abortion was legal based upon a woman's *right to privacy*, a right implied but not stated in the Constitution. According to *Roe*, a woman may have an abortion on request during the first two trimesters and may have a third trimester abortion with a justification like severe risk to her health. In the subsequent *Webster v. Reproductive Health Services*, the Supreme Court upheld *Roe*, but permitted states to limit the conditions of abortion. For example, in most states, Medicaid will not pay for therapeutic abortion.

## **NONMEDICAL PRACTITIONERS**

In *Nally v. Grace Community Church*, the US Supreme Court ruled that if sued by a client, nonmedical practitioners (e.g., Christian Science practitioners, folk healers) are liable for the consequences of negligent care. For example, a Christian Scientist with melanoma seeks cure by prayer with a Christian Science practitioner. They pray, the melanoma metastasizes, the client's health deteriorates, and the client sues because the practitioner should have suggested medical care.

## **MANAGED CARE**

The *Wickline* case is a landmark decision about managed care. A California court held that an MD must provide equivalent care under managed care direction as would be given without managed care. Managed care is no excuse for negligent care. Mrs. Wickline had surgery for aortic occlusion at the iliac bifurcation (Leriche syndrome). The occlusion recurred, the surgeon and Mrs. Wickline agreed to re-operation, Medi-Cal (California Medicaid) refused to cover the surgery, re-operation did not occur, and Mrs. Wickline consequently needed leg amputations. She sued the surgeon and Medi-Cal, but only the surgeon was held accountable, because he should have either 1) insisted that Medi-Cal cover the procedure, 2) performed the operation at no charge to Mrs. Wickline or 3) transferred Mrs. Wickline to a medical center which would do the surgery.

## CHAPTER 2. FORENSIC MEDICINE

Good medicine and good law usually coincide. The best defense against a successful lawsuit is good medicine well documented, not excessive medicine excessively documented.

### CONFIDENTIALITY AND WHEN IT NEEDS TO BE BREACHED

The Hippocratic oath and medical tradition require that you honor your patients' confidences to maintain their trust. You are liable for the consequences of breaching *confidentiality* unjustifiably, as in the *Hopewell vs. Adebimpe* case (p. 114). However, there are circumstances in which you may need to breach confidentiality.

In some *life and death emergencies*, you may have to breach confidentiality. For example, if a suicidal patient elopes (escapes) from the hospital, you may call hospital security or the patient's family or the police to "intercept" the patient and accompany the patient to the hospital or another facility.

If you are *subpoenaed into court and ordered to testify* about a patient, and the testimony is relevant to the case, you must testify or be in contempt of court. If you believe the testimony is irrelevant to the case, respectfully ask the judge to discuss this with you in chambers.

To ensure reimbursement for a patient's care, *insurance companies* require the patient to give written permission for you to release information to the company to justify the care. Patients inevitably sign, and you release just enough information to ensure that neither the patient nor you is stuck with a huge unpaid bill. Insurance companies maintain extensive computer data bases about patients and doctors. Solutions to the problem of invasion of patient privacy include 1) publicly opposing the erosion of privacy (as I am doing here) and 2) keeping private notes (*work process notes*) for very sensitive but diagnostically irrelevant matters. Although MDs, unlike lawyers, cannot legally keep work process notes, I believe that discreetly keeping such notes—while disguising the patient's identity—is ethical.

Another exception is *peer review*. In *quality assurance* programs, medical professionals review charts maintained by other professionals, expanding the number of persons who know about the patient's life. If you are on a peer review team, don't review the charts of patients whom you know in non-medical circumstances (e.g., friends).

Doctors must report *certain communicable diseases* to the local public health department. Each state decides which diseases are reportable, how prompt the report should be, and what must be the reporting method (e.g., by phone). Not reporting these is a misdemeanor. Some states do not require reporting of sexually transmitted diseases, including HIV, unless the patient endangers others by unprotected sex or sharing needles. The reason is that because of the consequences



(e.g., loss of a job or a marriage) of others learning about an HIV infection, patients would not seek medical attention if they expected a breach of confidentiality.

Doctors are required to report *threats to the life of the President*, or visiting heads of state, to the US Secret Service.

## **LAWSUITS (TORTS)**

*Lawsuits* are justified on the grounds that 1) they are one of the few civilized ways that one can obtain restitution for damages due to negligent care and 2) they deter malpractice. But they are not a good deterrent because only 2% of cases of actual malpractice with damages lead to lawsuits (i.e., most of the time, doctors get away with malpractice), and in only 50% of lawsuits is there actual malpractice with damages. If you are sued and can prove you provided good care, ask your attorney and the insurance company to defend your case. Likely you will win the case. Most malpractice cases that go to court (i.e., are not settled out of court) are won by the MD. The conventional payment for the plaintiff's attorney is a *contingent fee*, usually one third of the payment for damages collected. If the plaintiff loses, expenses are minimal. This system has been criticized for causing lawyers to sue for huge sums, and justified because most plaintiffs could not afford to pay attorneys for dozens of hours spent on the case.

## **COMPETENCE AND CAPACITY**

### ***Competence***

*Competence* is a legal, not a medical, term. With few exceptions, competence can be declared only by a court. Different situations (e.g., competence to consent for surgery, or to stand trial) have different competency standards, listed in Table 1.

### **Types of Guardianship**

When a court declares a person incompetent to decide about a specific situation, it appoints a guardian to make decisions. Guardianship can be plenary or limited. A *plenary guardian* makes all major decisions for the incompetent person. A *limited guardian* decides only for the specific situation, such as to consent for surgery. Courts usually prefer limited guardianships.

## **CAPACITY**

While MDs cannot declare a person incompetent, they should routinely assess their patients' *capacities* (cognitive abilities) to make decisions. Knowing your patient's capacities facilitates care. If you think a patient lacks the capacity to make an important decision, that impairment is a legitimate concern. For example, if a depressed person hopeless about the future decides to give away his prop-

### **Table 1. Standards for Competence and Capacity**

- Competence for making decisions *about one's person and for entering into contracts*:
  - The person knows the nature and consequences of the agreement.
- To give informed consent*. Understands the indications, expected outcome, precautions, benefits, risks and alternatives.
- To manage funds*. Would not squander or hurtfully hoard money due to mental illness, such as exhausting or exceeding one's savings during a manic spending spree.
- To stand trial*. Able to understand the charges and to participate intelligently and cooperatively in the defense.
- To be executed*. Understands the nature of the crime and that the punishment is death.
- To make a will (testamentary capacity)*
  - Knows that one is making a will.
  - Knows what are one's possessions (*bounty*); for example, if one owns a house, one must know this.
  - Knows the persons to whom one logically might give one's bounty (i.e., the *natural objects of one's bounty*); for example, if one has siblings or children, one must know this.
  - Is not coerced (e.g., "Will your possessions to me or I'll leave you.") or tricked (e.g., "Don't will anything to your sister. She stole from you.") and is not acting upon a delusion ("My brother is poisoning me.")
  - (Note: three percent of wills are contested, and 15% of challenges succeed.)

erty, advise him to consult first with a family member or lawyer. The MD's standards for assessing capacity are essentially the same as the court's competency standards (Table 1).

### **RESPONDEAT SUPERIOR**

Medical students must be supervised by resident or attending MDs, residents by attendings, attendings by department heads, and so forth. According to the doctrine of *respondeat superior*, if negligent care is provided and there are damages, the whole chain of authority is responsible and can be sued, at and above the point where the error occurred. Plaintiffs tend to sue the whole chain, as the higher in the hierarchy, the more likely there will be insurance coverage (*deep pockets*) for damages.

### **RIGHTS AND OBLIGATIONS OF TRAINEES**

*Medical students and residents* have a right and obligation to learn by participating in well supervised patient care. The care must be as good as an experienced practitioner would provide. Students and residents must inform the patient about

their trainee status. Patients have an absolute right to refuse care by a student and to demand care by an MD. Patients can refuse to be treated by a resident, but if the resident is qualified to care for the patient and an attending is not available to see the patient, the patient may have to choose between no care and care by the resident under telephone supervision.

## **GOOD SAMARITAN LAWS**

All U.S. states have *Good Samaritan laws*, which limit the liability of (i.e., the court will look with favor upon) a professional helping in an emergency outside of his or her usual practice site. The court will not expect the same level of care that would be provided by a subspecialist in a medical center. However, you cannot grossly malpractice or abandon the patient. For example, if an accident victim is bleeding from a skin wound, and instead of tamponading the bleeding you pray for the patient and leave the scene, you could be sued successfully.

## **TESTIFYING IN COURT**

As an MD, you may need or want to *testify in court* or in a *deposition*, the latter being an extension of the court in which the opposing attorneys question witnesses, and a court stenographer transcribes the testimony during the pre-trial discovery period. Subsequently, depositions may be read verbatim in court. An MD may be asked to testify as a defendant, as a witness to an occurrence, or as an expert. An *expert witness*, by special knowledge and experience, may testify about opinions as well as observations. Ask colleagues to be available to cover your professional responsibilities for a few days before or after the day you are scheduled to testify. Court scheduling is unpredictable. Table 2 lists suggestions for court testimony by MDs.

**Table 2. Recommendations for Court Testimony by MDs**

**—Before testifying**

- Review the chart and other aspects of the case that you think important.
- Anticipate the questions you are likely to be asked, including the most sensitive and most essential to the case.
- Review pertinent literature.
- Ask the *retaining attorney* (the attorney who hired you) whatever questions you think important.

**—When testifying**

- Be sure you understand the question before you answer.
- Think for several seconds after each question is posed, instead of replying spontaneously.
- Answer only the question posed, and do so concisely.
- Do not agree with blanket statements unless they are 100% true.
- Explain the medical terms you use, but not patronizingly.
- Respond respectfully even if you feel threatened or challenged.
- Treat attorneys on both sides with equal courtesy. Friendly cooperation with one attorney and defensiveness with the opposing attorney lowers your credibility.
- Decisions to elaborate on answers or to act righteously angry should be weighed carefully, in advance if possible, and discussed with your side's attorneys in pre-trial discussions or recesses.

**—As an expert witness**

- Expect the opposition attorney to imply that you are a high-paid “hired gun” who favors only of one medical position (e.g., for a certain type of treatment), or that you conferred in advance with the attorney who retained you. Answer truthfully. Most jurors know that experts have pre-trial conferences and are paid to testify.
- Realize that lawyers, judges and juries are not automatically awed by your MD or your CV.
- Expect to be asked whether you hold your opinion *with a reasonable degree of medical certainty*. As standards for reasonable medical certainty vary by state, ask the retaining attorney for the state's definition.

## CHAPTER 3. MEDICAL SOCIOLOGY

*Medical sociology* studies how group membership affects the prevention, incidence, manifestations, treatment and prognosis of illness.

### SOCIAL CLASS AND SOCIOECONOMIC STATUS

A *social class* is a group with similar economic opportunities and prestige. *Socioeconomic status (SES)* is the prestige associated with social class. SES, often used synonymously with social class, is measured by a two factor (occupation and education) or three factor (occupation, education, residence) *index of social position*.

### CASTES

In a *caste system*, social classes are legislated. Until the 1940s, India had a caste system backed by law. India still has castes, but castes have no legal standing. In the US, black-white segregation was a caste system “remedied” by court decisions and legislation.

### SOCIAL MOBILITY

*Social mobility* is moving up or down in SES. *Upward mobility* is best achieved by obtaining education and deferring pleasure, such as by studying behavioral science when you could be swimming. *Downward mobility* is often due to chronic illness affecting behavior. For example, schizophrenics usually are of lower SES than their parents (*downward drift*).

### SOCIOECONOMIC STATUS AND HEALTH

Compared to persons of middle or upper SES, those of low SES are more apt to show generosity and gratitude, to report child abuse, to tolerate behavioral deviance in family members, and to face adversity with cleverness and dignity. Major mood disorders, obsessive compulsive disorder, anorexia nervosa and marijuana use are as common or more common among persons of middle or upper SES than among persons of low SES.

However, as a rule, low SES is hazardous to your health. Sophie Tucker said “I’ve been rich and I’ve been poor. Rich is better.” Table 1 lists health related correlates of low SES.

**Table 1. Health-Related Characteristics of Persons of Low Socioeconomic Status**

**Family and Social Factors**

More parental separation, divorce and death  
More high risk occupations and job related death  
More single parent families and mothers as household heads  
Less parental supervision outside the home  
More directing of children and less explaining and posing questions  
More marriage at earlier ages  
Greater sex role differentiation (i.e., who performs which tasks)  
Less joint participation of both parents in leisure activities  
Membership in fewer organizations  
Greater reliance on nonverbal cues (e.g., tone of voice, facial expressions) compared to the content of what is said

**Primary Prevention (prevention of occurrence) of Illness**

Less formal knowledge about physiology  
Less chance of eating a balanced diet  
Less chance of obtaining health checkups costing money, or of receiving free vaccine  
Fewer preventive health visits  
Less orthodontic care

**Incidence and Prevalence of Illness**

**More of the following:**

Infant mortality	Tuberculosis
Premature birth	Sexually transmitted disease
Serious and fatal accidents	Antisocial personality disorder
Mental retardation	Schizophrenia
Obesity	Somatization disorder
Chronic illness	Committing homicide and being murdered
Blindness	Committing rape and being raped
Speech problems	Committing robbery and being robbed
Lung disease	

**Access to Care**

Less access to good care  
More treatment in municipal general hospitals, which have higher fatality rates unless associated with a medical school  
More likely to be treated by a "committee" of housestaff and students compared to having a private MD

**Sick Role Behavior**

More responsiveness to current crises than future goals  
Less sticking to schedules  
More use of nonmedical personnel, like folk healers, and less self-referral to MDs when ill  
More self medication in contrast to prescribed medication  
Less trust of the health care system  
Sicker when admitted to the hospital  
More somatic (e.g., lightheadedness) compared to "psychologic" (e.g., "I'm afraid I'm going to die") presentation of mental illness  
Longer hospital stays

## **SOCIAL NETWORKS**

### ***Advantages of Social Networks***

*Social networks* are links within and between groups. Networks provide support when one is ill. The more social ties one has, the longer one's life expectancy. Longer life expectancy is even more closely tied with *perceiving* support than it is with *being* supported.

### ***Disadvantages of Social Networks***

But networks also carry obligations. For example, the night before your big test, your friend calls to say his car broke down and he wants your help. Tough situation! Close networks often convey medical misinformation. For example, you need salmetrol for asthma, but your family says "Don't take it. Uncle Harry took it, and look what happened to him."

### ***Comparison of Family and Friend Networks***

*Family networks* are close knit. Family members tend to know each other. Family networks are more likely than friend networks to convey misinformation. Friend networks are less closely knit; likely, some of your friends don't know each other. But friend networks are more apt to give good "second opinions" when you are ill.

## **ROLES**

A *role* is a situation that confers rights and obligations. Persons have many roles simultaneously. For example, I am my parents' child and my children's parent, a doctor and a patient. *Role conflict* occurs when the rights and obligations of one role oppose those of another role. When this occurs, you must choose between roles and state which role you are taking and why. For example, your mother asks for a prescription for an antibiotic. Best to tell her you can't treat her objectively, but you'll help her find a doctor who will do so.

### ***Sick Role***

The *sick role* is an ill person's rights and obligations. The rights are 1) to be excused from responsibilities which might exacerbate the illness and 2) not to be blamed for one's illness ("You have TB. You should have avoided people with coughs. You're a bum."). The obligations are to 1) be motivated to get well, 2) seek competent care, and 3) cooperate with that care. The sick role does not apply well to 1) stigmatized illnesses such as HIV infection or drug abuse, 2) mild illnesses like colds where many persons go to work despite being uncomfortable and

contagious and 3) chronic illnesses, where others have less energy to help as time progresses.

## **ETHNIC GROUP AND ILLNESS**

An *ethnic group* is persons with common national origin, religion, customs, language and physical characteristics. There are hundreds of ethnic groups worldwide, and we cannot learn medical correlates for all groups. One shortcut is to ask the patient what he or she thinks about your diagnosis and your proposed treatment. Another is to learn medical-ethnic correlates for groups you treat frequently.

Jewish and Italian-Americans tend to respond more dramatically to pain than do Irish-Americans, Mexican-Americans and white Anglo-Saxon Protestants. For the Jews, increased responsiveness is due to concern about prognostic implications of the pain; for the Italians, from not wanting to suffer.

Japanese persons in Japan have less coronary disease than do Japanese-Americans in Hawaii, who in turn have less than Japanese-Americans in California, likely because of different diets. Japanese in Japan have higher suicide rates than do Japanese-Americans, likely because suicide is more socially condoned in Japan. Mexican-Americans have higher suicide rates than Mexicans.

African-Americans have increased risk for hypertension, prostate cancer, sickle cell anemia, certain thalassemias, sarcoidosis and homicide victimization. Of all US ethnic groups, African-Americans have the lowest suicide rates.

## **OCCUPATION AND ILLNESS**

Common sense informs us about hazards for many jobs. For example, common sense informs us, and epidemiology confirms, that roofers, cooks and auto mechanics have a higher risk for burns. Also, nurses have a high risk for back injuries from lifting and turning patients, and boxers for head injury. The more fights a boxer has had, the more atrophy on his CT brain scan.

Of all occupational groups, miners are most likely to die in accidents, and workers in agriculture (timber cutters and loggers especially) second most likely. Writers have an increased risk for alcoholism and bipolar disorder, policemen (but not psychiatrists) for suicide, coal miners for anthracosis, sandblasters for silicosis, carpenters for nasopharyngeal cancer, workers exposed to asbestos for mesothelioma, and auto mechanics for lead poisoning.

### ***Physicians' Health***

MDs are less likely than their age- and gender-matched counterparts to obtain checkups or see an MD for a serious illness. MDs in non-direct-patient care specialties are more prone to behavioral problems than MDs who care directly for patients.



## ADHERENCE (COMPLIANCE)

Sociologic research (Table 2) also documents what common sense informs us about patient *adherence (cooperation, compliance)* with medical care: 1) The quality of the MD-patient relationship and the personalities of the MD and patient are more important than demographics like SES, ethnicity and age, and 2) when the MD is friendly, attentive, prompt, empathic and responsive, adherence is better.

## FAMILIES

The *family* is the basic unit of reproduction, support, nurturance, transmission of cultural norms and use of goods and services. It is the commonest source of referrals to MDs. A *nuclear family* is a mother and father (in a 2-parent household) and their children. An *extended family* is a three-generation family. People tend to marry persons of similar SES, religion, attitudes, and location of residence or workplace. However, there is no statistically significant association of personality traits among married couples. Many people marry persons with similar traits, and many marry spouses with different traits. Married persons have higher life expectancies than unmarried persons.

In the US, there is much marital instability, with *marriage rates* of 10/1000 persons/year and *divorce rates* of 4.8/1000/year. In part because of divorce, the average marriage lasts four years. Divorce is more common among persons who married in their teens, who *cohabitated* before marrying, whose parents divorced, and who are poor. Abuse is more common among couples who cohabit than among married couples. Persons whose parents divorced are more apt to cohabit premaritally than persons whose parents' stayed married.

## DEVIANCE AND CONFORMITY

*Deviant behavior* is not necessarily a sign of illness. For example, having a high IQ or being a star athlete is statistically deviant. Just as deviance has many causes, so too does *conformity*, which can result from cooperativeness (getting along with others, a valuable trait), passivity or lack of creativity.

## LIFE EVENTS, STRESS AND ILLNESS

There are small ( $r = \text{about } +0.2$ ) but statistically significant correlations between life events and developing general medical and psychiatric illnesses. Events (e.g., death of a spouse, a daughter leaving home) can be assigned points which can be added for prediction purposes. Not all stressors are sad or negative; for example, the birth of a child is a stressor. The stress-illness association sometimes confuses cause and effect. Being ill (e.g., having a severe mood disorder) can cause stressful events (e.g., marital discord). Proneness to stressful events is actually heritable. Prior illness is a better predictor of future illness than life changes.

**Table 2. Adherence (Compliance, Cooperation) with Medical Care**

**Adherence increases when the patient**

thinks the doctor is sensitive to his or her concerns  
feels the doctor is respectful  
is satisfied with the visit  
participates; the MD doesn't do all the talking  
receives a good explanation of the diagnosis and treatment  
believes the treatment is effective  
remembers what he or she is told (in some clinics, a nurse confirms that the patient knows the instructions)  
feels able to carry out the MD's recommendations

**Adherence increases when the doctor**

in summarizing for the patient, conveys the most important information early in the summary, when learning is greatest  
combines spoken with written instructions  
believes the treatment is effective  
tells the patient that treatment must continue for the correct duration, even after symptoms abate  
tells the patient which side effects may occur, and what to do if the side effect occurs  
treats side effects promptly and well  
tells the patient not to try discontinuing the medication to see what happens  
is friendly  
has a good rapport with the patient  
is familiar with the patient's history and sensitive to his or her background  
solicits the patient's view of the illness  
solicits the patient's feedback about problems with the treatment  
arrives on time  
follows up on missed appointments  
has successfully treated the patient previously  
involves family members in administering the treatment, if appropriate

**Adherence increases when**

there is continuity of care  
the patient has mild or moderate anxiety about the illness and its consequences; severe anxiety and no anxiety are associated with noncompliance; in general, people are better motivated when they are mildly anxious or concerned  
there is a short waiting time for the visit  
the referral process is clearly defined and explained  
the treatment has few side effects, especially behavioral side effects (e.g., drowsiness, amnesia, sexual dysfunction)  
the treatment alters the patient's lifestyle minimally  
the regimen is simple; for example, for drugs with long half-lives, like almost all psychotropic drugs, once or twice daily doses are simpler than three or four times daily doses  
the treatment course is brief  
the treatment is affordable

## **SELF-FULFILLING PROPHECIES**

*Self-fulfilling prophecies* occur when persons respond to others' expectations. If a teacher expects a student to perform badly (or well), the teacher will convey this to the student, who is then more apt to do badly (or well).

## **STANTON-SCHWARTZ PHENOMENON**

The *Stanton-Schwartz phenomenon* is that when staff disagree covertly about care of a patient, that patient is more likely to become anxious or excited.

## CHAPTER 4. HEALTH CARE DELIVERY

More than any other chapter in this book, this one reflects my biases. Having become a doctor believing that medicine was a learned profession (which it is) that controlled itself, I am troubled that business controls more and more of medicine and that, for economic reasons irrelevant to health, many sages assert that we have too many doctors. You, the reader, are more likely than I to accept the new system because you may not have experienced the “good old days.”

This chapter will present 1) the achievements and problems in the U.S. health care system, 2) health care as a business, 3) the health and health care of the poor and of underserved minorities, 4) the resources (professionals and institutions) in the system, and, albeit briefly, 5) the Canadian and British systems.

### ACHIEVEMENTS OF THE AMERICAN HEALTH CARE SYSTEM

Our population of 248,000,000 is among the world’s healthiest. American life expectancy at birth is 75 years, compared to 43 in 1900. The *crude death rate*, the annual number of deaths per unit population regardless of the population’s characteristics, is 8.7/1000 persons, compared to 9.5 in the 1960s. American medical technology is splendid. Sixty per cent of the world’s Nobel Prizes in medicine since World War II were won by Americans.

### PROBLEMS IN THE AMERICAN HEALTH CARE SYSTEM

But the system is riddled with problems: 1) Until managed care became powerful recently, annual inflation in health care costs was huge. Now it resembles inflation in other industries. 2) Despite our wealth, none of our health indices (e.g., maternal mortality) is the world’s best, and for many indices we aren’t even among the top 10 countries. 3) Indices of health and access to health care of poor Americans, and for several minority groups, are awful. There is unequal access, and *41 million Americans have no health insurance*—not governmental, not private—at all. 4) Recent solutions *reduce the autonomy* of MDs over their practices.

#### *Why Don’t We Get It Right?*

We don’t get it right because we are a *pluralistic society* responsive to many interest groups, and we have many justifiable goals and needs: Most of us would want 1) *universal access* to health care; we and South Africa are the only two nations without this; 2) all Americans with optimal health; 3) a continued free market; 4) MD autonomy; and 5) minimal bureaucracy in health care. But we can’t seem to meet these needs simultaneously. We disagree on solutions, and do what is most politically expedient (*disjointed incrementalism*) rather than what is best for society. Today’s goal is to balance the budget, reduce costs for government and

employers, and maximize health insurance company profits. For example, one day in June 1996, a health insurance corporation merger led to a \$900 million profit for one executive. We do not consider a single payer national health system, as most countries have. This would require more taxation; we have the industrial world's lowest taxation rate.

### ***Health Care Cost Inflation: A Serious Problem with Troubling Solutions***

The portion of the *gross domestic product* (the sum of goods and services produced in the US) devoted to health care is 14%, the highest of any country. This is much inflated since 1965, when health care took 6.5% of the gross domestic product. Table 1 presents causes of the inflation of the past three decades. Inflation hits home many ways. The more you spend on health care, the less you have for more health care and for other costs. The more the government pays for health care (e.g., Medicare), the less politicians support health care. Budget cuts reduce government support of medical research, 1) increasing medical school tuition, 2) making medical research harder to conduct and 3) making research careers less attractive.

Besides paying for 26% of health care costs out of pocket at the time of service, you and I also pay the other 74%: Government expenditures such as Medicare and Medicaid account for 41% of the system's costs, and this is paid by taxes. Private insurance, accounting for 31%, is paid by insured persons and their employers.

## **HEALTH CARE AS A BUSINESS**

### ***Payers, Providers and Patients***

Health care business terms are best remembered using the letter "P": payers, providers and patients in a poorly run system. A *third party payer* is an insurance company or government agency or employer that pays providers to care for patients. A *provider* is a professional (e.g., an MD, an RN) who diagnoses or treats, or an institution (e.g., clinic, hospital) where this occurs. Payers pay providers by 1) fees for service, 2) salaries without bonuses, 3) salaries with bonuses and 4) capitated care contracts. *Patients* are still called patients, except when they obtain health insurance, at which point they are *subscribers*, *enrollees*, or *covered lives*.

### ***Fee for Service Care***

In *fee for service* care, there is payment for each *episode of care*, once called a doctor visit. If the patient (subscriber, covered life) has fee for service insurance, the payer—if it agrees to pay—typically pays 80% of the fee most medical-surgical outpatient (*ambulatory*) care, and the patient pays 20% out of pocket (20% *co-insurance*). Sometimes the patient makes a small additional *co-payment*, such as \$2 for each prescription. Fees for service are paid in addition to monthly premiums paid by the subscriber, and usually the subscriber's employer. Unfairly, mental

**Table 1. Factors Contributing to Inflation in Health Care Costs**

<b>Factor</b>	<b>Comment</b>
—Only 26% of health costs are paid <i>out of pocket</i> at the <i>point of service</i> (at the time of the visit or soon after). 74% is paid by <i>third party payers</i> .	—The less paid at the point of service, the more willing are the MD and the patient to provide and seek care.
—High malpractice costs, which peaked in 1990 and have plateaued since. On average, of every \$100 of an MD's bill, \$15 pays the MD's malpractice premiums.	—Many MDs take needless time and expense in <i>defensive medicine</i> (excessive care excessively documented).
—Doctors advertise nowadays, and advertising and marketing are costly. When I was a young doctor, advertising was unthinkable.	—“Suffer from mental illness? Call Fast Freddie. Swift diagnosis, quick cure, low cost, slightly dishonest.”
—Overhead and bureaucratic costs. On average, each visit to an MD generates 10 pieces of paper to justify payment for the visit.	
—Excessive use of emergency rooms. Of 100 ER visits, only 5 are for genuine emergencies, and only 45 are for <i>urgent care</i> (i.e., requiring treatment within hours). Managed care is reducing excessive ER use.	—Where else but ERs can people be treated promptly for what ails them?
—The elderly population is increasing in numbers. The elderly are 12% of the population, but because the elderly are more apt to have multiple illnesses and to take multiple medications, they account for 30% of health care costs.	—This is a logical result of good health care and of preventive medicine. A cynical speculation: Does this have anything to do with the first movement towards MD-assisted suicide in US history?
—High medical school tuition. Half of graduates of private US medical schools have debts > \$75,000. Half of graduates of public US medical schools have debts > \$50,000.	—Many doctors pay back loans by moonlighting during residency, and some must charge higher fees when they enter practice.
—Uninsured (there are 41 million in the U.S.) and underinsured persons are less likely to use preventive services and more apt to delay care until they require intensive expensive care.	
—Preponderance of specialists instead of generalists.	—Specialists charge more than generalists.
—High-tech medicine is very expensive in terms of equipment and the need for highly educated, well-paid persons to conduct procedures and manage the equipment.	—High-tech medicine (e.g., magnetic resonance imaging, angioplasty, fiber optic endoscopy) used wisely saves lives and improves health.

health care is not covered as well as medical-surgical care. In fee for service mental health care, the payer typically pays 50% and the subscriber pays 50%. There is a strong movement, unsuccessful so far, for *parity in mental health care coverage*.

### ***Salaried Care***

Many providers are salaried and receive no fees for service. MDs in government-run medical centers like municipal general hospitals, VAs and state psychiatric hospitals, and MDs in medical schools, are salaried. In many health maintenance organizations (HMOs), particularly staff model HMOs (see below), many MDs are salaried.

### ***Salaries Plus Incentives***

In many HMOs, besides their salaries, MDs are rewarded for recommending fewer hospitalizations, lab studies, procedures or referrals. For example, in some staff model HMOs, a pool of money is set aside for each MD that the MD keeps if he or she keeps down costs. For each test ordered, or consultation requested, money is deducted from that MD's pool. This is a big *conflict of interest* in which the more care the patient receives in terms of lab tests or consultations, the lower the MD's income, pitting the patient's health against the MD's income. Economists justify this because it saves the patient money—HMOs cost less than fee for service—and motivates the MD to keep the patient healthy (*health maintenance*).

### ***Capitated Care***

Another payment mechanism is granting *multispecialty provider groups* (e.g., all the clinicians in a medical center or a group of affiliated medical centers) or specialist groups (e.g., a group of psychiatrists, and other mental health professionals) a fixed sum of money to care for a roster of patients known as *covered lives*. For example, a county grants a million dollar contract to a private group to provide all the medical care (e.g., examinations, followup care, preventive care, medications) for the county jail's 1,000 inmates. This *capitated* (capitated means money per head) arrangement makes a large profit if services are delivered efficiently, and takes a loss if services are inefficient or many inmates are seriously ill. To reduce the risk of the latter, some counties pay a prisoner's costs beyond a certain amount, say >\$5,000, if the inmate has a *catastrophic illness* like AIDS. The most profitable arrangement for provider groups is to have large contracts to care for well persons. This gives medicine the appearance of a business.

### ***How Payers Save Money***

Coinsurance, co-payments and bonus systems save money for payers. Other ways are deductibles, lifetime limits, prospective payment systems and—most rankling of all—managed care.

### **Deductibles**

Fee for service systems, be they Medicare or private insurance, save money through *deductible costs* whereby, at the start of each year, the subscriber pays a fixed amount (say, \$300) out of pocket before the insurance kicks in. A *subscriber* is the person who pays for the insurance policy. With *family coverage*, family members other than the subscriber are *insured persons*.

### **Lifetime Limits**

Most private companies set *lifetime limits* for each insured person. For example, if an insured person develops a *catastrophic illness* like AIDS and the company's payments exceed \$1 million, it stops the coverage. The person must then be treated in the public sector, as in a nursing home with Medicaid coverage, a VA hospital, a state psychiatric hospital, or a municipal general hospital.

### **Prospective Payment Systems and Diagnosis Related Groups**

*Prospective payment systems* were started by Medicare to pay hospitals for medical/surgical (but not psychiatric) inpatient care. Some private companies have similar systems. In a *prospective payment system*, a standard sum of money is assigned in advance for an episode of care of a patient in a category termed a *diagnosis related group (DRG)*. For example, Medicare might pay the medical center \$4000 for surgical care of an uncomplicated inguinal hernia, regardless of the patient's length of stay or the quality or quantity of the care.

### **Managed Care**

In *managed care*, payment for care and delivery of care are linked directly. In managed care, third party payers influence the provider's care, usually before the care is given (*prospective payment system*). Before managed care, MDs and patients decided what care would be given, then the care was provided, and afterwards the third party payer was expected to foot a large portion of the bill (*retrospective payment system*).

The most conspicuous examples of managed care—where external control is greatest—are preferred provider organizations and health maintenance organizations. The least management of care occurs with salaried care in the public sector (so far, at least). Virtually no management of care occurs when patients pay out of pocket for outpatient care.

### **Preferred Provider Organizations**

*Preferred provider organizations (PPOs)* began in the 1980s. A PPO is a payer that receives monthly premiums from subscribers, and usually their employers, and pays fees for service to a professional selected by the patient from a listed of providers approved (*preferred*) by the PPO. Approval for listing by a PPO requires that 1) the MD charges discounted fees, and 2) the MD lets the PPO manage the care. For example, to ensure coverage of a patient's hospitalization, the MD must persuade the PPO's representative about the need for the care. For example:



Dr: "Ms. Smith needs admission for appendectomy. She has fever, nausea, tenderness at McBurney's point, elevated WBC, and a normal urinalysis. I will operate at University Hospital. PPO rep (education: BA degree): "Seems reasonable. Proceed. Expect a five day hospital stay. Notify me if you change your plans."

### **Health Maintenance Organizations**

*Health maintenance organizations (HMOs)* began in the U.S. in 1910. The first were Kaiser-Permanente in California and Health Insurance Plan of New York. HMOs received a big boost from the *1973 federal HMO Act*, which gave grants for the development of HMOs and required all employers with 25 or more employees to offer an HMO option if one was available locally. Less costly than fee for service plans, HMOs have expanded greatly to 1996 enrollments of 50 million persons. Twice as many HMOs are for profit (e.g., dividends for shareholders) as not-for-profit. HMOs provide managed care by linking the financing and delivery of care, the latter by giving salaries to HMO employees or making capitated contracts with provider groups.

There are no fees for service in HMOs, with one exception: Some HMOs charge small co-payments like \$5 for MD visits. Patients are covered only for care by an HMO-approved doctor. Patients are assigned a *generalist MD* (family physician, general internist, general pediatrician) responsible for the patient's care and for referrals for lab testing or consultation or hospitalization. Being the patient's main doctor, knowing the patient well, and making referrals, are *primary care functions*. In most HMOs, these functions are termed *gatekeeper functions*. In most HMOs, patients cannot choose their MDs.

Besides financial disincentives for gatekeepers for referring and lab testing, HMOs use other *cost containment strategies*, like requiring the MD to prescribe only drugs in the HMO formulary, or letting the HMO pharmacist switch the drug.

There are different types of HMOs: In *staff model HMOs*, all MDs are full time salaried HMO employees. In a *group model HMO*, the HMO makes contracts with *independent practice associations (IPAs)*, groups to provide capitated care for the HMO's enrollees (*covered lives*). A *network model HMO* has both salaried HMO MDs and IPA contracts.

### **Backlash Against PPOs and HMOs**

These business strategies have generated a backlash from the public and from MDs. Some states passed *any willing provider laws*, in which a PPO must accept on its panel any qualified MD who agrees to the PPO's terms. In 1996, the Federal Government forbade HMOs to forbid their MDs to discuss conflicts of interest (*gag rules*), and required that uncomplicated deliveries be covered in the hospital for 48 hours, not just 24 hours that had been the limit set by most HMOs.

### **Employers as Payers**

By enrolling employees in group health programs and by paying portions of employees' health insurance premiums (and, in some instances, by being the in-

insurance company itself), employers are also payers. Today, 70% of Americans under 65 are insured through employer programs. Employers create a group whose members' premiums are lower than if the employee paid individually. Individual premiums are forbiddingly high. Large employers usually offer several plans, including HMO, PPO and high-premium fee for service coverage. The employee group practices *cost sharing*, in which premiums paid by healthy employees defray costs for employees requiring considerable care. Insurance companies raise group rates if too many of a company's employees require expensive care.

Some employers (or unions) create their own health insurance programs and receive premiums from their employees (or union members). These employer-run or union-run plans usually hire persons experienced in health care administration (*third party administrators*) for day-to-day management of the plan.

## **HEALTH STATUS AND HEALTH CARE OF THE POOR AND OF UNDERSERVED MINORITIES**

In recent years little public attention has been paid to the care and health indices of the poor and of underserved minorities. Access to care and health indices for these persons is awful. For example, life expectancy at birth for African-American males is 65 years, vs. 71 for Euro-American males. Infant mortality for black infants (18/1000 live births) is twice that of white infants (9/1000 live births). Survival past age 40 for men in Harlem, a primarily African-American community in New York, are less than survival past 40 for men in Bangladesh.

The poorer health and access to health care of the poor is the fault of society, not just our profession. Poor health in a community results in part from fewer and poorer local jobs and services like education and public safety, unsafe neighborhoods, and lower living standards. Medicaid, founded in 1965 as medical care for poor persons, pays poorly and pays late, discouraging doctors from caring for Medicaid recipients. Poor persons often receive their care in municipal general hospitals which—unless they are affiliated with medical schools—have higher mortality rates.

## **RESOURCES IN THE HEALTH CARE SYSTEM**

### ***Doctors***

There are about 645,000 US doctors, who consist of *allopathic physicians (MDs)* and *osteopathic physicians (DOs)*. There are relatively few DOs, who have essentially the same training, privileges and skills as MDs. To save space, I will refer to both MDs (and MBBS, the British equivalent of MD) and DOs as MDs. Most commentators perceive an MD *oversupply*. Few perceive an under-supply. Almost everyone perceives MD *maldistribution*, with too few MDs in rural areas, inner cities, state psychiatric hospitals and prisons. Unfortunately,

most commentators don't distinguish *economic demand (market forces)* from *epidemiologic need* (what's good for people's health). If economic demand is the standard, there is an oversupply.

### **Generalist Physicians**

Until the recent national generalist initiative, fueled in part by the need for generalists in HMOs, the vast majority of US MDs have been specialists. Between 1945–1963, the number of general practitioners (GPs) fell from 70% to 20%.

Generalist physicians serve *primary care functions*. Officially, the primary care specialties are family medicine, general internal medicine and general pediatrics. Given that most MDs are subspecialists, most subspecialists unofficially serve primary care functions. For example, a rheumatologist treating a person with arthritis is often the patient's main doctor, knows the patient well, and makes most referrals.

### **Hospitals**

In 1990, there were 6900 US hospitals. In the future, there will be fewer—one commentator predicted that only 4000 would remain by the year 2010—because of reduced payer reimbursement for inpatient care and more support for ambulatory care. Many hospitals have low occupancy rates, making them vulnerable to closing if they don't use unoccupied space cleverly such as by creating step-down or intermediate care units.

Of the 6900 hospitals in the US in 1990, 6700 were community hospitals, and 200 were specialty hospitals like children's and mental hospitals. A *community hospital* is a short-term general hospital, of which there are three types: 1) *proprietary (for-profit, investor-owned) hospitals*, one of whose goals is shareholder income; 2) *not-for-profit hospitals* like those run by religious orders or medical schools which invest their income to operate the facility, and 3) *government hospitals* like municipal general hospitals. Whatever of the type of hospital, the largest item in its budget is personnel costs. When hospitals merge or lose money, layoffs are management's typical first move. Table 2 lists types of community hospitals. Most hospitals now label themselves as medical centers to convey a breadth of services.

### **Long Term Care**

*Long term care* is services for persons unable to care for themselves. It includes nursing homes, adult day centers, respite care and hospice care.

### **Nursing Homes**

In 1985, 1,300,000 Americans lived in nursing homes (NHs). Five percent of elderly Americans live in NHs. The corollary is that 95% live in the community. Most NHs are proprietary, and 41% are owned by NH chains; NHs are profitable.

## **Table 2. Hospitals**

### **Veterans Affairs (VA) Medical Centers**

- Of the 30 million US veterans, 15% use the VA system. Reasons:
  - Appreciation of good care they've received.
  - Poverty. The modal user of VA services has a low income, and Medicare has deductibles and coinsurance. The VA only has a \$2 co-payment for each prescription.
- The VA primarily serves veterans with disabilities acquired on active military duty (*service connected disabilities*), but now seeks to care for non-service connected veterans.

### **Teaching Hospitals**

- Teaching hospital = a hospital affiliated with a medical school.
- Of all hospital types, teaching hospitals provide more care for the poor and more free/charity care.
- When a municipal general hospital is affiliated with a medical school, health outcomes are as good as those of the average community hospital.
- Medicare provides higher reimbursement to teaching hospitals than to non-teaching hospitals, by paying *direct* (salaries) and *indirect* (time and resources for teaching) costs of educating residents.

### **Municipal General Hospitals**

- Examples: Cook County (Chicago), Charity (New Orleans), Metropolitan (New York), Boston City, Los Angeles County.
- Have provided care, saved lives and delivered babies for tens of millions of poor persons.
- Understaffing is common, amenities (e.g., carpeted floors) few, and many jobs provided by political patronage.

### **Rural Hospitals**

- Almost half of US hospitals are rural.
- Average US rural hospital occupancy rate is 55%, a low rate; more prone to closing than any other hospital type.

Medicare covers NH care only for brief post hospital discharge stays. In contrast, 48% of NH care is paid by Medicaid. Medicaid eligibility for NH care begins promptly for poor persons. It does not begin for other persons until they exhaust their savings and sell their homes to cover their NH care, leaving them *medically indigent*.

### **Adult Day Centers and Respite Care**

*Adult day centers* provide daytime supervision and a modicum of medical care to disabled persons whose families work by day. *Respite care* is round the clock supervision for several weeks to permit family members a vacation (respite). This permits free time and relaxation to encourage their caring for their relative the rest of the year.

## **Hospices**

A *hospice program* is a team approach for terminally ill persons for whom the goal is comfort, not cure. Often, patients may medicate themselves, including with narcotics for pain. A hospice may be a team that consults for patients at all locations, or may be a hospital unit, a building or an office suite. The hospice movement does not endorse physician assisted suicide.

## ***The Federal Government***

The federal government's huge role in health care is summarized in Table 3. The Health Care Financing Administration (HCFA) administers Medicare and, along with the states, Medicaid. Table 4 summarizes Medicare and Medicaid.

## ***Other Systems***

We and South Africa are the only nations without universal coverage. Two other systems that receive much attention are the Canadian and British systems.

### ***Canadian System***

The *Canadian system* consists of a main system that covers all Canadians, and a private system to cover *rationed procedures*. In the main system, all Canadians are covered, and there are no copayments or deductibles. Canadians can choose any MD. Unlike our system with its myriad payers, the main Canadian system has *one payer (single payer system)*, the government, financed by taxes. Fees are negotiated by local provinces with local medical societies. There is rationing by all provinces for some procedures. For example, Quebec will not pay for annual physical examinations, and no province covers in vitro fertilization.

The Canadian system, once cited as a model system, has had big troubles recently. The inflation rate is second only to that of the US. There is a perceived oversupply of MDs with a reduced number of residency positions.

### ***British System***

The *British system* is two coexisting systems, a *National Health Service (NHS)* that covers everyone, and a private system for those who can afford it. All British are covered by the NHS, in which most care is provided by GPs, who may refer patients to specialists with no penalties. MDs work for a salary determined by the number of patients under the MD's care. MDs have specific hours each week to work in the private system.

**Table 3. Federal Government's Role in Health Care**

<b>Agency</b>	<b>Area of Responsibility</b>	<b>Comment</b>
<i>Department of Health and Human Services</i>		
—Health Care Finance Administration (HCFA)	—Medicare and Medicaid	—See Table 4
—United States Public Health Service (USPHS)	—National Institutes of Health (NIH)	—Main source of federal funding for medical research
	—Centers for Disease Control (CDC)	—Monitoring public health and controlling epidemics
	—Food and Drug Administration (FDA)	—Deciding whether a food or drug can be marketed
	—Substance Abuse and Mental Health Services Administration (SAMHSA)	—targeted mental health areas such as HIV and child mental health
— <i>Department of Labor</i>	—Workplace safety	
— <i>Department of the Interior</i>	—Mine worker health	
— <i>Department of Agriculture</i>	—Farm worker health	
	—Control of animal diseases	
— <i>Department of Defense (DOD)</i>	—Health of active duty military and their families	
— <i>Department of Justice</i>	—Federal prison health	
— <i>Department of the Treasury</i>	—Narcotic related health issues	

**Table 4. Medicare and Medicaid**

	<b>Medicare</b>	<b>Medicaid</b>
—History	—Proposed in Kennedy Administration and passed into law in Johnson Administration in 1965 as part of Bennett Amendment to Social Security Act	—same as for Medicare (1965, Bennett Amendment)
—Original purpose	—Medical care for the aged —Preamble: Goal was to finance health care for the aged, not control it. In reality, Medicare often pioneers influential new standards for care and its reimbursement. Threatened penalties of withdrawal of Medicare coverage to medical institutions are used as leverage to ensure that Federal policies are followed.	—Medical care for the poor
—Administrative oversight	—100% federal.	—Federal government and states.
—Who is covered?	—All American citizens and permanent US residents age 65 and over, regardless of means (no means testing) —All chronic dialysis and renal transplantation patients (nobody could afford these otherwise) —All persons on Social Security disability (unable to work due to due to prolonged severe medical illness) —Railroad retirement pensioners. When AMTRAK became bankrupt, the US government took over AMTRAK and its retirees —Federal government determines eligibility	—Recipients of state cash assistance (green card) —Poor children under age 5 —Poor pregnant women who will receive state cash assistance after delivery —Pregnant women in two parent families in which main wage earner becomes unemployed —Medically indigent persons needing nursing home care

**Table 4 (cont.)**

	<b>Medicare</b>	<b>Medicaid</b>
	—35,000,000 Americans covered	—States decide who is eligible. State by state variations leave millions of persons uncovered. —About 25,000,000 Americans covered. —Payments are low and slow
Deductibles	—None for Part A —\$100 for Part B	—None
Co-payments	—20% for Part B	—None
Categories	—Part A —Inpatient hospital care —Brief nursing home care after hospital stay —Hospice care —Chronic dialysis and kidney transplantation —Home health care —Part B (optional; has monthly premium) —Physician care —Medications —Medical equipment —Ambulance service	
—Medigap	—Some persons purchase private medigap insurance to cover copayments and deductibles.	—Recipients cannot afford medigap.



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## SECTION 2. THEORIES OF BEHAVIOR

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### CHAPTER 5. PSYCHOANALYTIC THEORY

*Sigmund Freud* graduated from medical school in Vienna and became a basic neuroscientist. Unable to support his family this way, he began private practice in psychiatry. To improve his skills, he studied under Charcot, who hypnotized patients with conversion disorders. When they reported childhood memories of sexual fantasies or abuse, their symptoms disappeared. This impressed Freud, who hypnotized patients to make unconscious memories conscious. However, his hypnotic cures were transitory, and he founded *psychoanalysis* as a means of making unconscious memories conscious.

#### CORE CONCEPTS OF PSYCHOANALYSIS: DYNAMIC UNCONSCIOUS AND PSYCHIC DETERMINISM

The core concepts of psychoanalysis are the dynamic unconscious and psychic determinism. The *dynamic unconscious* posits that our behaviors are largely influenced by memories, fantasies and conflicts outside conscious awareness. *Psychic determinism* posits that our most influential unconscious memories relate to relationships with our parents and siblings.

#### FREUD'S TOPOGRAPHIC MODEL

We noted the value of making unconscious thoughts conscious. The topographic model refers to levels of awareness: the conscious, preconscious and unconscious.

- 1) The **conscious** is like a complex sense organ noticing what we're aware of at one point in time. Right now, in your conscious mind you're thinking about the topographic model or, better yet, you're having a sexual fantasy.
- 2) The **preconscious** is what we call to mind by a simple act of will, such as our address, or what we will say next.
- 3) Existence of our **unconscious** can only be inferred. It is our memory stores that are not conscious or preconscious.

#### FREUD'S STRUCTURAL MODEL

Freud's topographic model didn't explain all he observed, so he developed a *structural model*. The mind's structures are defined by their functions, not by anatomic structures. The mind's structures are the id, the superego and the ego.

- 1) The **id**, entirely unconscious, consists of our primitive sexual and aggressive drives and urges. These urges are more primitive than wanting to make love or assert ourselves. An id impulse might be to have sex with everyone at your medical school. The id operates on the *pleasure principle* of *instant gratification*: “I want what I want when I want it, which is now.”
- 2) The **superego** has unconscious, preconscious and conscious components and two subdivisions, the conscience and ego ideal.
  - A. The **conscience** is our internalized sense of right and wrong. Not meeting its standards causes *guilt*.
  - B. The **ego ideal** is our internalized sense of whom we want to be like. I’d like to be like Michael Jordan and Mother Theresa, but I settle for me. Not living up to the ego ideal’s standards causes *shame*.
- 3) The **ego** is defined by its functions:
  - A. sensation and perception
  - B. movement
  - C. cognitive and intellectual functions
  - D. getting along with others (*object relations*)
  - E. self control (*impulse control*)
  - F. moods (*affects* in psychoanalytic theory)
  - G. executive functions: mediating between i) the id and the superego, and ii) the id and the outside world
  - H. ego defense mechanisms

If you have trouble with these concepts, you’re not alone. They’re inaccessible to scientific scrutiny.

### ***Why Do We Have Ego Defense Mechanisms?***

If we acted on id impulses (e.g., we tried to have sex with everybody), the police would immediately jail us. We don’t want this. Consequently when an id impulse threatens to erupt, the ego becomes anxious. The ego’s anxiety about an id impulse is termed *signal anxiety*. Ego defense mechanisms alleviate signal anxiety.

### ***Specific Ego Defense Mechanisms***

Vaillant divided the defense mechanisms into mature and not mature. He found that Harvard students who used larger numbers of *mature mechanisms* were more apt, years later, to be “successful” (e.g., listed in *Who’s Who in America*) than those using fewer mature mechanisms.

### ***Mature Ego Defense Mechanisms***

- 1) **Altruism**. Acting selflessly. Example: An MD treats patients who cannot afford to pay her.
- 2) **Humor**. Being funny to reduce stress. Example: Dr. S made the class laugh during a high-stress lecture.

- 3) *Suppression*. Willfully postponing expression of a mood until a more opportune time. Example: Angry at Tuesday's poor class attendance, Dr. A didn't express anger at those present. Instead, he thought of ways to improve his presentations to increase attendance.
- 4) *Sublimation*. Channeling an unacceptable impulse into an acceptable behavior. Example: As a teenager, Ron LeFlore stole. As a young adult, he became a professional baseball player, and led the American League in stolen bases.

### ***“Not Mature” Ego Defense Mechanisms***

- 1) *Repression*. Unconscious relegation of unacceptable information into the unconscious. Repression is very common, underlies forgetting, and is often paired with other defense mechanisms. Example: Ms. L was angry at her boss. At a holiday party that night, while introducing her boss to a friend, she momentarily forgets her boss' name.
- 2) *Displacement*. Expressing towards one person the feelings that one held originally towards another. Common in phobias. Example: Angry at poor class attendance, Professor B returned home and insulted his daughter.
- 3) *Rationalization*. Explaining something truthfully but incompletely, leaving out what is unacceptable. Example: Not offered the promotion he expected, Mr. L says “I didn't really need the promotion.”
- 4) *Reaction formation*. Adopting an attitude opposite to an unacceptable feeling. Example: Dr. R, who unconsciously hates disabled persons, becomes an advocate for the disabled. Can I prove this scientifically? No.  
How does reaction formation differ from sublimation? In reaction formation, the unconscious wish becomes its opposite. In sublimation, the wish is channeled acceptably.
- 5) *Doing and Undoing*. Symbolically reenacting and counteracting an unacceptable wish. Underlies obsessions and compulsions. Example: Mr. D unconsciously wants to kill his family by burning down his home with his family in it. To counteract this wish, he compulsively checks his stove by a) turning it on (symbolically burning down the house) and b) clicking it off to reassure himself that his house won't burn.
- 6) *Projection*. Attributing one's unacceptable impulses to others. Examples: 1) A competitive medical student dislikes his classmates because “they're backbiting competitive gunners.” 2) An employee falls in love with a co-worker. These loving feelings are unacceptable to him, so he develops the delusion that the co-worker loves him (*erotomanic delusion*). Projection underlies many delusions.
- 7) *Passive aggressivity*. Showing anger indirectly by lateness, slowness, forgetting, sloppy work. Example: Angry at her boss, Ms. L completed tasks slowly.
- 8) *Somatization*. Expressing feelings through general medical symptoms. Example: Anxious about a forthcoming USMLE, Dr. Y had diarrhea.

- 9) *Denial*. Nonrecognition of obvious information. Transient denial is normal upon learning of personal tragedy. Example: "Tell me it isn't true." Denial is pathologic when it impedes a person's acting in his or her best interest (e.g., refusing a life-saving treatment). Severe denial occurs in psychotic disorders and nondominant parietal lobe disease. Example: A woman with left hemiparesis due to right parietal stroke keeps trying to walk despite falling and injuring herself each time.
- 10) *Regression*. Returning to an earlier stage of functioning; losing developmental milestones. Example: Jim, previously toilet trained, wet his bed for several weeks after his brother was born.
- 11) *Splitting*. Perceiving others as all good or all bad; inability to see a person's strengths and weaknesses simultaneously. Common in borderline personality disorder. Example: During one visit, Mr. Brown tells his doctor "You're the best doctor I've ever had." Next visit, he tells the doctor "You don't know what you're doing."

Note: With the exception of suppression, all defense mechanisms are unconsciously driven, not willful.

### ***Complex Defense Mechanisms Associated with the Doctor-Patient Relationship***

- 1) *Transference*. Experiencing towards one's doctor (or nurse, or social worker) the feelings that one had originally for important persons in one's childhood. Example: Mr. C's father was often sarcastic. Mr. C feels his surgeon is sarcastic, although the surgeon isn't sarcastic.
- 2) *Countertransference*. When the doctor experiences towards a patient the same feelings that the doctor had originally for important persons in his or her own childhood. Example: Dr. Z's father was often disrespectful to Dr. Z. Dr. Z feels that his patient, Mr. A, is disrespectful, although Mr. A is respectful.

## CHAPTER 6. LEARNING AND BEHAVIOR MODIFICATION

*Learning* is an observable change in behavior based on experience. *Behavior modification* is using learning theory to treat patients. There are two types of learning, associative and nonassociative.

### ASSOCIATIVE LEARNING

*Associative learning* results from *pairing* of stimuli, as in this example: 1) A dog naturally salivates when it smells meat powder; 2) A bell rings every time the meat powder is presented, and the dog salivates when the meat powder and the bell are presented together; 3) The dog eventually salivates when the bell rings and no powder is presented. The dog has *paired* the meat powder with the bell.

### NONASSOCIATIVE LEARNING

*Nonassociative learning* requires no pairing of stimuli. There are three types of associative learning: habituation, sensitization and observational learning (modeling).

#### *Habituation*

In *habituation*, repeated presentations of the same stimulus reduce responsiveness. For example, the first time a medical student attends anatomy lab, he feels queasy. By the 15th time, he is comfortable enough to eat a snack there. Habituation is neither categorically good nor categorically bad. If the stimulus initially causes an unwanted response (e.g., queasiness in the anatomy lab) to a valuable stimulus (e.g., being in the anatomy lab), habituation to the stimulus is good. Habituation underlies *in vivo* exposure, a treatment for phobias, obsessive compulsive disorder (OCD) and several other conditions.

In *in vivo exposure*, patients are exposed over several months to a hierarchy of anxiety-provoking events (least anxiety-provoking first, most anxiety-provoking last) until they are comfortable during the event. For example, Ms. A has social phobia. He lists a hierarchy of anxiety-generating social events (least anxiety-provoking: conversing with a neighbor in the hall; most anxiety provoking: addressing a large audience), and one by one, week by week, he faces each situation until he is comfortable with it.

Or, Ms. B, who has OCD, is obsessed about becoming dirty and contaminated. As her treatment begins, her therapist accompanies her to a washroom and the pa-

tient practices touching the floor and then rubbing her hand on her sleeve. Late in the treatment, Ms. B can comfortably clean a toilet.

### ***Sensitization***

In *sensitization*, repeated presentation of a stimulus increases responsiveness to it. For example, on the first day of a new course with a new lecturer, a student is mildly uncomfortable with the lecturer and the course, thinking, “I don’t like the lecturer or the topic but I’ll get used to it.” During the second lecture, she becomes progressively more annoyed. By the fourth lecture, she is so angry that she stops attending class.

Like habituation, sensitization is neither categorically good nor categorically bad. If the stimulus is undesirable (e.g., a bad lecturer), changing the stimulus (e.g., changing the lecturer’s style or replacing the lecturer) is best. If the undesirable stimulus cannot be changed, some of the audience would benefit from habituation (e.g., learning to appreciate the lecturer), and others in the audience would benefit by sensitization (leading to cutting the lecture and learning from another source).

If the stimulus is desirable (e.g., a beloved sex partner), pleasurable sensitization to the stimulus (e.g., increased sexual responsiveness) is good. Sensitization underlies sensate focus exercises in sex therapy.

In many sexual dysfunctions (e.g., male erectile disorder, female arousal disorder, premature ejaculation), patients view intercourse as a skill, talent, or ability—a performance—rather than a source of pleasure. It is natural to be anxious before “performances” (*performance anxiety*), but this reduces the pleasure of sex and impedes the autonomic physiology of intercourse. In *sensate focus exercises*, a couple is told to avoid intercourse until later in the treatment, and taught exercises focusing on pleasure—not performance—like kissing, nongenital stroking, and genital stroking, each without intercourse. Intercourse is the last step.

Why one person experiences habituation to a stimulus and another person experiences sensitization to that stimulus depends on the person’s personality traits, beliefs, and past experience.

### ***Observational Learning (Modeling)***

Much learning occurs by watching someone else. For example, we observe an interview strategy we admire, and use that strategy in subsequent interviews. In medical care, modeling is used in various ways, such as the following:

#### **“Ethan Has an Operation”**

“Ethan Has an Operation” is a film in which a child actor narrates how he successfully handled the pre- and post-operative phases of his herniorrhaphy. When this film was shown to children awaiting herniorrhaphy, there were fewer pre- and post-op complications (Ethan was a *role model*) than for children not shown the film.

## **Group Therapy for Phobias**

In group therapy for phobias, new members of the group learn ways to overcome their phobias by observing how more experienced group members overcame their phobias.

## **CLASSICAL AND OPERANT CONDITIONING: TWO TYPES OF ASSOCIATIVE LEARNING**

There are two types of associative learning, classical and operant conditioning.

### ***Classical Conditioning***

In *classical conditioning*, a *reflex* response (e.g., a dog's salivation) is *elicited* in response to a stimulus (e.g., meat powder) that precedes it. The meat powder is the *unconditioned stimulus*, and the salivation response to the meat powder is the *unconditioned response*. Then, a bell (a *conditioned stimulus*) is presented along with the meat powder; eventually, the dog salivates in response (a *conditioned response*) to the bell alone. This and similar experiments earned *Ivan Pavlov* the Nobel Prize.

### **Examples in Medicine of Classical Conditioning**

Examples of medically related classical conditioning include the effects of white lab coats and of antineoplastic agents. In past years, most physicians who treated children wore white lab coats. Often, children paired the lab coat with the painful or frightening aspects of the medical visit; merely seeing a white lab coat evoked anxiety. Consequently, many clinicians who treat children don't wear white coats. This doesn't eliminate anxiety from medical visits, only the anxiety generated by the lab coat.

Many patients taking IV antineoplastic agents have nausea or vomiting. Some of them pair aspects of coming to the hospital (e.g., the hospital parking lot) with their original nausea, and become nauseated while approaching the hospital. This can usually be deconditioned.

### **Behavioral Treatment of Idiopathic Enuresis: An Example of Classical Conditioning**

Treatment of idiopathic enuresis uses classical conditioning. For bedwetters, urination (an unconditioned response) during sleep occurs when the bladder is full (unconditioned stimulus). The child is told to drink less fluid before bedtime, and then one of two strategies is used to awaken the child to urinate when the bladder is full. (1) With a *moisture-sensitive blanket*, when the child begins to urinate in bed, a buzzer rings, awakening the child, who finishes urinating in the toilet. (2) Alternatively, a parent awakens the child at intervals to urinate. Eventually, the child automatically awakens (conditioned stimulus) to urinate (conditioned response) when the bladder is full, and the bedwetting ceases. The child has paired being awake with a full bladder and urinating.

## ***Operant Conditioning***

In *operant conditioning*, a behavior is *emitted* in anticipation of an event termed a *reinforcer* or *reinforcing stimulus*. Rephrased, we act in anticipation of consequences. The three types of operant conditioning are positive reinforcement, negative reinforcement and punishment (aversive conditioning).

### **Positive Reinforcement**

In *positive reinforcement*, something valued is presented after a desired or desirable behavior is emitted. There are countless positive reinforcers, like praise, gratitude, food, sex, money, intellectual stimulation, laughter, and attention, which drive our lives. It helps to praise patients for their efforts to improve their health, and to treat patients in a pleasing, friendly setting.

### **Negative Reinforcement**

In *negative reinforcement*, something unpleasant (e.g., hearing loss and discomfort from impacted cerumen) is removed following a desired behavior (e.g., visiting an ENT doctor). Because most visits to MDs are to treat symptoms, most medical care works through negative reinforcement. Don't confuse negative reinforcement (taking away something unpleasant) with punishment.

### **Aversive Conditioning (Punishment)**

In *aversive conditioning (punishment)*, we try to decrease undesirable behaviors by presenting an unpleasant (aversive) stimulus or removing a pleasant one. The number of aversive (punishing) events is almost infinite, including avoidance and inattention, rudeness, tactless criticism, disrespect, low grades, spanking, dull activities, fines, imprisonment, and withdrawal of privileges. Of the three types of operant conditioning, punishment is least effective in improving behavior, because punishment angers people and does not teach them what they should do. To teach people what they should do, punishment must be paired with positive or negative reinforcement. Aversive conditioning is sometimes valuable, however, such as in smoking cessation or alcoholism programs.

*Rapid smoking* is used in smoking cessation programs. The smoker is asked to take deep rapid puffs on a cigarette, which is very uncomfortable. The goal is to link smoking with an aversive consequence.

*Butt jars* are also used in smoking cessation programs. The smoker is told to 1) obtain a large glass jar, 2) place the jar in the living room, and 3) to empty ashtrays into the jar. The goal is to link smoking with a disgusting conspicuous "butt jar."

*Covert sensitization* scripts are used in alcoholism programs. The patient reads a script which depicts drinking as disgusting, such as "You are at the bar and you order a drink. You begin drinking, you feel like vomiting, and you do vomit, all over the bar and your clothes and the person next to you. Then, . . ." (Dr. Sierles, stop!)



## Reinforcement Schedules

In operant conditioning, the reinforcer (positive, negative or aversive) may be presented on a schedule.

In a *fixed ratio schedule*, the reinforcer is presented when we emit a behavior a certain number of times, like every second or fourth time. In *1:1 fixed ratio reinforcement (continuous reinforcement)*, the behavior is reinforced whenever it is emitted. For example, every time a doctor sees a patient, she receives a fee.

In a *variable ratio schedule*, the reinforcer is presented unpredictably, as if random. A person cannot tell how many times he or she must emit the behavior before it is reinforced. This very potent schedule is hard to extinguish; witness the success of slot machines.

In a *fixed interval schedule*, as with paychecks, elections and tests, the reinforcer is presented after a specific amount of time. This schedule increases behaviors just before the reinforcing event, and reduces behaviors right afterwards (a *scalloped pattern*). For example, politicians campaign hardest just before elections, and students study hardest just before tests.

In *variable interval schedule*, unpredictable periods of time elapse before reinforcement occurs, as for an MD trying to reach a hard-to-find relative of a patient by phone (or vice versa!).

The distinction between ratio-based and interval-based schedules is sometimes blurry. For example, the MD trying to reach the relative will also make an unpredictable number of calls before reaching the relative.

*Extinction* occurs when a behavior's frequency diminishes as rewards for it cease. For example, many children's temper tantrums can be extinguished if parents pay no attention (attention is a positive reinforcer) to the child during the tantrum.

## Stimulus Control: Using Discriminative Stimuli

*Discriminative stimuli* are *environmental cues* that a behavior is likely to be reinforced a certain way. For example, stop signs are cues that if you stop, you won't be broadsided. An MD's office is a cue that it's OK to talk about symptoms.

The concept of discriminative stimuli is used in *stimulus control* of insomnia or of smoking. In treating most types of insomnia, tell the patient to use his or her bedroom for three things only: 1) dressing and undressing; 2) sex; and 3) going to sleep when sleepy. If the patient feels sleepy, tries to sleep, and doesn't fall asleep after 5–10 minutes, he or she should leave the bedroom, go to another room to do something else, and return to the bedroom only when he or she feels sleepy.

In smoking cessation programs, the patient is instructed to avoid bars (where smoking is endemic) and to sit in the nonsmoking section of restaurants.

## Cognitive Psychotherapy

Moods affect attitudes. For example, if you're sad, you're less apt to engage in and enjoy activities. *Cognitive psychotherapy* is based on converse: *attitudes affect moods*. For example, socially phobic persons mistakenly believe that others will notice that they're anxious and ridicule them for it. Discussions in therapy,

and homework assignments, persuade patients that these perceptions are inaccurate. They are told that other people expect only that we be considerate and competent, and if we are, people don't care whether we're nervous.

### **Assertiveness Training and Behavioral Rehearsal**

Some persons have trouble standing up for their rights, or assert themselves too aggressively. They may benefit from *assertiveness training*, in which they are coached in, and practice in a group, asserting themselves effectively. This practicing is *behavioral rehearsal*.

### **Biofeedback**

Ordinarily, we can't willfully control our autonomic functions because we cannot monitor these functions accurately. In *biofeedback*, functions like sweating or blood pressure or muscle tension are monitored with devices like a galvanic skin resistance sensor (to measure sweating), a sphygmomanometer, or electromyogram (EMG). If we learn to relax by slow, deep abdominal breathing, and then monitor a physiologic function, we can affect that function. Biofeedback is effective for tension headache and for postural hypotension due to spinal cord transection. It can lower blood pressure slightly, but not enough to replace low salt diets and antihypertensive drugs.

### **Reciprocal Inhibition and Systematic Desensitization**

*Reciprocal inhibition* is the principle that you can't be anxious if you're relaxed. In *systematic desensitization* of phobias, the patient 1) develops a hierarchy of anxiety-generating situations, 2) learns to relax by abdominal breathing or *progressive muscle relaxation* (relax forehead muscles first, then facial muscles, and so forth), and 3) experiences situations along the hierarchy while relaxed.

## CHAPTER 7. OBESITY: APPLICATION OF LEARNING THEORY

Much of the treatment of obesity relies on learning and behavior modification principles.

### FACTS ABOUT OBESITY

- 1) *Heritability*: Family, twin and adoption studies suggest a genetic contribution.
- 2) *Socioeconomic factors*: In industrialized countries, obesity is more prevalent in persons of *low socioeconomic status* (SES) than among persons of middle or upper SES. In some third world countries, the reverse is true and many poor persons starve.
- 3) *Medical consequences*: Consequences of obesity include hypertension, diabetes mellitus, cardiovascular disease, gallstones and orthopedic problems. Cardiovascular complications of obesity are greater for *male pattern* (abdominal) than *female pattern* (hips and thighs) obesity.
- 4) *Childhood- and adult-onset obesity*:
  - a. In *childhood-onset obesity*, there are more fat cells—fat cell *hyperplasia*.
  - b. In *adult onset obesity*, existing fat cells are filled with fat—fat cell *hypertrophy*.
  - c. Some persons have both types of obesity.
- 5) *Physiology*:
  - a. The *hypothalamus* has feeding (perifornical region) and satiety (paraventricular nucleus of the medial hypothalamus) centers influenced by many neurotransmitters including cholecystokinin, norepinephrine, dopamine and serotonin.
  - b. *Set point theory*: Our bodies limit how fast we gain or lose weight. When we gain, metabolism increases. When we lose, metabolism decreases.
  - c. Compared to thin persons, obese persons *respond less to internal cues* of satiety and fullness, and *more to external cues* like seeing food.
- 6) *Psychology*: Compared to thin persons, obese persons do not work harder to obtain food; for example, they don't go to the grocery store more often. However, what obese persons buy at the grocery store differs.

### WEIGHT REDUCTION

Weight reduction strategies include the following:

- 1) A low calorie diet with foods that the patient likes, so that when the patient loses weight, he or she can maintain the diet.
- 2) A medically coordinated *exercise program*

- 3) *Self reward*: In addition to the positive reinforcement of appearing thinner and the negative reinforcement of declining obesity, patients may be instructed to reward themselves for certain amounts of weight lost. For example, "When you lose the first five pounds, buy yourself that silk blouse you listed."
- 4) *Stimulus control*: The goal is to reduce the number of cues for eating:
  - a. Eat only in one room.
  - b. Eat only at certain times.
  - c. Don't eat during pleasurable activities like watching TV.
  - d. Don't buy fattening foods.
  - e. Don't shop for food while you're hungry. If you do, you'll buy more food and more fattening food.
  - f. Don't buy junk foods.
  - g. Don't use snack trays. The surest way to "overdose" on M&Ms is to display them in a tray.
  - h. Remove food from the table after each course, or at least after the meal. Obese persons continue to eat what remains on the table.

### ***Outcome of Weight Reduction Programs***

For most patients, weight loss programs are initially successful, but relapse is common. But persons who have lost weight in the past are able to lose weight again.

## CHAPTER 8. GENETICS OF BEHAVIOR

Genes determine our species, sex, physical characteristics and the structure and functions of our organs, including our brains, and contribute to our personality traits, general intelligence, specific cognitive abilities, and creativity.

### MONOMORPHIC PROPERTIES OF THE GENOME

Most of our genome is *monomorphic*; that is, *homozygous* (AA or aa) and hard-wired to ensure that we share qualities with other animals, mammals, social mammals, primates and other people. Well over 95 percent of our genotype is the same as that of other mammals.

### WHAT DISTINGUISHES US HUMANS FROM EACH OTHER?

Three factors distinguish us humans from each other: 1) our unique experiences; 2) our social and physical environments; and 3) *polymorphic* (heterozygous, like Aa) *properties of our genome*.

Studies of tens of thousands of persons in over 40 countries suggest that the genetic contribution to the uniqueness of each of our personality traits is 35–50%, and the contribution of the environment is 50–65%. Among family members, the *nonshared environment* (events unique to each person, such as one sibling attending one college and another sibling attending another college) influences each family member's personality more than does the *shared environment* (events shared by family members, such as sharing meals at dinner and attending the same church).

### GENE NETWORKS

Personality traits, general intelligence and psychiatric disorders are not determined by single *major genes* (*sledgehammer genes*, *big genes*). Instead, they are influenced by *networks of genes*, each gene separately influenced by the environment. Characteristics of these networks include polygenic, pleiotrophic, and oligogenetic influences.

*Polygenic Influence:* Multiple genes affect one phenotype.

*Pleiotrophic Influence:* One gene simultaneously affects multiple phenotypes.

*Oligogenetic Influence:* Genes affect each other.

# STRATEGIES FOR DETERMINING THE GENETIC CONTRIBUTION TO THE VARIANCE IN THE PHENOTYPE OF A DISORDER OR PERSONALITY TRAIT

Determining the genetic contribution to the variance in the phenotype of a disorder or personality trait begins with family studies and proceeds to twin, adoption, pedigree/linkage, and restriction fragment length polymorphism (RFLP) studies.

## *Family Studies*

Family studies come first because of one fact: genetic conditions are *familial* (they run in families). But not everything familial is genetic; for example, speaking French or being Catholic are both familial, but neither could be genetic. Table 1 defines terms used in genetically related studies.

In a family study, the research group creates two teams. Team 1 identifies a group of probands and a group of controls. Team 2 evaluates first degree relatives of probands and first degree relatives of controls, while blinded to whether they are evaluating first degree relatives of probands or first degree relatives of controls. If first degree relatives of probands are significantly more likely to have the condition than are the first degree relatives of controls, the condition is familial.

## **Relative Risk**

The *relative risk* is the probability of first degree relatives of probands having the condition compared to the probability of first degree relatives of controls

### **Table 1. Common Terms in Behavioral Genetic Research**

*Proband*: the first ill family member identified by the research team

*First degree relatives*: persons who share 50% (parents, siblings, children) to 100% (monozygotic twins) of their genotype

*Second degree relatives*: persons who share 25% (grandparents, grandchildren, nieces, nephews, aunts, uncles) to 50% (if the second degree relative is a monozygotic twin of a first degree relative) of their genotype.

*Monozygotic (identical) twins*: persons who share 100% of their genotype

*Dizygotic (fraternal) twins*: twins who share 50% of their genotype; during pregnancy, each has a separate placenta

*Heritability*: the extent to which genotype affects phenotype

*Penetrance*: the proportion of circumstances in which the genotype produces its phenotype; with complete penetrance, as in Huntington's disease, everyone with the genotype (who lives long enough) develops the disease.

*Expressivity*: the extent (slight or extensive) to which the genotype is manifested in the phenotype; whether the condition or trait presents clinically as mild (e.g., cyclothymia) or severe (e.g., bipolar 1 disorder)

having the condition, or compared to the general population. If first degree relatives of probands are twice as likely to have the condition as first degree relatives of controls, or twice as likely as the general population, relative risk is 2.

### **Morbid Risk**

*Morbid risk* is the probability that a relative of an ill person has or will develop that illness. Siblings of a schizophrenic person (diagnosed by DSM-III criteria) have a 10% chance of having or developing schizophrenia. Children of a schizophrenic have a 10% chance. Children of two schizophrenics have a 46% chance. Children with one schizophrenic parent and one schizophrenic sibling have a 17% chance.

### **Summary of Family Studies**

All family studies of well-validated psychiatric disorders indicate that virtually all psychiatric disorders are familial. What runs in families is the illness itself or closely related conditions. Table 2 lists psychiatric disorders and the conditions which run in the families of persons with the disorder.

### **Twin Studies and Heritability**

*Twin studies* are naturalistic experiments which help us to assess heritability. As *monozygotic (MZ) twins* share 100% of their genotype, and *dizygotic (DZ) twins* share 50% of their genotype, if MZ twin concordance significantly exceeds DZ concordance, this suggests—but does not prove—a genetic contribution to the disorder. For example, the MZ:DZ concordance ratio for major mood disorders, with bipolar disorder and major depressive disorder considered the same illness, is 65:14. Interestingly, if bipolar disorder and recurrent depression are viewed separately, neither is heritable. The MZ:DZ ratio for schizophrenia is 45:14.

### **Heritability Formula**

Heritability equals (MZ concordance minus DZ concordance) times two.  $H = (MZ - DZ) \times 2$ . For illness A, if MZ concordance is 50% and DZ concordance is 25%, the heritability of A is  $25\% \times 2 = 50\%$ .

### **Nongenetic Causes of Concordance and Discordance**

The reason twin studies do not absolutely prove heritability is that there are nongenetic causes of concordance and discordance. For example, MZ twins may be reared more similarly (e.g., dressed more similarly, referred to as “the twins,”) than DZ twins, which could—hypothetically—yield behavioral concordance explainable by social, not genetic causes. Interestingly, however, studies of MZ twins reared similarly show that they are as similar behaviorally as MZ twins reared differently (e.g., parents focus on the individuality of each).

**Table 2. Conditions which Run in Families of Persons with Psychiatric Disorders**

<b>DISORDER</b>	<b>CONDITIONS MORE PREVALENT IN FAMILY</b>
Bipolar disorder	Bipolar disorder, major depressive disorder, schizophrenia
Major depressive disorder	Major depressive disorder, bipolar disorder, schizophrenia
Schizophrenia	Schizophrenia, schizoid personality disorder, schizotypal personality disorder, bipolar disorder, major depressive disorder
Delusional disorder	Delusional disorder, paranoid personality disorder
Obsessive compulsive disorder	Obsessive compulsive disorder, Gilles de la Tourette syndrome
Panic disorder	Panic disorder, agoraphobia, social phobia, generalized anxiety disorder (GAD)
Agoraphobia	Agoraphobia, panic disorder, social phobia, GAD
Social phobia	Social phobia, panic disorder, agoraphobia, GAD
Generalized anxiety disorder (GAD)	GAD, panic disorder, agoraphobia, social phobia
Antisocial personality disorder	Antisocial personality disorder, somatization disorder
Somatization disorder	Somatization disorder, antisocial personality disorder
Anorexia nervosa	Anorexia nervosa, bulimia, major depressive disorder
Enuresis	Enuresis
Encopresis	Encopresis

MZ twins share the same placenta. The amounts of placental blood each twin receives during pregnancy are not identical (*transfusion effect*). Birth weights of MZ twins often differ. Here, the intrauterine environment, not genetic factors, could cause behavioral discordance.

### **Genetic Influences on Environment**

Further complicating matters is the fact that genetic factors influence the environment. For example, religiosity is heritable. If one is religious, one will attend church (mosque, synagogue, temple, etc.) more often than if one is not religious. In turn, events in church influence one's behavior.

### **Twin Studies in Psychiatric Disorders**

Twin studies suggest heritability of schizophrenia, major mood disorders, alcoholism, antisocial personality disorder, alcoholism, anorexia nervosa, obsessive compulsive disorder, posttraumatic stress disorder and autism.



### **Twin Studies of Personality Traits**

Studies of twins, sometimes combined with adoption studies, suggest heritability of achievement motivation, altruism, assertiveness, empathy, extraversion, *harm avoidance* (cautiousness, tendency to worry), introversion, leadership ability, neuroticism, *novelty seeking* (risk-taking, thrill-seeking), nurturance (capacity to provide affectionate care), *reward dependence* (sentimentality, eagerness to please), social closeness, traditionalism and well-being.

### **Adoption Studies**

*Adoption studies* eliminate the extent to which concordance of behaviors among biologic relatives is due to the shared environment. There are many varieties of adoption studies. For example, one could study the prevalence of a disorder or trait among adopted-away children of parents with and without the disorder. In one study, the prevalence of schizophrenia in adopted away children of biologic mothers with schizophrenia was higher than its prevalence among adopted away children of well biologic mothers.

Or, one could compare the prevalence of a disorder or trait among biologic parents of adopted away children with the disorder with the prevalence of the disorder among biologic parents of adopted away children without the disorder.

In a *cross-fostering analysis*, comparisons are made of the prevalence of the illness among children with and without the disorder, their biologic parents, and their adoptive parents.

### **Summary of Adoption Studies of Psychiatric Disorders**

Adoption studies suggest a genetic contribution to the development of schizophrenia, major mood disorders, alcoholism and other drug dependence, and antisocial personality disorder.

### **Restriction Fraction Length Polymorphism Studies**

*Restriction fraction length polymorphism (RFLP) studies*, covered in biochemistry texts, have associated some disorders with specific chromosomes. RFLP studies show “big gene” effects for some relatively rare disorders like cystic fibrosis and Huntington’s disease. For several common disorders, such as juvenile onset diabetes mellitus and Alzheimer’s disease, RFLP studies suggest that a network of perhaps 4–10 genes is associated with the disorder. For none of the common psychiatric disorders has a sledgehammer gene been found.

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# SECTION 3. EVERYDAY BEHAVIORS

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## CHAPTER 9. SEX

### BIOLOGY OF SEX

1. The basic *gender anatomic template is female*. Persons with the *SRY gene on the Y chromosome* become male by developing *androgen receptors*, and producing *androgen* and *Mullerian duct inhibiting substance*. Persons with an XY genotype who lack androgen receptors develop *androgen insensitivity syndrome*; they have the appearance, voice and identity of a woman but cannot develop ovaries or conceive children.
2. Androgen increases sex drive for both sexes.
3. The *skin* is the largest sex organ.
4. The *clitoris* is the only human organ whose sole function is pleasurable sexual arousal.
5. *The left testicle* is almost always lower than the right one; the testicles are never of equal height. This lets men sit with their legs crossed without shrieking.
6. Most sex partners suit their behaviors to each other. This is fortunate because, unmodified by a partner's needs, the *mean sexual response cycle in males* takes three minutes and that of *females* 15 minutes. Foreplay goes a long way.
7. *Circumcision* is medically necessary only for *phimosis*.

### AGE AND SEX

#### *Childhood and Adolescence*

1. By *age 5*, children can experience the full sexual response cycle, including orgasm. Fortunately they don't seek sex partners and don't have mature sperm or ova.
2. On average, *puberty* begins earlier in girls than in boys. This contributes to awkwardness between the sexes in the junior high school years. With each new generation, puberty begins earlier. Your children will start puberty by the time they're four (sorry, I'm kidding).
3. Almost all teenage boys, and most teenage girls, *masturbate*.

## **Middle Age**

The *physiologic intensity of sex* diminishes in middle age, but middle aged persons can enjoy sex as much as when they were younger. Usually, this reduction in physiologic intensity is greater for men than for women.

### **Male Physiology**

1. Typically, it takes longer for a middle aged man to have an erection than when he was younger. Some middle-aged men may, for the first time in their lives, request foreplay to stimulate erections.
2. The angle of the erect penis changes: In teenage boys standing up, the erect penis points upwards. In middle-aged men, the erect penis points parallel to the floor or slightly downwards. But not to worry, it still serves the same purpose.
3. The *volume of the ejaculate* decreases with age, and—for those readers for whom distance matters—the *distance* it travels is less.

### **Female Physiology**

In middle age, the *vaginal wall* narrows, the *labia majora* become smaller, and there is less *vaginal lubrication*. If the latter is a problem, it can be corrected with jelly lubricants or estrogen (if the latter is medically safe). These changes are gradual and precede menopause.

## **Old Age**

In old age, the *reduction in physiologic intensity* that began in middle age continues. But elderly persons can enjoy sex as much as when they were younger.

As a rule, elderly persons who enjoyed sex as younger adults are more apt to have and enjoy it as they age. But many elderly persons are widowed and have no partner.

*Inappropriate sexual behaviors* in old age suggest evaluation for brain disease. This is also true for younger persons.

## **PREGNANCY AND SEX**

Sex can continue throughout pregnancy, with some precautions about orgasm and vaginal bleeding. During *orgasm*, the uterus contracts, increasing the risk of third trimester premature labor. There is no evidence that sex with one orgasm during the first two trimesters poses a medical risk to mother or fetus, or that third trimester sex without orgasm poses a risk. After the 34th week, sex is mechanically awkward, but some couples continue sex.

If there is *vaginal bleeding* during pregnancy, medical evaluation should be sought promptly and sex should be avoided. This bleeding could signify placental abruption or ectopic pregnancy.

There is a *type of cunnilingus* in which the partner blows air into the woman's vagina. This is contraindicated in pregnancy; it can cause a fatal air embolism.

Following delivery, women who had an *episiotomy* can resume sex when the episiotomy scar is healed, usually 2–4 weeks postpartum.

## MENSTRUATION AND SEX

Some couples forego intercourse during menstruation for religious or aesthetic reasons. Others prefer sex during menstruation because pregnancy is less likely.

There is a theory, not yet proven, that *frequent* sex during menstruation increases the risk of *endometriosis*.

## HOMOSEXUALITY

If an illness is a condition that causes suffering or reduces functioning, *homosexuality is not an illness*. Homosexuals' only suffering comes from social stigma. The only "disability" is that homosexuals are *less apt to have children* than heterosexuals. According to sociobiology's *good uncle theory*, reduced procreativity doesn't matter because, on average, homosexuals earn higher incomes and are better educated than heterosexuals, making them better able to help family members who procreate, facilitating passage of the family's DNA into the next generation. Furthermore, a considerable minority of lesbians and gay men are married, or once were, and perhaps a million lesbian women and a million gay men are parents. About 6 million children have homosexual parents. In 1980, the American Psychiatric Association, under pressure from gay rights groups who insisted that homosexuals are not ill, voted to delete homosexuality from its list of disorders.

### *Biology of Homosexuality*

1. There are analogs of homosexual behaviors in over 60 species.
2. Homosexuality is *familial and heritable*. Brothers of male homosexuals and sisters of lesbians are more apt to be homosexual than brothers or sisters of heterosexuals. Among twins, the MZ:DZ concordance ratio for homosexuality is 40:10 to 40:15.
3. Male homosexuals are more likely than male heterosexuals to have *older brothers*.
4. Anatomy
  - a. The size of the *third intersitial nucleus of the anterior hypothalamus (INAH-3)* in gay men is intermediate in size between the size of INAH-3 in heterosexual men (largest size) and that of INAH-3 in heterosexual women (smallest).
  - b. The *suprachiasmatic nucleus of the hypothalamus* and the *anterior commissure* are larger in homosexual than in heterosexual men.

### *Gay and Lesbian Parents*

A spurious argument against gay and lesbian parents rearing a child is that because of role modeling or persuasion, their children would become homosexual.

However, children reared by gay or lesbian couples are as likely to grow up heterosexual as are children reared by heterosexual parents.

## **THE SEXUAL RESPONSE CYCLE AND SEXUAL DYSFUNCTIONS**

For both genders, there are five phases of the sexual response cycle: 1) appetitive; 2) excitement; 3) plateau; 4) orgasm; and 5) resolution. A common cause of sexual dysfunction in any of the phases is the key triad, which consists of 1) problems in the relationship, 2) performance anxiety and 3) fear of the consequences. Because the key triad can affect any phase of the cycle, *sexual dysfunctions often occur together* (e.g., erectile dysfunction with premature ejaculation).

### ***The Key Triad, a Common Cause of Sexual Dysfunctions***

**Problems in the relationship:** If a problem in the relationship between partners causes chronic resentment or disgust, sexual desire decreases. Treatment is to counsel the couple about the relationship.

**Performance anxiety:** Many persons mistakenly view sexual intercourse as a skill, ability, talent or *performance* rather than as a *source of pleasure*. Before performing, people naturally feel anxious about how they will perform. This is fine before a sporting event or speech, but it interferes with the autonomic components of sex (e.g., *parasympathetic discharge for arousal and penile and clitoral erection, sympathetic discharge for orgasm*). The patient or couple should be persuaded to view the goal of sex as pleasure or procreation, and assigned *sensate focus exercises* (p. 35). Couples having sex for the first time should appreciate that performance anxiety is common in early encounters and will diminish as mutual comfort increases.

**Fear of Consequences:** Another cause of anxiety about sex is fear of the consequences. If one fears pregnancy, a heart attack, a sexually transmitted disease, or being “caught in the act,” desire or arousal will be affected. Education about coping (e.g., advice about contraceptives, advice about sex in persons with heart disease) with the feared situation is indicated.

### ***Appetitive Phase of the Sexual Response Cycle***

The *appetitive phase* is the phase of *initial sexual desire*, influenced by a person's *lovemap*, which consists of personal preferences for the partner, the situation and the method. Disorders of the appetitive phase are 1) hypoactive sexual desire disorder 2) hypoactive sexual desire due to drugs or medical conditions; and 3) paraphilias.

### **Hypoactive Sexual Desire Disorder**

In *hypoactive sexual desire disorder*, there is notably reduced, or absent, interest in sex in a patient who wants to desire sex.

### **Hypoactive Sexual Desire Due to Drugs**

The following can reduce sexual desire: antidepressants, neuroleptics, anxiolytics, antihypertensives, digitalis, anticonvulsants, cimetidine, clofibrate, alcohol or street drugs.

### **Hypoactive Sexual Desire Due to Psychiatric or General Medical Conditions**

During most acute medical illnesses, sexual desire is diminished because the patient is concentrating on that illness. Some neuropsychiatric disorders reduce sexual desire frequently: epilepsy, somatization disorder, chronic bipolar disorder, major depression with melancholic features.

### **Paraphilias**

*Paraphilias* are persistent sex urges—acted upon or causing distress—where the sexual object is unwilling (e.g., rape), a child (*pedophilia*), nonhuman (e.g., sex with a dead person, termed *necrophilia*, or with a nonhuman animal, termed *bestiality*), or one of the partners is humiliated or suffers (e.g., sexual *sadism* or *masochism*). Another paraphilia is *exhibitionism*, in which a man exposes his penis to a stranger to surprise her, not to have sex with her.

Paraphilias are *much more common in men* than in women. Management includes treating coexisting conditions, using behavior modification and, for patients whose sexual behaviors endanger others, the antiandrogen *medroxyprogesterone*.

### ***Excitement Phase of the Sexual Response Cycle***

During excitement, the following occur:

1. clitoral and penile erection from engorgement with blood, not voluntary muscle contraction
2. nipple erection in both sexes
3. skin flushing of the chest, neck and face
4. breast or testicular enlargement
5. increased muscle tension, heart and respiratory rates, and blood pressure

Dysfunctions of the excitement phase are male erectile disorder and female arousal disorder.

### **Male Erectile Disorder**

In *male erectile disorder*, there is inability to attain or maintain erection.

### **Female Arousal Disorder**

In *female arousal disorder*, there is inability to attain or maintain lubrication.

The causes of male erectile disorder and female arousal disorder are the key triad and some general medical conditions and prescribed or street drugs. General

medical conditions include Leriche syndrome, diabetes mellitus, spinal cord transections and extensive peroneal surgery.

*Leriche syndrome* is occlusion of the aorta at its iliac bifurcation. *Diabetes mellitus* can cause a *peroneal peripheral neuropathy*, or a *central neuropathy affecting the hypothalamus, the limbic system and paralimbic structures*. The longer the duration of the diabetes, the greater the risk of neuropathy. *Spinal cord transection* makes it impossible for the mental experience of excitement to translate into erection or lubrication. However, some persons with spinal cord transection can have reflex erection or lubrication from direct genital stimulation.

Drugs that can cause erectile or arousal disorders include *antidepressants, neuroleptics, barbiturates, diuretics, alpramethyldopa, digoxin, fenfluramine, cimetidine and clofibrate*.

### ***Plateau Phase of the Sexual Response Cycle***

The plateau phase is an intensification of the excitement phase.

### ***Orgasm***

Orgasm begins with a feeling of *orgasmic inevitability*—that one is about to “come.” In males, four or five involuntary perineal muscle contractions force seminal fluid through the urethra. In females, 4–16 involuntary contractions of the outer third of the vagina are followed by sustained uterine contraction. Blood pressure, pulse and respiratory rate remain high.

Females can have *multiple successive orgasms*. After orgasm, males have a *refractory period*, lasting from perhaps 20 minutes to several days, during which they cannot experience the full sexual response cycle with orgasm.

Problems in the orgasm phase include premature ejaculation and female orgasmic disorder.

### ***Premature Ejaculation***

Because there is no “official” time when a man is expected to ejaculate, each couple defines when ejaculation is premature, meaning that it occurs sooner than the partners want. The causes are the key triad. There is no premature orgasm diagnosed for females; it must be extremely rare for a couple to complain that a woman “comes” too soon.

The couple is treated by assignment of home exercises in which the woman begins to masturbate the man, stops rubbing his penis before ejaculation is inevitable, and then resumes rubbing. Practicing this *stop-start technique* leads to ejaculatory control.

### ***Female Orgasmic Disorder***

In *female orgasmic disorder*, a woman wishing to have orgasm does not experience it, or she takes longer to have it than she wants. Of course, not every woman wants orgasm with every sexual experience. Causes include the key triad, local

genital pathology, endocrinopathy, alcohol and street drugs. If any of the key triad is the cause, treatment includes education, sensate focus exercises, and a *bridge maneuver* in which the woman first experiences orgasm with manual or oral clitoral stimulation, and then pairs clitoral stimulation with penile penetration.

***Resolution***

After orgasm, unless sex play resumes, a baseline non-aroused status returns.



## CHAPTER 10. SLEEP

Sleep begins with *serotonergic activity of the pontine median raphe nuclei*. This causes accumulation of *hypnogenic polypeptides from the circumventricular organ* outside the blood brain barrier. These peptides dampen activity of the *ascending reticular activating system*, inducing sleep.

### SLEEP STAGES

Normal adults have 4–6 90 minute sleep cycles nightly. The sleep cycle has five stages, stages 1–4 plus rapid eye movement (REM) sleep. We begin with stage 1, proceed to 2, 3 and 4, and then to REM. From the first REM, we move from 4 to 3 to 2 to 1, and back to the second REM, then 1, 2, 3, 4, REM, and so forth.

*Stage 1:* Light sleep with theta (3–7 cycles/sec.) on the EEG.

*Stage 2:* Also light sleep with EEG K complexes and sleep spindles (12–14 cycles/sec.).

*Stage 3:* Begins deep sleep with delta waves (< 3 cycles/sec.).

*Stage 4:* Deepest sleep, with more delta waves.

*REM sleep:* Has beta, alpha and theta waves.

### *Characteristics of REM Sleep*

The night's first REM period lasts several minutes. By the last cycle, before awakening in the a.m., REM lasts 20–40 minutes. During REM, there is 1) reduced skeletal muscle tone, except for the extraocular muscles, which move rapidly; 2) increased blood pressure and heart and respiratory rates; 3) penile and clitoral erection; and 3) dreams. REM sleep is when we dream. Persons awakened from REM sleep recall vivid dreams. *Nightmares* are dreams in which the dreamer is anxious, regardless of the dream's content.

### SLEEP AND AGE

The older we are, the less we sleep and the less stage 3 and 4 and REM sleep we have. For newborns, 75% of sleep is REM sleep. For the elderly, REM is less than 25%.

### SLEEP AND HEALTH

Sleep probably serves functions beyond rest and recuperation, but we don't know what they are. People tend to feel sleepy in mid-afternoon, even after a good night's sleep, and regardless of whether they had lunch. This tendency is utilized in *siesta cultures*, where work ceases mid-afternoon and resumes in the evening.

Persons who sleep less than four or greater than 10 hours per night are statistically more apt to have reduced life expectancy. Sleep abnormalities often accom-

pany acute general medical or psychiatric illness. Up to 30% of the population reports sleep problems.

### **Sleep and Mood Disorders**

In *major depression with melancholic features*, there is a shortened period between sleep onset and the first REM period (*shortened REM latency*), more frequent eye movement during REM sleep, and less slow wave sleep. There is more REM sleep early in the night than towards morning, a reversal of the normal REM sleep pattern.

*Manic* persons feel increased energy and reduced need to sleep, and can stay up through the night embarking on projects.

## **PRIMARY SLEEP DISORDERS**

### ***Narcolepsy***

*Narcolepsy* affects 0.1% of the population and is more common in males. Onset is usually between ages 15 and 30. It is familial and may be associated with a chromosome 6 abnormality. There is *inopportune REM sleep*—REM sleep at the wrong time—characterized by 1) sleep attacks, 2) cataplexy and 3) hypnogogic and hypnopompic hallucinations, as discussed below.

### **Sleep Attacks**

*Sleep attacks* are irresistible urges to sleep due to sudden daytime onset of REM sleep. If a narcoleptic person is in a risky situation like driving a car, there is usually enough time to pull over to the roadside and sleep. However, *accident rates* are higher for narcoleptics.

Before we knew enough to educate families and prescribe stimulants and antidepressants, the *divorce rate* in narcolepsy was 100%. Can you imagine your partner falling asleep during sex?

### **Cataplexy**

When narcoleptics with *cataplexy* have intense moods like anger, fear, or laughter, their muscle tone diminishes (as in REM sleep) and they slump to the ground. Usually, they can brace their fall to avoid injury.

### **Hypnogogic and Hypnopompic Hallucinations**

Normally, sleep begins with stages 1–4, not REM. Narcoleptics begin sleep with REM sleep: As they fall asleep, they begin to dream, which is experienced as a vague visual hallucination—a *hypnogogic hallucination*. They also have *hypnopompic hallucinations*, which occur while awakening.

## ***Sleep Apnea***

*Sleep apnea*, which affects 1–2% of the population and 40% of the elderly, is more common in males. There are two types: childhood onset central sleep apnea and adult onset obstructive sleep apnea.

### **Childhood Onset Central Sleep Apnea**

The physiology of *central sleep apnea*, which begins in infancy, is reduced responsiveness of the medullary respiratory center to CO<sub>2</sub> buildup, which automatically triggers breathing in healthy people. For obvious reasons, central sleep apnea can cause *sudden infant death*. The first sign of central sleep apnea is cyanosis upon awakening which gradually subsides by midday.

Diagnosis is confirmed in a pediatric sleep laboratory. Treatment is medication (e.g., theophylline) which increases medullary responsiveness to CO<sub>2</sub>. In the hospital, an alarm system is attached to the child's chest to warn the nurse or parent if the child stops breathing; waking the child restores breathing.

### **Adult Onset Obstructive Sleep Apnea**

*Adult onset obstructive sleep apnea* is associated with excessive daytime sleepiness and snoring. More common in very obese persons, it also occurs in thin persons. During sleep, the airway is obstructed in the nose (deviated septum or polyps), pharynx (enlarged tonsils) or trachea.

Complications include hypertension, reduced libido and cognitive impairment. Treatment includes weight loss in obese persons, a positive pressure air mask during sleep, or surgical repair by septum correction, polypectomy, uvulectomy, tonsillectomy or tracheostomy.

### ***Somnambulism (Sleepwalking)***

*Somnambulism* affects 15–30% of children and 1% of adults. Adult onset somnambulism is sometimes associated with drug abuse. Sleepwalking is familial.

Somnambulism is a *parasomnia*, a behavior during sleep that makes the sleeper appear to be awake. The patient arises during stage 4 sleep and walks, hesitatingly, with a puzzled facial expression and reduced attentiveness. If you speak to the somnambulist, he or she may look at you, but won't converse or respond to your requests. If you awaken the walker forcefully, by shaking or speaking loud, the patient will become delirious, which doesn't help. After 10–30 minutes, the patient will lie down en route, or back in bed, resume regular sleep, and not recall the walk. The somnambulist is at risk for *injury*; for example, he or she could walk into traffic.

If you are present during the walk, accompany the patient to prevent injury. But routine surveillance is impractical. Treatment is a *stage 4 sleep suppressant*, usually a benzodiazepine like *clonazepam (Klonopin)* for 3–4 months, followed by tapering the drug to prevent dependence. Until the sleepwalks stop, the environment

should be protected, including locking doors, closing windows or, in severe cases, tethering the sleeper to the bed.

### ***Pavor Nocturnus (Night Terrors)***

Like somnambulism, *pavor nocturnus* is a parasomnia that occurs in stage 4. It affects 6% of children and 1% of adults. It is not a response to a dream; dreams occur only during REM. The patient sits and looks terrified, cries or screams, cannot be consoled, resumes regular sleep 10–30 minutes later, and does not recall the event. If the night terrors are frequent and disrupt the family, the child should be treated with a stage 4 sleep suppressant like *clonazepam* for 3–4 months. Unless the child is also a sleepwalker, safety precautions are unnecessary.

### ***Sleep Paralysis***

In *sleep paralysis*, which may occur with narcolepsy or by itself, the patient cannot move for several seconds while awakening. Although sleep paralysis is benign, it is frightening if it lasts more than several seconds—say, 5 to 10 seconds—and the patient appreciates being gently shaken by a bed partner to facilitate initial movement.

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# SECTION 4. HUMAN DEVELOPMENT

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## CHAPTER 11. CHILD DEVELOPMENT

### ERIKSON'S, FREUD'S AND PIAGET'S THEORIES

The following are the stages of development according to Erik Erikson, Sigmund Freud and Jean Piaget (Table 1). *Erikson* viewed developmental stages as *normative crises*, meaning age-appropriate accomplishments (e.g., early autonomy in a two year old) contrasted with the consequence of non-accomplishment (e.g., shame and doubt persisting later in life). *Freud* wrote that events centering around *erotogenic* (pleasure-giving) zones influence later behavior. For example, in the anal phase, strict toilet training leads to stubborn, obsessive behavior later. *Piaget* focused upon *cognitive* (intellectual) progress. For example, a six month old child believes that a ball that rolls out of sight behind a chair has disappeared, while a two year old knows otherwise.

### PRINCIPLES OF DEVELOPMENT

*Development* is changes in a person's abilities resulting from an interaction between physical *growth*, physiological *maturation*, and *environmental influence*. Not all newborns are alike, and their rates of growth and maturation also vary. Under severe stress (e.g., after a child's hospitalization), a developmental milestone (e.g., bladder control) may be transiently lost (*regression*).

### *Neuroanatomy of Development*

Development of skills proceeds stepwise (e.g., standing before walking). Neuronal growth and maturation are dramatic, begin during gestation, and include competition between cells and *pruning*, the selective chemically-induced deletion of unused neurons. Timing of a brain injury is crucial. Before age 5, if one hemisphere is injured, the other one can take over functions very well. For example, after left hemisphere damage in a 3 year old, the right hemisphere assumes language functions. Between ages 5 and 10, the right hemisphere may still assume some language functions after left hemisphere injury, but by this time, there will be some permanent language impairment.

**Table 1.**

<b><u>AGE GROUP</u></b>	<b><u>ERIKSON</u></b>	<b><u>FREUD</u></b>	<b><u>PIAGET</u></b>
<b>Infancy</b> (Birth–18 months)	<i>Basic trust</i> (that others can be counted upon) vs. <i>mistrust</i>	<i>Oral period</i> (pleasure in sucking); if needs are met, child later can meet needs of others	<i>Sensorimotor period</i> ; learning by direct action on environment; first, out of sight, out of mind; later, object permanence
<b>Toddler</b> (18 months–3 years)	<i>Autonomy</i> (I have a mind of my own) vs. <i>shame and doubt</i>	<i>Anal period.</i> Child's readiness and self control in toilet training vs. parental demands and control; with parental over-control, child may become stubborn or compulsive as an adult	<i>Preoperational phase</i> in which intuition supersedes logic; reversibility conservation (see below) not present
<b>Preschool Child</b> (3–6 years)	<i>Initiative</i> vs. <i>guilt</i>	<i>Oedipal period.</i> Child wants to marry opposite sex parent, fears retaliation by same-sex parent; resolved through identifying with same-sex parent	Preoperational phase continues, with <i>animism</i> (moving things are alive and have feelings, e.g., cars sleep at night), <i>egocentrism</i> (e.g., "the sun shines to keep me warm"), <i>artificialism</i> (e.g., "rain is caused by God pouring water from a can")
<b>School Age</b> (6–12 years)	<i>Industry</i> (pride in mastering tasks in school, hobbies and sports) vs. <i>inferiority</i>	<i>Latency</i> (sublimation of sexuality into schoolwork and play)	<i>Concrete operations.</i> During phase, learns <i>conservation</i> (e.g., quantity of liquid poured from tall thin glass to short wide one stays the same) and <i>reversibility</i> (As $20 - 10 = 10$ , so too must $10 + 10$ be 20)

**Table 1 (cont.)**

<b>AGE GROUP</b>	<b>ERIKSON</b>	<b>FREUD</b>	<b>PIAGET</b>
<b>Adolescence</b> (teenage years)	<i>Identity</i> (Who am I? What are my strengths, weaknesses and motivations?) vs. role confusion	Intensification of sexuality	<i>Formal operations:</i> Capable of abstract complex reasoning and of <i>postconventional morality</i> (personal ethical conduct may transcend routine rules)

### ***Childhood Behaviors Predicting Later Behaviors***

To an extent, childhood phenomena predict later childhood and even adult behaviors. High birth weight is associated with adult obesity. Children who don't get along well with others are more apt to require psychiatric treatment later in childhood or in adulthood. IQ tends to predict school performance.

### ***Influences on Development***

#### **Inheritance**

Many factors affect development. *Inheritance* is important. Family, twin and adoption studies suggest a genetic contribution for handedness, intelligence, personality traits, sexual object choice, susceptibility to illnesses, rhythms of spurts and lags in development, and age at menarche.

#### **Prenatal Care**

Good *prenatal care* correlates with better childhood health. Maternal illnesses, medications, toxins, and malnutrition can cause fetal death and childhood illness. Complicated, premature (<37 weeks) or postmature (>42 weeks) deliveries are associated with later negative consequences.

#### **Socioeconomic Status**

*Low socioeconomic status* (SES) is associated with greater vulnerability to many illnesses and poorer access to medical care (p. 11).

#### **Gender**

People treat boys and girls differently. When research subjects are given an unfamiliar infant to hold, they describe the infant based upon whether they are told the infant is a boy ("energetic, strong") or a girl ("sweet, pretty").

#### **Temperament**

The fit between a child's temperament and family expectations and responsiveness is crucial. *Temperament* has eight components: 1) attention span, 2) activity level, 3) distractibility, 4) responsiveness to stimulation, 5) regularity

(rhythmicity) of physiologic functions like sleep or appetite, 6) adaptability, 7) tendency to approach or withdraw from people and 8) mood quality (e.g., friendly and pleased or unfriendly and sad).

### **Attachment and Bonding**

Development occurs in the context of the *parent-child relationship*. On the first day of life, newborns synchronize their actions with an adult voice. Within a week an infant can distinguish by smell his or her mother's breast pad from those of other mothers. Within a month infants can distinguish their parents from others. *Attachment* is not to one person alone, but to a hierarchy of people. In all cultures, for the majority of children, the mother is first in the hierarchy, and the father (if present) is high on the list.

### **Stranger Anxiety**

Although an infant *begins* to distinguish among people from the first week, between ages 5 and 9 months a dramatic change occurs in a baby's responses to family and strangers. The child becomes scared and withdraws when a stranger is around (*stranger anxiety*). This anxiety is greater when the stranger is an adult (other children are less frightening), when the stranger makes direct eye contact or stands near the infant, or when the setting is unfamiliar. It is less when the stranger sits near the parent and responds to the child's cues.

### **Separation Anxiety**

*Separation anxiety* starts at about 6 months, and disappears by three years: When the parent leaves the house, the child becomes upset.

### **Quality of the Relationship**

The quality of the bond between caregiver and child is based more upon responsiveness of the caregiver to the child's cues than it is to time spent with the child. Children reared in kibbutzes in Israel were supervised daily by communal caregivers while their parents worked, but were more strongly attached to their parents than to the caregivers.

## **STAGES OF CHILDHOOD**

### ***Gestation (Pregnancy)***

The newborn is actually 37 weeks old. By 28 weeks gestation, the fetus has all the neurologic reflexes of newborns. By mid-pregnancy, fetuses hear as well as do newborns.

### **Anticipatory Guidance for Parents**

Newborns should be driven home from the hospital in a car seat attached to the rear seat with the child facing the rear of the car. Parents should be attentive to the



child's older siblings, who normally resent the attention given the newcomer, and who tend to regress, such as by clinging or bedwetting.

### ***The Neonate (Newborn)***

#### **Sensory Skills**

Sensory skills of newborns are excellent; for example, they can distinguish among tastes. They may cry when another child cries (empathy?), but don't respond to most loud noises (a protective mechanism?). They may mimic a parent's interesting actions (e.g., protruding the tongue).

#### **Reflexes**

The newborn has many reflexes: The child automatically grasps a finger placed in the palm of his hand (*grasp reflex*, which remains until the child is 2 months old), turns in the direction of stroking on the cheek (*rooting reflex*, which remains until age 3 months), makes stepping movements when held upright (*walking reflex*), and has a *Babinski reflex*, which remains until age 12 months.

The newborn breathes irregularly, sleeps 75% of the time (half of the sleep is REM sleep) and is capable of some conditioning.

### ***Infancy (Birth–18 months)***

#### **Smiling**

Spontaneous smiling, termed *endogenous smiling* because it is not responsive to actions of others, begins within a week and disappears by 3 months.

By a month, infants smile when they are pleased. At 2 months infants smile at a face (the *social smile*). At 3 months, facial expressions reveal a variety of emotions. At 4 months they laugh.

#### **Transitional Objects**

About 67% of American children (percentages vary from country to country) use a *transitional object*, usually a soft object like a stuffed animal or blanket, for comfort when they are afraid, alone or tired. Some children use this object well into their grade school years.

#### **Motor Development**

On average, the following occur: At 2–4 months the child rolls over; at 6–8 months he or she crawls, and at 7 months sits unsupported. At one year the child stands unsupported, walks if a hand is held and speaks his first word. At 13–15 months, the child walks alone and can build a tower of two cubes.

#### **Language Development**

At 3–4 months the baby *babbles* (mixes consonants and vowels). The first words are echoes of words spoken to them (*echolalia*). The first meaningful word is usually something familiar (e.g., *mama*).

### **Anticipatory Guidance for Parents**

Before the child can crawl, the house should be child-proofed. For example, sharp objects should be inaccessible. Because of the concentration required when the child first walks, the child should not walk and eat simultaneously. When stranger anxiety begins, guests should be advised not to be offended when the child cries.

### ***The Toddler (18 months–3 years)***

#### **Autonomy**

*Toddlerhood* begins at about 18 months, with *negative head-shaking (normal negativism)*, where the child shakes his or her head “no” to a parental request even when the child ordinarily would say yes, asserting “I have a mind of my own (*autonomy*).”

#### **Language**

The two year old can speak two-word sentences (2 years 2-word sentences; 3 years, 3-word sentences; four, four words; and five, five). The two words consist of the essential noun and verb, like “dada sing.” Though spoken vocabulary is small (e.g., 50 words at 24 months), comprehended vocabulary exceeds 500 words. *Stuttering* is normal before age 3. *Tantrums* occur because toddlers can’t see events from others’ viewpoints, hide anger, or delay pleasure.

By 24 months most children can run, climb stairs and throw a ball. Toddlers cannot tell TV ads from regular TV programs, making them especially vulnerable to advertising. It is unethical to target TV ads to three year olds.

#### **Toilet Training**

Most children are bowel trained by 3 years, and bladder trained by 4. Parents should begin toilet training when the child gives cues of being ready. Clues to readiness are squatting to expel feces after a meal, displeasure with soiling, and curiosity about the toilet.

### **Anticipatory Guidance for Parents**

Rearing toddlers requires respecting the child’s developing autonomy and pride in control. The best toys are those where the child crafts the play, like puppets, blocks, and crayons. Tantrums can be managed by 1) appearing not to notice the child, 2) distracting the child, or 3) giving the child a “time out” period in his or her room, along with the explanation “You must go to your room. When you can control yourself you may come back.”

### ***The Preschooler (ages 3–6)***

#### **Language**

By age 4 the vocabulary contains thousands of words. The 3 year old can use pronouns, and state age, sex, and full name. The 4 year old can use prepositions and

knows the meaning of many important adjectives like hungry and angry. Five year olds can use the future tense, which requires understanding the concept of time.

### **Motor Skills**

A 3 year old can ride a tricycle. A 4 year old can throw a ball overhand. Many 5 year olds can begin to learn complex motor activities like figure skating. By age 4, the child can engage in interactive, *cooperative play*. Before age 3, children play side by side without interacting (*parallel play*). Beginning at age 3 and then throughout childhood, children prefer playing with same-age same-sex friends.

### ***The School Age Child (6–12)***

MDs usually enjoy examining and treating children in this age group, because they tend to cooperate and to understand questions and instruction. They enjoy riddles and jokes, and can practice skills they value.

Although lacking full capacity for abstract reasoning, school age children can categorize; for example, given wooden beads, some blue and some green, and asked if there are more wooden beads than green beads, the child responds “wooden.” Friendships with same-sex *chums* occupy as much as half the child’s time.

### ***Adolescence***

#### **Puberty**

Adolescence begins with puberty. During puberty, organs (including bones) grow at different rates, leading to a gangly appearance and the teenager’s preoccupation with this. Don’t kid teenagers about their appearance. Puberty usually begins earlier in girls than in boys, causing gender differences in social maturity and awkwardness between the sexes in the junior high years. Despite early adolescent gangliness, motor development approaches adult skills, and some extraordinary teenagers become professional athletes.

#### **Autonomy and Identity**

During adolescence there is improved self control, strides in autonomy, finalization of sex object preference, clarification of body image, and development of *identity* (e.g., “Who am I?” “What do I want from life?”). Parents should be very supportive and only mildly to moderately controlling.

#### **Sexuality**

Sexual feelings are intense. The majority of 19 year olds have had intercourse. About one-third of sexually active teenagers become pregnant, and 40% have abortions. Sexually transmitted disease (STD) is common.

#### **General Health**

Between ages 5 and 18, people have fewer doctor visits than in any other age group. The most common cause of death is *accidents*. Many accidents can be prevented by *anticipatory discussions* (e.g., Drive carefully, the roads are slick).

### **Doctor Visits**

Compared to younger ages, where the family is really the patient, the teenager is the primary patient. MD visits with teenagers should include some time alone with the patient. Teenage girls should be taught about breast self examination and teenage boys told to observe for scrotal or testicular swellings. Testicular cancer, though not common, can occur in teenagers. Teenagers should be warned about the consequences of smoking. Although smoking by adults has declined in the US in the past decade, *smoking by teenagers, especially girls, has increased.*

## **SPECIAL SITUATIONS**

### ***Adoption***

Adopted children are more apt to have behavior problems than their nonadopted counterparts. The reasons might include genetic factors, diminished self esteem over being adopted, and preference by some family members for biological relatives. Adoptive parents should discuss adoption early in the child's life even before the child comprehends adoption's meaning. Hearing this information from someone else could be frightening. Adoption should be presented to make it a source of pride for the child; for example, "We wanted to have a child badly, and we were so happy when we adopted you." During adolescence or adulthood, some adopted children ask to meet their biological parents, and recently, authorities and parents have been more willing to honor this request.

### ***Hospitalization***

About one-third of children are hospitalized at least once. Hospitalization is stressful and often associated with regression following discharge. If hospitalization cannot be avoided, explain the reason for the hospitalization, describe in advance the hospital setting and allow generous visiting hours and parental rooming-in. Table 2 includes some factors pertinent to hospitalization.

### ***Medication***

In prescribing for children, take body weight into account (e.g., mg/kg).

### ***Divorce***

At current divorce rates, 40% of parents will divorce. Children of divorce have more behavioral problems than those who live with both biological parents. Table 2 lists age-related responses to divorce. The post-divorce period involves suffering for both children and parents; only 10% of children feel relief. Economic status usually declines for both parents and social supports often decrease. If marital counseling cannot avert the divorce, the MD should encourage continued involvement with the child by both parents. Children should be told that they did not cause and cannot mend the divorce, and that they needn't take sides.

**Table 2 Age-Related Responses to Hospitalization and Divorce**

**Hospitalization**

*6 months-4 years:* Greatest distress at this age. Stranger and separation anxiety. Poor comprehension of illness.

*4-7 years:* May feel responsible for causing illness. Knows separation from parents is not permanent.

*School age (7-12):* Can make friends in the hospital.

*Teenage:* Good comprehension of illness and need for treatment; possible problem with self image.

**Divorce**

*2.5-5 years:* Regression, irritability, separation anxiety, sleep disturbance, reduced play, fear of being abandoned by custodial parent, feeling responsible for the divorce.

*5-8 years:* Open grieving, sobbing, feeling rejected, pining for departed parent, decline in schoolwork.

*8-12 years:* Feigned nonchalance, hesitation to discuss the divorce, decline in peer relations, severe anger at one or both parents, reduced school performance.

*Teenage:* Depression, suicidal behaviors, sexual activity, truancy, drug abuse.

(modified from Stein MT. Children's encounters with illness. In: Dixon SD, Stein MT, eds. *Pediatric Behavior and Development*. Mosby Year Book, 1987; Wallerstein JS. Separation, divorce and remarriage. In: Levin MD et al, eds. *Developmental-Behavioral Pediatrics*. Saunders, 1983)

**Child Abuse**

Over a million children are abused yearly in the U.S., causing about 2000 deaths annually. Children of all ages may be abused but children under 5 are most often the victims, as they cannot readily escape, complain or fight back. Often, child abuse is first identified in ERs, where it is suspected if the child has multiple injuries, certain sites of injury (especially the lower back or buttocks), or multiple visits due to injury. Child abuse often occurs in children who don't live up to their parents' expectations or gratify their parents' dependency needs, or who are scapegoated, premature, or unplanned. In perhaps half the cases the abusing parent was himself abused. In many cases, the parent is immature or a drug user who does not foster a strong parent-child bond by meeting the child's needs, even though he or she may make a good initial impression and attribute the family's problems to others.

**Sexual Abuse**

Sexual abuse is a subtype of child abuse. Uncles are the most frequent intrafamilial sex abusers, followed by stepfathers and fathers, first cousins, and brothers.

**Premature Birth**

*Premature birth* is a common theme in much childhood psychopathology. It is more common when the parent is single, poor, a young teenager, a substance

abuser, or a smoker. The child is more likely to be scapegoated and abused and fail to thrive, and is less likely to be held and given sufficient attention. Parents are more likely to feel distance from the child.

### ***Failure to Thrive***

*Failure to thrive* is a final common pathway of subacute or chronic systemic medical conditions (e.g. ear infections) or deprivations. Failure to thrive is most common in infants and includes delayed growth and development and irritability or apathy.

### ***Accidental Poisoning***

Toddlerhood is the time of greatest risk for *accidental poisoning*. Some correlates of accidental poisoning include previous poisoning, attention deficit hyperactivity disorder, and low socioeconomic status.

### ***Runaways***

Many teenagers, girls more than boys, *run away* from home. Often, there is some problem in the family, like an abusive alcoholic parent, which is intolerable to the teenager. Most runaways eventually return home. Runaways have an increased prevalence of depression and sexually transmitted disease and adult sociopathy. Some agencies help runaways.

### ***Stealing and Shoplifting***

Probably most school-age or teenage children steal something just for the excitement and to see if they can get away with it. Out of context, the meaning of an act of *shoplifting* is impossible to determine; it could mean anything from a normal behavior to an early sign of sociopathy.

### ***Teenage Pregnancy***

Many teenagers marry and become pregnant—not necessarily both, and not necessarily in that order. *Teenage pregnancies* have increased medical risks, in part because of a tendency for teenagers to take poorer care of themselves prenatally. Teenage pregnancies predict future teenage pregnancies and increased risk of divorce and school dropout. The earlier the first pregnancy, the lower the future income and the greater the risk of being on welfare eventually.

### ***Mental Retardation***

Prematurity and *low birth weight* are the factors most frequently correlated with *mental retardation*, which is more common in boys. For the vast majority of the retarded, the cause is unknown. Causes can include anoxia, head trauma,

infections, single or multiple genes, defective chromosomes, *pseudoretardation* due to deafness or an artifact of poorly-conducted I.Q. testing. *Alcohol*, the most common teratogen and the most common preventable cause of retardation, causes *fetal alcohol syndrome*: low birth weight, small size, *microgyria* and an *elongated philtrum* (the groove from below the nose to the upper lip). *Cocaine* is also teratogenic. *Fragile X syndrome* and *Down syndrome* are common genetic causes of retardation. Down syndrome (*trisomy 21*) can occur with 46 (*translocation Down syndrome*) or 47 chromosomes and is more common with older maternal or paternal age.

### ***Autistic Disorder***

Like mental retardation, *autism* is more common in boys and affects about 2.5/100,000 children. The autistic child shows virtually no interpersonal relatedness with parents or other children; for example, unlike most children, he does not position himself in anticipation of being picked up. The child has below average intelligence and is mute or aphasic, or has reduced emotionality or gesturing with speech (*dysprosodia*). Frequent twirling, rocking, headbanging, or other stereotyped or agitated behaviors are common. The disorder is chronic and severe but deterioration does not usually occur. *Asperger's disorder* is a mild form of autism.

### ***Rett's Disorder***

*Rett's disorder*, which affects only females, is a degenerative disorder with deterioration of motor and cognitive functions starting at around 18 months of age. Findings include handwringing, *stereotypies* (complex, repetitive purposeless movements like rotating one's arms), awkward gait and decelerated head growth. Death usually occurs by age 30.

## CHAPTER 12. AGING, DYING AND DEATH

### THE AGED

#### *Numbers*

The aged are the fastest-growing age group in the population. There are now about 23,000,000 Americans over 65, about 10% of the population. By 2030, the aged will be about 20% of the population. This results partly from the increasing American life expectancy.

#### *Social Problems*

The aged have many *social problems*: They are *poorer* than other groups, with only 25% in the labor force. Pensions are usually fixed incomes vulnerable to inflation. Many of the elderly are agricultural workers, and many cannot afford telephones. They are 10% of the total population, but 20% of the poor.

They are *more isolated* because of disability, spousal death and death of friends, a greater chance of being hospitalized when ill, greater geographic mobility in recent decades, and decline of the extended family. They are less well schooled. Also, our culture emphasizes the future over the present and past. Elders are often seen as less attractive and less valuable, and are often stereotyped by persons with minimal exposure to them.

They are *medically-underserved*, receiving fewer consultations for the same illnesses, with cognitive dysfunctions often missed, MD underestimation of the prognosis of their diseases, few geriatricians (many find it a depressing specialty), and underemphasis on primary prevention and overemphasis on maintenance. In one recent study, only 50/120 medical schools formally taught about aging.

#### *Physiology of Aging*

*Physiologically*, there is greater variability among the elderly than among the young. They are more susceptible to *drug reactions* and therefore require lower doses, typically 50–67% of a standard adult dose. They have only 10% of the *immune capacity* of an adolescent. There is a loss of height, diminished taste and smell, impaired dentition, decreased *fluid intelligence* (problem-solving ability with new information), EEG slowing in the alpha, delta and theta frequencies, diminished serum estrogen levels, increased brain monoamine oxidase, increased plasma norepinephrine in response to stress, and increased frequency of psychiatric illness (with depression and dementia most common). Declines in intellect are often related to problems of systemic health such as increase in blood pressure and treatment for it.

However, *crystallized intelligence* improves to age 50 and often holds steady or increases after that, and some persons maintain cognitive excellence or brilliance. First episodes of *mania* in the elderly are less severe than for the young, and are



associated with fewer recurrences. In general, diseases curable in the young are curable in the elderly. Results of psychotherapy in the elderly can be excellent because many of the elderly perceive themselves as having little time to waste.

*Stability of the environment* is important to the elderly. Moving an elderly person to more modern facilities, or hospitalizing him, can worsen his mental status and overall health.

## **DYING AND DEATH**

### ***Types of Death***

Before the 1960s death meant cessation of life functions and was diagnosed by inspection for movement, assessment of pupillary light reflexes, palpation for pulses, and listening for heart sounds. Now there are two “types” of death.

*Somatic death* refers to the above description. *Brain death* occurs when the heart and kidneys still work, the lungs are mechanically aerated, and the brain is totally nonfunctional, with somatic death virtually certain in 3 months. *Harvard criteria for brain death* are unresponsivity, no movement or breathing, no reflexes, and a *flat EEG*.

### ***Kubler-Ross Stages of Dying***

In 1969, *Kubler-Ross* described normal behaviors of dying persons and their families: *denial*, *anger* (e.g., at oneself for imperfect self-care, at MDs for failure to promptly diagnose and cure), *bargaining* (the posture that if one behaves well, one will somehow be rewarded) *depression* (see below), and *acceptance* (a composed coming-to-terms with death).

### ***Bereavement Versus Depression***

Clayton surveyed *bereaved* relatives of recently-deceased persons and found that many mourning relatives had symptoms seen in clinical depressions, symptoms which usually lasted about 6 months with no medical treatment required. These symptoms included sadness, crying, anxiety, sleep and concentration disturbance, and appetite and weight loss. Table 1 contrasts bereavement and depression.

### ***Causes of Death***

Table 2 reviews causes of death for each age group.

**Table 1. Comparison of Bereavement and Depression**

	<b>Bereavement</b>	<b>Depression</b>
Perception of being ill	Person usually knows he or she is not ill.	Person usually believes he or she is ill, but will not usually label illness as depression.
Preceding events	Always follows an event, usually death of a loved one.	May or may not follow a notable event.
Presence of a full depressive syndrome	Rarely. Usually has only a few symptoms.	Usually is readily diagnosed and has many symptoms.
Suicidal ideation	Occurs in <10% of bereaved persons, usually with an idea of joining the dead person, rarely associated with suicidal intent or serious attempts.	Most depressed persons have suicidal ideation, many have suicidal intent, and many attempt or commit suicide.
Management	Treatment not usually sought or needed, at most supportive or a support group.	Treatment almost always needed

**Table 2. Causes of Death by Age**

<b>Overall for all Ages</b>	<b>Birth to Age 4</b>	<b>Ages 5–14</b>	<b>Ages 15–24</b>
1. Heart disease	1. Accidents	1. Accidents	1. Accidents
2. Cancer	2. Congenital disease	2. Cancer	2. Homicide
3. Stroke	3. Cancer	3. Congenital diseases	3. Suicide
4. Chronic obstructive pulmonary disease	4. Homicide		4. Cancer
5. Accidents			5. Stroke
6. Pneumonia and influenza			6. Chronic obstructive pulmonary disease
7. Diabetes mellitus			
8. HIV infection			
9. Suicide			
10. Homicide			
<b>Ages 25–44</b>	<b>Ages 45–64</b>	<b>Ages 65 and Over</b>	
1. Accidents	1. Cancer	1. Heart disease	
2. Cancer	2. Heart disease	2. Cancer	
3. Stroke	3. Pneumonia and influenza	3. Stroke	
4. HIV infection	4. HIV infection		
	5. Stroke		

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# SECTION 5. BEHAVIORAL ASSESSMENT

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## CHAPTER 13. INTERVIEWING

### GOALS

The goals of an initial medical interview are to *obtain data to make a diagnosis*, to develop the *rapport* that enhances the patient's *compliance with care*, and to *begin the inspection and mental status portions of the physical examination*. Experienced MDs usually develop a *differential diagnosis* and a *working diagnosis* within the first several minutes of the interview, and then proceed methodically to confirm or exclude their initial impressions.

### CONTENT OF THE INITIAL INTERVIEW

The major components of the interview are the identifying data and chief complaint, the history of the present illness including age of onset of the first episode of the illness, past medical history, family history, personal and social history including childhood development, substance abuse history and systems review.

### TECHNIQUES TO FACILITATE RAPPORT AND DATA COLLECTION

#### *Making the Patient Comfortable*

Ideally, you should interview the patient in a quiet private room or office, in a comfortable chair or a hospital bed, with both you and the patient seated (equal positioning conveys equal status) at a *kitty corner angle*, not "head on." If there is a desk, you and the patient should sit at adjoining sides of the desk, not fully separated by it. In unavoidable circumstances, such as having to interview a patient on a gurney in an ER, apologize that the setting is not optimal and add "but we'll do the best we can."

#### *Starting the Interview: Introduction, Development of the Chief Complaint and Present Illness*

Introduce yourself, state *your role* (e.g., consultant, medical student) and the purpose of the interview if these are not already obvious, and then proceed with a general open-ended statement like "Tell me what is the problem that brings you here" or "Tell me about your health." This *chief complaint* leads into the *history*

*of the present illness*, about which you must be familiar in every visit. If the patient cannot present a chief complaint or present illness, obtain data from *additional sources* like the chart or a nurse or a relative. If your question “What is the problem?” yields a reply of “I have no problem,” or the patient cannot present the chief complaint or present illness clearly, the chief complaint should also include *your perception of why the patient is there*.

### ***Balancing Open Ended and Closed Ended (Direct, Directive) Questions***

If possible, as you begin the interview and at various points later on, ask *open-ended questions* and *listen* while the patient answers spontaneously.

#### **Open-Ended Questions**

*Open-ended questions* solicit 1) the patient’s spontaneous perceptions of his or her problems and 2) detailed answers. Examples of open-ended questions are “What’s the problem?” “What was the problem that caused you to come to the hospital?” “You said you are depressed. Could you tell me more about that?” “How did the depression affect you?” “What were the symptoms of your depression?” This (1) improves rapport by demonstrating interest in what the patient says; (2) allows articulate patients to summarize their histories more efficiently than if you ask dozens of closed ended (direct, directive) questions; and (3) permits assessment of the patient’s language skills and deficits. There is a common misperception that directive questions are more efficient than open-ended ones, especially if time is limited. For most patients, if you avoid open-ended questions, you are making a big error.

#### **Closed-Ended (Direct, Directive) Questions**

Inevitably, you will also ask many *closed-ended (direct, directive) questions* to clarify what the patient has told you and to round out the details of the history. Closed ended questions solicit brief answers. Examples include “You said you have trouble sleeping. Is it trouble falling asleep, or staying asleep, or both?” or “How old are you?” For some patients, though, closed-ended questions yield elaborate answers.

#### **Patients With Severe Psychiatric Illness or Cognitive Dysfunction**

For some patients who have severe cognitive dysfunction or severe psychiatric illness, you must ask primarily closed ended questions, to speak softly and shift your posture minimally.

#### ***Knowing the “Scripts”***

It is harder for some medical students to know what questions to ask than it is for them to talk empathically to patients. The reverse is true for some practicing MDs. For each common syndrome in each specialty, you must, by memorizing

and practicing, know “the script”; that is, the syndrome’s key symptoms and how to ask about these.

Which script you use depends on what is the chief complaint and what diagnostic clues you have obtained. For example, if a patient appears worried and mentions suicide, this will require a “depression script,” which begins with an open ended question like “Tell me more about that,” and proceeds to question about sleep, appetite, weight, enjoyment of activities, ability to work, self-esteem, hope, guilt, suicidal thoughts, medications and street drugs. Table 1 lists scripts for mania and alcoholism.

**Table 1. Screening Questions for Past Mania and Past Alcoholism**

**Past Mania or Hypomania**

“Was there ever a time in your life when, for days or weeks or months, you felt full of energy, were very active doing lots of things, went without sleep to work on projects, had thought after thought come into your head, had people comment you were speaking fast, were very irritable or happy or moody, thought you had special talents, spent more money than you could afford, or things like that?” If the answer is no, the patient probably never had a manic episode.

**Past Alcoholism**

(1) “Was there ever a time when you or anybody else thought that you had a drinking problem?” Then, if the answer is yes, or if you still think alcoholism is a possibility, ask (2) “Ever get shaky if you couldn’t have a drink?” “Ever have a blackout, where you’d been drinking and then couldn’t remember what you’d done while you were drinking?” “Ever have a seizure?” “Ever go on a bender, where for several days all you did was drink, where you didn’t work or engage in your hobbies or help your family?” “Ever have a hangover?” “Ever have jaundice, hepatitis, or cirrhosis?” “Ever drink first thing in the morning or instead of breakfast?” “Ever have to limit the times you could drink because, if you started drinking, you couldn’t stop?” “Ever have trouble with your family because of drinking?” “Ever have a fight while you were drinking?” “Ever have a DUI (arrest for driving under the influence)?” “Ever have work problems, or get suspended or fired from a job, because of drinking?” “Ever feel guilty about drinking?”

Asking all these alcoholism questions takes 1–5 minutes at most. If all the answers to (1) or (2) are no, either the patient was never alcoholic or is lying to conceal alcoholism.

***Facilitation***

*Facilitation* is encouraging communication by gestures, manner or words without specifying the kind of information sought. Facilitations include the following:

1. a concerned facial expression
2. a smile
3. a nod
4. a word or phrase like “yes,” “uh-huh,” “go ahead,” or “I see.”
5. comments like “What do you mean?” or “I don’t understand.”

## **Reflection**

In *reflection*, you repeat something interesting that the patient just said, and suggest nonverbally or verbally that the patient should elaborate. For example, a patient says “My biggest problem is this hospital,” and you reply “This hospital? How?”

## **Silence**

When patients are speaking spontaneously and providing valuable information (and most information from most patients is valuable most of the time), listen attentively without interrupting. Sometimes, even when the patient is not speaking, your *silence* encourages the patient to give important information. In initial interviews, however, silences >15–30 seconds are unhelpful.

## **Empathy**

*Empathy* is a humane sentiment in which you convey by word or action that *you understand what the other person feels*. It can be expressed in infinite ways, including facilitation, silence, comments like “That must have been extremely annoying,” or “You seem very sad about that” or “I can imagine how that comment affected you.” Validation is also empathic.

## **Validation**

In *validation*, you comment on the reasonableness and understandability of the patient’s feelings. An example: “Most people in your situation would be furious.”

## **Self-Revelation**

One form of validation is self-revelation, where you reveal to the patient that you had a common experience and response. An example: “I remember waiting in line at that airport, and I almost blew a fuse.” However, as discussed below, use self-revelation cautiously.

## **Recapitulation (Reiteration)**

To confirm that you perceive accurately what the patient has said, and to convey that you are trying to understand, you may summarize (*recapitulate*) a portion of the patient’s history. An example: “You say you first became ill when you were 48, and you’ve been hospitalized twice since then. Am I correct?”

## **Confrontation**

*Confrontation* is commenting to patients about their behaviors about which they may be unaware. For example, a patient with a nondominant parietal lobe stroke has trouble putting on his hospital robe. He says “It’s a tricky robe. It isn’t sized

properly.” The doctor then asks “Are you sure the problem isn’t you rather than the robe?” and the patient replies “Maybe it is me, because the robe fit just fine this morning.” Another example: Asked “How are you doing today?” a patient replies “just fine.” The doctor replies “But just a moment ago, you were wincing in pain.”

### ***Challenging Self-Deprecatory Misconceptions***

Patients sometimes make *self-deprecatory comments*, and it is useful to challenge these. An example: Patient: “The way he treated me made me feel like a jerk.” Doctor: “Why should you take his insensitivity personally?” Self-esteem-enhancing comments typify *cognitive psychotherapy*.

### ***Education (Information-Giving)***

You must *educate (teach, inform)* the patient about your diagnosis (i.e., the patient’s illness) and assessment and treatment plans. In the course of treating patients, you also inform them about other pragmatic matters (e.g. what to expect during a disability evaluation). In providing this information, solicit and answer the patient’s questions. Doctors do not educate their patients enough. In one study, for every 20 minutes of a medical visit, only one minute was spent in patient education.

### ***Humor***

If you have a good sense of humor, use it, as long as the humor is not derogatory. *Laughter increases endorphin production*.

### ***Positive Reinforcement***

Although *praise* is judgmental, it helps to praise or congratulate patients periodically for efforts they make at improving their health, overcoming obstacles, or reaching milestones. Praise is a type of *positive reinforcement*.

### ***Concluding the Examination with an Informative Summary and a Hopeful Attitude***

As you bring the examination to a close, summarize your findings and recommendations. Summarize (1) further investigations and their rationale, precautions and risks, (2) treatments and their rationale, precautions and risks, and (3) specific plans for followup—when, where and with whom. During this summary, encourage the patient to ask questions and voice concerns. If the interview was for educational purposes, as in an oral examination or a videotaped interview, thank the patient energetically for his or her assistance. Then, *conclude on a friendly, hopeful note*, even if the patient is seriously ill.



## **NO-NOS**

Some doctor behaviors reduce rapport and compliance. These include the following:

### ***Being “Judgmental”***

Convey by your demeanor and choice of words that if a patient says something about which he or she is ashamed, you will not respond demeaningly. For example, instead of asking a patient “Are you a drug addict?” or “Do you abuse drugs,” ask “Did you ever do drugs?” or “Did you ever take street drugs.” Instead of “Did you ever beat up your wife?” ask “Did you ever physically fight with your wife?” Instead of “Did you ever work as a pimp?” ask “Did you ever need money so much that you had someone work for you as a prostitute?” Instead of “What’s wrong?” ask “What’s the problem?”

Sometimes, the word “Why?” is judgmental. For example, if a patient says “I told off my husband yesterday,” let her continue or ask “What were the circumstances?” or “Then what happened?” Don’t ask “Why did you do that?”

### ***Interrupting***

People hate being interrupted. If you must interrupt, wait for a pause.

### ***Arriving Late Without Prior Notification and Without Apologizing***

Although I’m unfamiliar with the research on the subject, I bet the most common cause of patient anger is the doctor coming late without prior notification and an apology.

### ***Giving Unqualified Reassurance***

Avoid giving *unqualified reassurance*, like saying “Everything will be just fine.” You can’t make such a guarantee. Reassurance based on solid data, stated at the conclusion of a thorough examination, is often helpful and necessary. Example: “There’s an excellent chance that you’ll recover from this depression.”

### ***Addressing an Adult Patient by a First Name***

Medical tradition is to *address adult patients by a title* (Mr. Brown, Ms. Green, Dr. Gray, Sgt. Gold), not a first name. If you call the patient by his or her first name, and expect to be addressed as “Doctor,” no fair. If a patient asks to be called by his or her first name, and you want to comply, ask the patient to call you by your first name.

## **OTHER ASPECTS OF INTERVIEWING**

### ***Patient Expressions of Emotions***

Many patients *cry or express anger*. Usually when you allow the patient to express one of these emotions, and don't change the subject because of your own anxiety, the interview is enhanced. However, if the crying or anger intensify and threaten to disrupt the interview, you must try to modify this emotion.

Patients may *laugh or joke*. If a patient says something funny because it is insightful, or tells a genuinely good joke (manic patients sometimes have an infectious sense of humor), smile or laugh. But if the joking is cruel or self-deprecatory, don't laugh.

### ***Personal Questions***

Patients often ask *personal questions*, like "Do you have experience in treating my kind of problem?" or "Are you married?" or "Are you a veteran?" If you feel a question is reasonable and appropriate, answer it briefly and truthfully. Exceptions: 1) Likely, you have a few *secrets* you don't want to reveal. You don't have to reveal them. But decide in advance what questions you won't answer, and what you'll say if asked. 2) Don't let a patient *turn the interview around* by asking a series of questions. Of course, when you summarize your findings and recommendations, encourage the patient to ask questions.

### ***When You Have Bad News***

If you are caring for a patient with a life threatening, incurable, chronic or stigmatized illness, you have to inform the patient about the illness and prognosis. Be truthful without being cruel. If a patient with cancer asks "Do I have cancer?" say yes, and then discuss the cancer, its treatment and prognosis. Usually, this can be done with a hopeful tone, stressing what can be done to cure the illness or maximize the quality of the patient's life.

### ***Your Preoccupation with Something Else***

Occasionally, your attention will lapse momentarily if you are preoccupied with something else. Patients may notice and comment about this. One response: "You're right. I was preoccupied. It has nothing to do with you. I'm sorry, and you have my full attention now."

### ***Disrespectful Behaviors by a Patient or Supervisor***

You need not accept personal abuse from anybody, including patients or supervisors. One response: "I know that you're (busy . . . ill . . . doing your best to help . . .), but I must ask that you treat me with respect. If you do this, I'll do a better job on your behalf."

## CHAPTER 14. PSYCHOLOGICAL TESTING

*Psychological tests* should be used like other laboratory assessments, to confirm or refine clinical diagnoses. Except for brief screening tests like the Mini-Mental State examination of Folstein, psychological tests should be administered by a doctoral level psychologist.

The American Psychological Association endorses only those instruments with proven standardization, reliability and validity. *Standardization* means the instrument is administered and scored the same way each time to all patients. *Reliability* (actually, *test-retest reliability*) means that two administrations of the same test to the same person yield the same results. *Validity* means the instrument measures what it is supposed to measure; for example, a valid aphasia battery must have high sensitivity and specificity in identifying aphasias. *Sensitivity* is a test's *true positive rate*, the proportion of persons with a specific type of aphasia who have positive results. *Specificity* is a test's *true negative rate*, the proportion of persons who do not have the specific aphasia who have negative test results.

### INTELLIGENCE (IQ) TESTS

#### *Definition of General Intelligence*

General intelligence is the ability to solve problems by reasoning and to process information rapidly. General intelligence is correlated with, but not identical to, specific cognitive functions like reading or motor skills. For example, a person with a high IQ could be a below-average reader.

#### *Validity of IQ Tests*

A person's IQ tends to be stable over time. Thus, IQ tests have 1) test-retest reliability and 2) *predictive validity*, meaning that present results correlate highly with results obtained years later. However, a person's IQ can be influenced by illness, motivation and testing conditions. For example, if a piano falls on your head, your IQ will drop.

#### *Uses of IQ Tests*

IQ testing is effective in *predicting school performance*. IQ tests also suggest patterns of brain disease, although they were not designed to do so. Most IQ tests contain two main sections, 1) *verbal IQ*, which assesses dominant hemisphere functioning primarily, and *performance IQ*, which assesses the nondominant hemisphere. When a person's verbal IQ is much lower than his or her performance IQ, this suggests dominant hemisphere disease. When performance IQ is much lower than verbal IQ, this suggests nondominant hemisphere pathology.

Although IQ tests are culturally influenced, they correlate well with results of culture-free assessments. Intelligence and *creativity* are related but not identical.

For example, a person with an average IQ could be uniquely creative in one sphere (e.g., music, painting).

### ***Interpreting IQ Scores***

Fifty per cent of the population have IQs between 90–109. 70–89 is a *border-line IQ*. Scores below 70, plus low scores on social maturity assessments such as the *Vineland Social Maturity Scale*, define mental retardation (MR). Mild MR is more common than moderate MR, which is more common than severe MR. When a specific ability (e.g., reading) is much poorer than the person's general IQ, we diagnose *specific learning disability* such as *developmental reading disorder*.

### ***Selecting IQ Tests for Each Age Group***

Certain IQ tests are preferable for certain age groups.

- a) Birth–age 2: developmental scales like the *Denver Developmental Scale*.
- b) Ages 2–4: *Stanford-Binet*. Actually, the Stanford-Binet is good for ages 2–18, but over age 4, Wechsler instruments are preferred.
- c) 4–6 ½: *Wechsler Preschool and Primary Scale of Intelligence (WPPSI)*.
- d) 6 ½–16: *Wechsler Intelligence Scale for Children-Revised (WISC-R)*
- e) 17 throughout adulthood: *Wechsler Adult Intelligence Scale (WAIS)*

## **NEUROPSYCHOLOGICAL TESTS**

Neuropsychological tests assess brain functioning and brain-behavior relationships. They are used to identify, characterize and localize brain diseases and dysfunctions. They consist of thorough test batteries like the *Halstead-Reitan* and the *Luria-Nebraska* batteries, and tests for specific cognitive functions.

### ***Tests for Specific Cognitive Functions***

- a) *Aphasia*: The most widely used instrument for characterizing aphasia is the *Boston Aphasia Battery*.
- b) *The frontal lobe's ability to shift sets* (move from one strategy to another) is assessed by 1) *Trail Making A and B* tests and 2) *The Wisconsin Card Sorting* test.
- c) *Memory functions* are assessed with the *Wechsler Memory Scale*.
- d) *Nondominant parietal function, dominant hemisphere attention to details, and visual memory* are assessed with the *Rey-Osterreith figure* (Figure 1). The patient is asked 1) to copy the drawing and 2) to reproduce it from memory immediately afterwards and 20 minutes later.

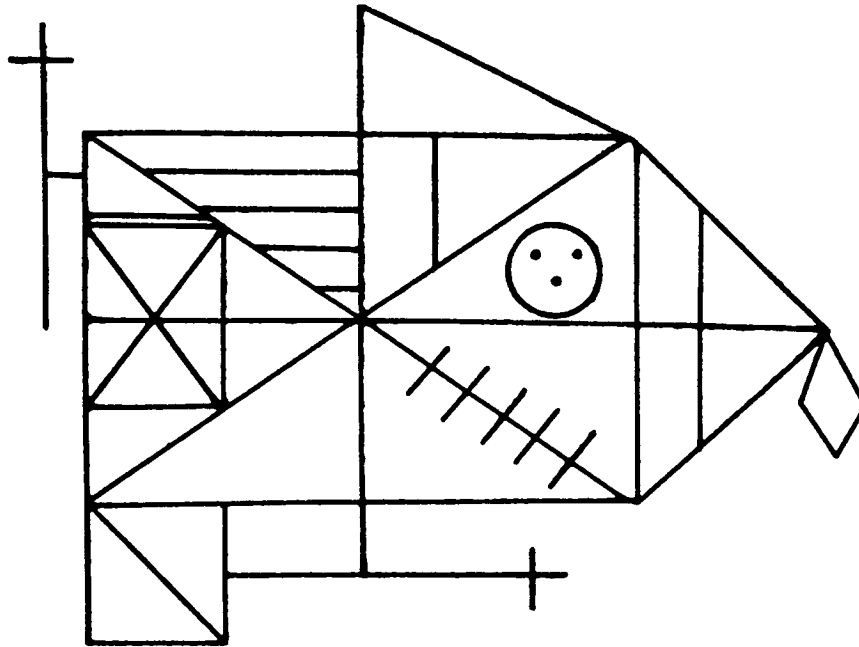


Figure 1. Rey-Osterreith Figure

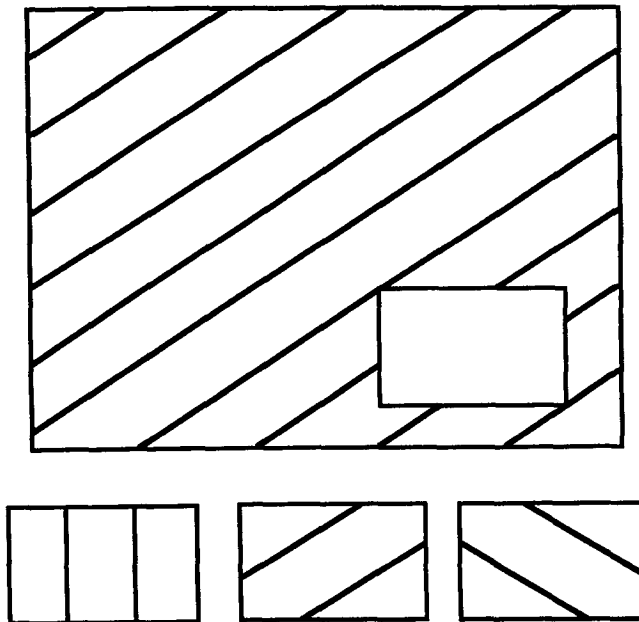
- e) *Visual pattern analysis and recognition* are assessed with *Raven's Matrices*. The patient is shown drawings containing an empty rectangle, and asked to select the design that best fills the rectangle (Figure 2).
- f) *Constructional praxis*, the ability to copy the outline of a simple geometric shape, is assessed using the *Bender Visual Motor Gestalt* test (the *Bender-Gestalt*). The patient tries to copy drawings like the one in Figure 3.

## PERSONALITY INVENTORIES

A person's personality is the extent to which he or she shows long-standing, essentially lifelong, traits of temperament and character. Persons who are assertive at 25 don't become meek at 50, although the intensity of their assertiveness may become tempered over the decades. Personality inventories can be grouped as 1) objective personality inventories and 2) projective tests.

### *Objective Inventories*

The *objective inventories* include the Temperament and Character Inventory of Cloninger, the Eysenck Inventory and the Maudsley Inventory, the Minnesota



**Figure 2.** Figure resembling a Raven's Matrix

Multiphasic Personality Inventory-Second Edition, and the Friedman-Rosenman Type A/B Personality Scale.

- a) *The Temperament and Character Inventory (TCI)* is a 240-question-true-false paper and pencil test that assesses the extent to which a person shows the heritable traits of *novelty-seeking* (excitement-seeking, risk-taking), harm avoidance (caution, worry), *reward dependence* (sentimentality, eagerness to please), *cooperativeness* (getting along with others) and *self-directedness* (taking responsibility for one's actions).
- b) The *Eysenck Personality Inventory* and the *Maudsley Personality Inventory* assess the extent to which a person shows 1) extroversion, 2) "neuroticism" and 3) "psychoticism."
- c) *The Minnesota Multiphasic Personality Inventory-2nd Edition (MMPI-2)* is the most widely used and studied personality inventory. It helps in ascertaining if a person is exaggerating good or bad mental health (lie scale and

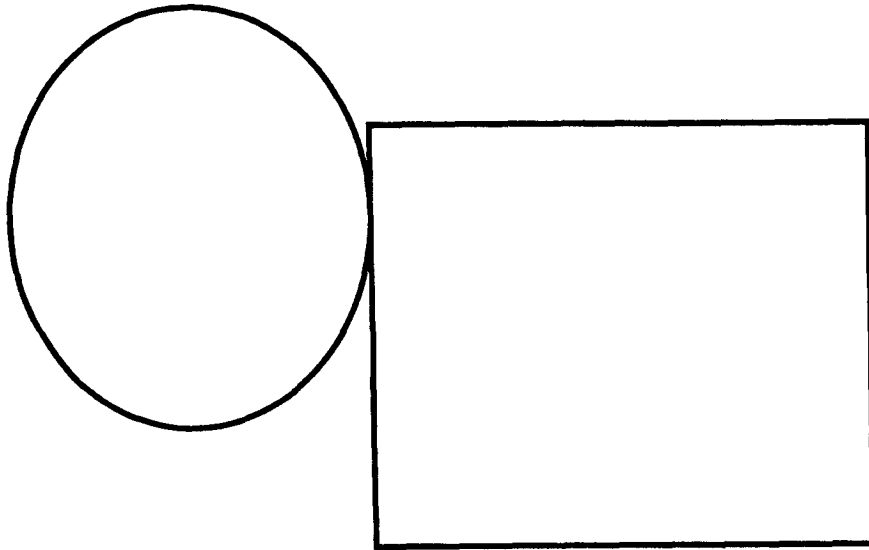


Figure 3. Figure resembling a figure from the Bender-Gestalt

fake scale). However, it doesn't help in research on normal personality, since it assesses for pathologic traits (e.g., schizophrenia scale, depression scale), and it doesn't help in psychiatric diagnosis.

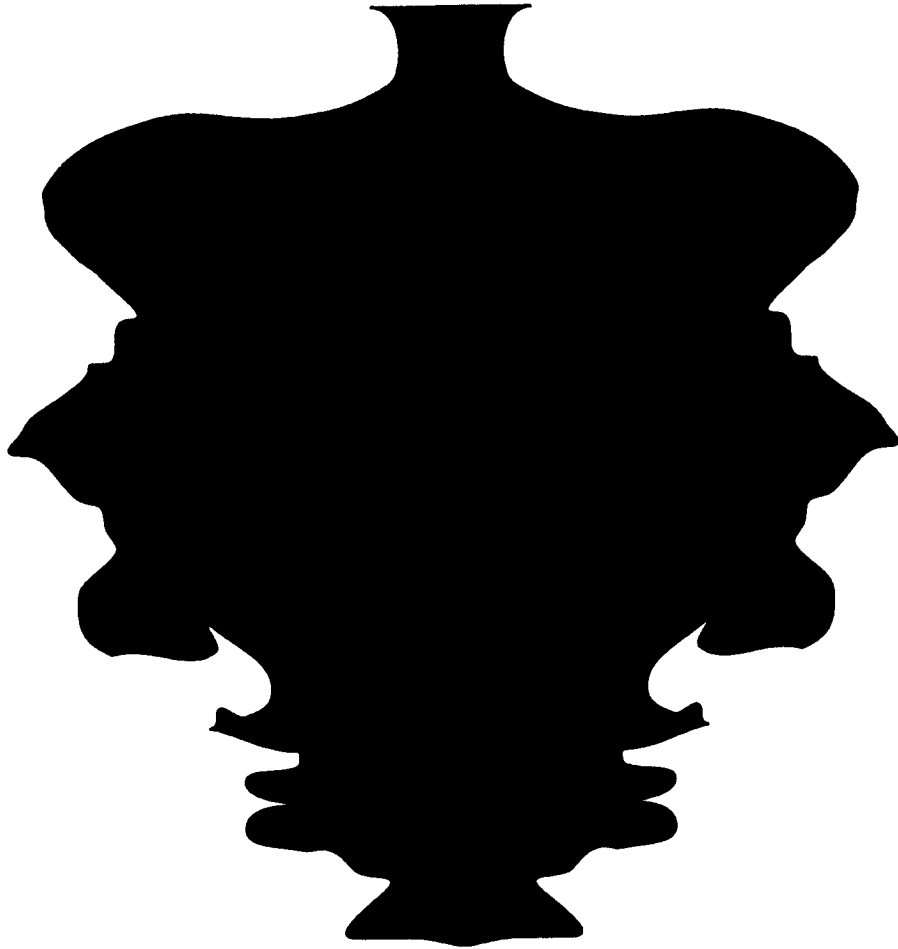
- d) *The Friedman-Rosenman Type A/B Personality Scale* assesses the extent to which a person shows type A or type B traits. *Type A persons* are hard-driving, aggressive, numbers-oriented and achievement-oriented ("I did five successful transplants this week. This year, I'll publish eight papers and make \$300,000. Of course, that's only if I survive my next heart attack."). *Type B persons* are laid back.

Actually, these are inaccurate stereotypes. It was once thought that Type A was a risk factor for coronary artery disease. Not so. All we know for sure is that persons with pre-existing coronary artery disease have a worse prognosis if they score high on the aggression subscale of the Type A scale. So get back to the OR!

### ***Projective Tests***

In *projective tests*, the subject is presented with an ambiguous stimulus and asked to describe what he or she sees. These tests are based on the *projective hypothesis*; that is, what we see in an ambiguous stimulus reflects our personality traits and unconscious wishes. The best known projective tests are the

*Rorschach*, where the stimuli are a series of standardized inkblots (Figure 4), and the *Thematic Apperception Test (TAT)*, where the stimuli are standardized drawings. The Rorschach and the TAT are adjuncts in psychodynamic psychotherapy.



**Figure 4.** Figure resembling Rorschach Inkblot



## CHAPTER 15. ELECTROENCEPHALOGRAPHY AND EPILEPSY

The *electroencephalogram (EEG)* was invented by Hans Berger, a psychiatrist. Techniques to enhance the likelihood of obtaining seizure or other abnormal brain activity on the EEG include 1) sleep induction by hypnotic agents and sleep deprivation; 2) hyperventilation and 3) photic stimulation. Wave types, moving from slowest to fastest, are delta, theta, alpha and beta. *Delta and theta* are seen in 1) in foci of brain pathology; 2) delirium, where these slow waves are seen in all EEG leads; and 3) sleep, where theta occurs in stage 1 sleep and delta occurs in stages 3 and 4. *Alpha* occurs with relaxation with eyes closed in awake persons. *Beta* occurs during attentiveness with eyes open, and with certain medications.

*Spiking, spike and domes and sometimes sharp waves* are seen during seizure activity. One exception is 6 and 14 per second positive spiking which can be normal or seen in attention deficit hyperactivity disorder. *Three per second spike and domes* are seen in petit mal epilepsy and were reported in one patient with digitalis toxicity. *Sleep spindles and K-complexes* occur in stage 2 sleep. A *flat EEG* occurs in brain death.

*Evoked potentials* are recordings of electrical responses of the brain to stimuli such as sounds (*auditory evoked potentials*) or visual images (*visual evoked potentials*). Evoked potentials are especially helpful in diagnosing *multiple sclerosis*.

*Brain atlas studies* are EEG tracings that yield computer-generated cross sectional images of the brain that can localize areas of brain dysfunction.

### EPILEPSY

*Epilepsy* is a condition in which a patient has *recurrent seizures*. A person who has several grand mal seizures during an episode of meningitis (or high fever or hypoglycemia or other illness affecting the brain) is not epileptic, unless the illness causes residual brain pathology that continues to generate seizures. A *seizure* is a transient (usually several seconds to 15 minutes) episode characterized by impaired consciousness, involuntary movements, or sensory abnormalities. Seizure types include grand mal, simple partial, complex partial, and petit mal.

#### *Grand Mal*

In *grand mal epilepsy*, the patient loses consciousness and posture (e.g., may fall to the floor while standing or sitting), first with a generalized increase in muscle tone (*tonic phase*) and then generalized rhythmic flexion and extension of mus-

cle groups (*clonic phase*), then awakening with reduced alertness and cognitive dysfunction (*postictal confusion*). During the seizure, tongue-biting and incontinence of urine and feces are common, and the EEG shows spikes in all leads. The best anticonvulsants for grand mal are 1) *phenytoin* (Dilantin), 2) *carbamazepine* (Tegretol), and 3) *valproic acid* (Depakene, Depakote).

### ***Simple Partial Seizures***

*Simple partial seizures* are brief (1–15 minute) episodes of involuntary motor activity (e.g., rhythmic contractions of one upper extremity) or sensory experiences (e.g., olfactory hallucinations) while the patient is fully conscious. Usually there is EEG spiking or sharp waves in the irritable (epileptogenic) brain area. *Jacksonian seizures* are unilateral simple partial motor seizures that begin with a tonic contraction of the fingers of one hand, the face on one side, or one foot, and progress to clonic (jerky, rhythmic) contractions of the affected area or the entire half of the body. Sometimes simple partial seizures progress to complex partial or grand mal seizures. The best anticonvulsants for simple partial seizures are 1) phenytoin, 2) carbamazepine, 3) *lamotrigene* (Lamictal) and 4) *gabapentin* (Neurontin).

### ***Complex Partial Seizures***

*Complex partial seizures* are brief episodes of involuntary motor activity (e.g., staring or scanning, scratching, picking at clothes, screaming, coughing, repeating the same phrase) or abnormal sensory experiences (e.g., hallucinations in any modality, illusions, *deja vu*, *jamais vu* (p. 93), depersonalization, intense moods) while the patient is not fully conscious. During a partial complex seizure, the patient cannot hold a good conversation. There is usually EEG spiking or sharp waves at the epileptogenic site. The most common focus is in the *temporolimbic system*, but frontal or parietal lobe foci can also generate partial complex seizures. Complex partial seizures are the most common type of epilepsy, and the type most closely associated with psychoses or mood disorders between seizures (*interictal*). Complex partial seizures can progress to grand mal seizures. The best anticonvulsants are 1) carbamazepine (if there are behavioral complications), 3) lamotrigene and 4) gabapentin.

### ***Absence (Petit Mal) Seizures***

*Absence seizures*, which begin in childhood, are characterized by episodes of impaired awareness lasting less than several seconds without interruption of normal activity (e.g., the child maintains a conversation or continues to ride his bike) and without engaging in odd behaviors. Frequent episodes impair learning and other performance. The best anticonvulsants are 1) *ethsuccamide* (Zarontin) and 2) valproic acid.

## ***Status Epilepticus***

*Status epilepticus* is when any type of seizure continues indefinitely and, untreated, could be fatal. Status epilepticus requires immediate medical care, which includes protecting the patient from injury, maintaining vital signs, and giving intravenous anticonvulsant medication.

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# SECTION 6. ABNORMAL BEHAVIOR

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## CHAPTER 16. ADULT PSYCHOPATHOLOGY

In medicine, disorders are classified. *Classification* permits us to determine treatment and prognosis and to conduct research.

### SYNDROMES AND DISEASES

#### *Syndromes*

A *syndrome* is a group of symptoms, signs and lab findings which tend to occur together but for which there is no known single etiology (e.g., chromosome 4 abnormality, tubercle bacillus) and for which there are no pathognomonic lab tests (e.g., genetic screening, Ziehl-Nielsen staining). Although *almost all psychiatric disorders are familial* and probably have a genetic causal contribution, *we don't really know the etiology of psychiatric illnesses* and we have *no pathognomonic laboratory tests*, so *psychiatric disorders are syndromes*, not diseases.

#### *Diseases*

Historically, when a *definitive etiology* (e.g., treponema pallidum infection), a *pathognomonic lab test* (dark field microscopy), and an *effective treatment* (e.g., penicillin) are found for a behavioral syndrome, the syndrome is reclassified as a *disease* and transferred to other specialties. For example, at the turn of the 20th century, syphilitic general paresis of the insane was the diagnosis for a third of patients in state psychiatric hospitals.

#### *Societal Beliefs and Classification*

*Societal beliefs affect the definition of illness.* Some examples:

1. In some South American cultures, white skin discolorations of *pinta* are considered attractive, and persons with *pinta* are viewed as especially marriageable and not ill.
2. Until 1980, *homosexuality* was labeled an illness, which it is not.

### DIAGNOSTIC VALIDITY

Because diagnosis determines treatment and predicts prognosis, diagnoses must be *valid* and reflect discrete disorders. *Robins and Guze's 1970 standards for va-*

lidity of a psychiatric diagnosis are that patients with the putative condition share the following:

1. *common clinical features* including *common demographic characteristics* (e.g., more common in males), *age of onset* (e.g., onset of bipolar disorder is usually 15–30), and *signs and symptoms* (e.g., rapid speech, hyperactivity and labile mood in mania).
2. *differentiation from other illnesses*; for example, if most patients with not-yet validated Condition A also have well-validated Condition B, the validity of Condition A is doubtful.
3. tendency of the illness to run in the patient's family (*familial tendency*); almost all psychiatric disorders are familial.
4. *common laboratory findings* (e.g., many patients with major depression with melancholia have elevated plasma cortisol after receiving oral *dexamethasone*).
5. *a common prognosis*: followup years later should reveal that patients with a well-validated condition either have the same condition or are cured.

## DIAGNOSTIC RELIABILITY

To be valid, diagnoses must be *reliable*. Diagnostic reliability is the extent to which examiners agree on diagnosis (*inter-rater reliability*) measured by the *weighted kappa* statistic. The first diagnostic system with good reliability was the 1972 Washington University criteria, which eventually became—in 1980—the first reliable national diagnostic system, the *DSM-III (Diagnostic and Statistical Manual of the American Psychiatric Association, Third Edition)*. The latest revision is the *DSM-IV*, published in 1994.

## PREVALENCE OF MENTAL ILLNESS

*Point prevalence* is the percentage of ill persons in the population at the time of the study. During the *Epidemiologic Catchment Area (ECA) Study*, 20% of the U.S. population (47 million persons) had a diagnosable mental illness at the time of the study.

*Lifetime prevalence of mental illness* is the percentage of persons in the population who have ever had a mental illness. In the ECA study, the U.S. *lifetime prevalence* was 30%.

## THE BIOPSYCHOSOCIAL MODEL AND THE FIVE DSM-IV AXES

Diagnoses are recorded on five axes in the DSM-IV system to respond to the *biopsychosocial model*, the idea that biological, psychological and social factors are intertwined in the etiology, manifestations, treatment and prevention of illness. The five axes are the following:

*Axis I: All psychiatric disorders except for personality disorders and mental retardation.* Examples of Axis I disorders are bipolar I disorder, alcohol dependence, and depressive disorder due to multiple sclerosis.

*Axis II: Personality disorders, or mental retardation, if present.* A personality disorder is a lifelong combination of traits of temperament and character that impair one's ability to get along with others (*impaired cooperativeness*) and to take responsibility for one's actions (*impaired self-directedness*).

*Axis III: General medical conditions, regardless of whether they cause or intensify behavioral problems.* Examples of Axis III disorders are ingrown toenail, myocardial infarction, and multiple sclerosis. In the latter case, Axis I would read depressive disorder due to multiple sclerosis and Axis III would read multiple sclerosis.

*Axis IV: Psychosocial and environmental problems that can contribute to, intensify, or be caused by behavioral problems.* Examples of Axis IV problems are problems with family (e.g., impending divorce), educational problems (e.g., poor school performance), or occupational problems (e.g., unemployment).

*Axis V: Global Assessment of Functioning (GAF)* is a scale with scores ranging from 1–10 (persistent danger of severely hurting self or others) to 91–100 (superior functioning in many activities).

## MENTAL STATUS FINDINGS

The *mental status examination* is the psychiatric portion of the physical examination. Categories of the mental status examination include appearance, motor functions, affectivity, language, delusions, perceptual disturbances, first rank symptoms and cognitive functions.

### **Appearance**

*Disheveled appearance* with ill fitting unfashionable clothing is common in schizophrenia, chronic mood disorder, and dementia. *Endomorphic build* (chubby, high fat to muscle ratio) is common in mood disorder. *Fibrotic scars over veins* is common in intravenous drug abusers.

### **Motor Behavior**

*Hyperactivity* (intruding into others' conversations, greeting everyone in the room) is common in mania. *Psychomotor slowing* (slow speech, slow gait) is common in major depression with melancholic features and in subcortical dementias. *Agitation* is increased frequency of purposeless movements (like hand wringing, pacing, or jumping up and down) in response to an intense mood. It is common in major depression with melancholic features, mania, delirium, winning a sports event, or anxious anticipation of an important activity like a USMLE exam.

*Catatonia* is a motor syndrome which often includes the patient responding to sensory stimuli despite instructions to the contrary. Some examples of catatonic behaviors:

1. *Echopraxia*: Copying actions based on visual sensations despite instructions to the contrary. For example, tell the patient "When I touch my nose, you touch your ear." Then touch your nose. If the patient then touches his nose, that is echopraxia.
2. *Mitgehen*: Letting the examiner move one's extremity despite instructions to the contrary. For example, tell the patient "I will try to raise your arm, but don't let me. When I push up, you push down." Then gently try to elevate the patient's forearm. If the patient lets you elevate this extremity, that is mitgehen.

Catatonia also may include *cataplexy* (motionlessness), *mutism* (speechlessness) and *posturing* (maintaining an abnormal posture, like keeping one's arms elevated over one's head). The most common cause of catatonia is mood disorder. Catatonia also occurs in schizophrenia, drug intoxication, and diseases of the frontal lobes, basal ganglia, thalamus or brainstem.

### ***Affect and Mood***

*Affect* is the capacity to experience and express emotions (*emotional expressivity*) and to be motivated and concerned (*volition*). *Mood* is one's emotional state at a point in time. An analogy: Affect is to mood as climate is to weather. *Reduced emotional expressivity* is common in major depression with melancholia, with nondominant frontal lobe disease, and with schizophrenia. *Reduced volition* (motivation, concern) is associated with schizophrenia and dominant frontal lobe disease. *Intense moods* are associated with panic attacks, mania and major depression.

### ***Language***

*Circumstantiality* (providing excessive details) is common in mania, alcoholism and epilepsy. *Pressured speech* (talking excessively) is common in mania. *Flight of ideas* (moving gradually from topic to topic) is common in mania. *Clang associations*, the linkage of ideas based on sounds (e.g., "I'm a seismologist and a proctologist") is common in mania. *Paraphasic speech* (misuse of words) is common in posterior aphasia and schizophrenia. *Reduced spontaneity* of speech is common in depression, schizophrenia, subcortical dementia and dominant frontal lobe disease.

### ***Delusions***

*Delusions* are fixed false beliefs that are not commonly shared. Fixed means you cannot persuade the patient that he or she is wrong. Commonly shared beliefs are not signs of illness. For example, atheists might think that religious persons are de-

luded, and vice versa, but neither is deluded. Delusions occur in all psychotic disorders, including mania (often delusions are grandiose), schizophrenia (often delusions are persecutory), major depression with psychotic features (often delusions are nihilistic, somatic or guilty), delusional disorder (the delusions are on mundane topics like love and trust), chronic drug induced psychoses, and psychoses due to general medical conditions.

### ***Perceptual Disturbances***

The *perceptual disturbances* are hallucinations, illusions, and synesthesias.

#### **Hallucinations**

*Hallucinations* are perceptions for which there are no obvious stimuli. They are not pathognomonic for any disorder. *Vivid auditory hallucinations* are common in schizophrenia. *Olfactory, gustatory, and haptic (visceral) hallucinations* are common in psychomotor/psychosensory epilepsy. *Visual hallucinations* are common in delirium, cocaine, amphetamine and hallucinogen intoxication, and psychosensory epilepsy. *Kaleidoscopic visual hallucinations* are common in hallucinogen intoxication. *Geometric visual hallucinations* are common in migraine and cocaine intoxication. *Hypnogogic* and *hypnopompic hallucinations* are common in narcolepsy (p. 55).

#### **Synesthesias**

*Synesthesias*, which are double perceptions like smelling a sound, are most common with hallucinogen intoxication.

#### **Illusions**

*Illusions* are misperceptions of real stimuli. Illusions occur in normal persons (you mistake rustling leaves as a person lunging at you) and are the stock in trade of magicians. Illusions are common in delirium, hallucinogen intoxication, psychomotor/psychosensory epilepsy, and intense anxiety.

*Depersonalization* is an illusion in which one feels detached from oneself. *Derealization* is feeling detached from one's surroundings, as if in a dream. Depersonalization and derealization occur with severe anxiety and psychomotor/psychosensory epilepsy. *Deja vu* occurs when one is in an unfamiliar place and feels that the place is familiar. *Jamais vu* occurs when one is in a familiar place and feels that the place is unfamiliar. *Deja vu* and *jamais vu* occur in temporal lobe psychomotor/psychosensory epilepsy.

### ***First Rank Symptoms***

*First rank symptoms* occur in 70% of schizophrenics, but they also occur in mania, major depression with melancholic features, psychomotor/psychosensory epilepsy, and nondominant parietal lobe disease. The first rank symptoms are



complete auditory hallucinations, thought broadcasting, experiences of alienation, experiences of influence and delusional perceptions.

*Complete auditory hallucinations* are crystal clear voices speaking full sentences that seem to come from outside one's head. *Thought broadcasting* is the experience that one's thoughts are being transmitted to others as if from a mechanical device like a radio. *Experiences of alienation* are the feeling that one's thoughts or actions are not one's own, but belong to someone else. *Experiences of influence* are the feeling that while one's thoughts and actions are one's own, they are controlled by someone else. *Delusional perceptions* are correct perceptions (e.g., I see two clouds) followed by delusional conclusions having nothing to do with the perception (e.g., therefore, the FBI is monitoring my phone calls).

## **ANXIETY DISORDERS**

Anxiety is part of the normal fight and flight reaction needed for survival. In moderation, anxiety can enhance performance. Mild to severe anxiety may accompany any general medical or psychiatric disorder. When excessive for the situation in which it occurs, anxiety causes suffering and decreases efficiency.

*Anxiety disorders* are common. Severe forms occur in 4–6% of the population, and 12% of persons have specific phobias. Except for social phobia and obsessive compulsive disorder, which are equally common in females and males, anxiety disorders are more common in females. Except for specific phobia, which usually has a childhood onset, anxiety disorders usually begin between 15 and 30. Onset of an anxiety disorder past 30 raises suspicion of an anxiety disorder *due to a general medical condition such as a cardiopulmonary emergency, a stroke, epilepsy, Parkinsonism, postconcussion syndrome, alcohol or sedative hypnotic withdrawal, caffeine or amphetamine toxicity, or endocrine disorders, especially hypoglycemia, thyrotoxicosis and Cushing's disease*. Except for severe obsessive compulsive (OCD) or posttraumatic stress disorder (PTSD), anxiety disorders rarely require hospitalization.

The anxiety disorders are panic disorder, agoraphobia, social phobia, generalized anxiety disorder (GAD), specific phobia, OCD, acute stress disorder, and PTSD. Panic disorder, agoraphobia, social phobia and GAD may be variants of the same illness, for these reasons: 1) the majority of persons with these disorders have a coexisting anxious personality disorder; 2) many patients have two or more anxiety disorders simultaneously; 3) in families of persons with any of these conditions, there is an increased prevalence of all these conditions; 4) treatments are similar for these conditions; and 5) nonmelancholic depression occurs frequently late in the illness course.

### ***Panic Disorder (PD)***

*Panic disorder* is characterized by spontaneous panic attacks lasting 5–30 minutes, occurring from several times yearly to many times daily, and not linked to specific situations. During a panic attack, several of the following are present:

1) dyspnea; 2) paresthesias from respiratory alkalosis; 3) lightheadedness; 4) chest discomfort; 5) tachycardia with palpitations; 6) slight rise in blood pressure; 6) tremor; 7) sweating; 8) depersonalization; and 9) fear of dying. Given these symptoms, it is not surprising that the average patient with panic disorder has multiple medical visits and laboratory tests before PD is diagnosed.

Among PD patients, there is an increased prevalence of *mitral valve prolapse*. Intravenous administration of *sodium lactate* induces panic attacks in patients with PD (or agoraphobia), but not in other persons. Placebo does not induce panic attacks in PD patients.

Treatment is with antidepressants and behavior therapy, including education about the physiology of the disorder and teaching of diaphragmatic breathing. Benzodiazepines can be used during panic attacks. Continuous use of benzodiazepines beyond 4 months is inevitably associated with physiologic dependence. Properly treated, most PD patients improve.

### ***Agoraphobia***

*Agoraphobia*, which often co-occurs with PD and social phobia, is *fear of a situation in which one could panic, lose self-control (such as by fainting, vomiting, urinating, crying out or dying), and not have prompt access to help*. Each agoraphobic person fears several of the following: 1) leaving home or 2) leaving the neighborhood; 3) being in traffic, 4) in a tunnel, 5) on an elevator, 6) on a bridge or 7) in a hairdresser's chair or 8) a shopping mall.

Occasionally, fear of leaving home leads to becoming *housebound* and requiring initial home visits, but most agoraphobics aren't housebound. The prognosis is like that of PD. Treatment includes antidepressants and behavior therapy, often in vivo exposure.

### ***Social Phobia***

*Social phobia* is the most common anxiety disorder of males. Its defining feature is *anxiety in anticipation of and during social situations in which the patient fears—almost always inaccurately—scrutiny by others who will detect the patient's anxiety and then disparage the patient*. Feared situations include public speaking, interviews, dating, parties, weddings, dances, eating or writing in public, or using public lavatories. The prognosis is good if the patient is treated with cognitive-behavioral therapy—including in vivo exposure—and selective serotonin reuptake inhibitors.

### ***Generalized Anxiety Disorder***

Generalized anxiety disorder involves *persistent unrealistic or excessive worry about life circumstances associated with motor tension, autonomic hyperactivity, and increased vigilance*. It is a mild, but chronic and continuous form of PD. Treatment includes a combination of beta blockers, antidepressants and relaxation.

## ***Specific Phobia***

Of all the phobias, specific phobias are most apt to begin in childhood. Unlike social or agoraphobia, specific phobias are *fears of a single object or situation*. The most common specific phobias are 1) specific types of animals like insects or cats, 2) situations such as heights (acrophobia), flying or storms and 3) blood and injury.

Persons with specific phobias usually seek treatment only if they are likely to be exposed to the feared object or situation. For example, persons living in large midwestern cities who fear snakes are not likely to seek treatment because they are not likely to encounter snakes (at least, the reptilian kind) except at the zoo. Second year medical students with blood and injury phobias usually seek treatment before starting clerkships.

Treatment includes cognitive and behavioral therapy with systematic desensitization or in vivo desensitization.

## ***Acute Stress Disorder***

In *acute stress disorder*, the patient experiences a *traumatic event in which there was actual or threatened death or serious injury or personal violation (e.g., rape)*. During the event, the patient felt fearful or helpless, and during or within four weeks after the event, the patient felt symptoms in each of the following categories:

1. *Numbing or detachment*: feeling "in a daze," derealization or depersonalization, or being unable to remember large portions of the event.
2. *Reexperiencing the event*, as in daydreams, nightmares, obsessions, hallucinations or distress from reminders of the event. Vietnam veterans with these symptoms often label them flashbacks.
3. Symptoms of *hyperarousal*, such as trouble falling or staying asleep, hypervigilance, irritability or an exaggerated startle response.

The condition lasts a maximum of one month.

## ***Posttraumatic Stress Disorder (PTSD)***

As for acute stress disorder, patients with *posttraumatic stress disorder (PTSD)* experience a traumatic event and then have numbing, reexperiencing and hyperarousal. Unlike acute stress disorder, PTSD persists beyond one month. Some cases begin months or years after the event, and are termed *delayed onset PTSD*. Most PTSD patients have coexisting conditions, including panic disorder, major depressive disorder, obsessive compulsive disorder, substance dependence, and antisocial personality disorder. Treatment includes treating the co-morbid condition.

## ***Obsessive Compulsive Disorder (OCD)***

*Obsessive compulsive disorder (OCD)* is characterized by recurrent intrusive thoughts (*obsessions*) perceived by the patient (early in the illness, at least) as il-

logical and which limit the patient's functioning. Common obsessions: 1) daily decisions (Should I take Lake Avenue or Willow Road?); 2) outlandish behaviors (e.g., Could I have recently run over a child with my car?); 3) order and symmetry (is everything placed just right?); 4) dirt and contamination; and 5) religion and philosophy.

In most OCD patients, obsessions are accompanied by repetitive behaviors (*compulsions*) which are usually (not always) driven by obsessions. For example, a woman obsessed with becoming dirty and contaminated may wash her hands many times per hour. One obsessive patient washed her garbage before she removed it from her home. Unfortunately, the compulsive behaviors don't relieve the anxiety of obsessions.

OCD is associated with PET, SPECT and MRI *abnormalities in the basal ganglia and frontal lobes*. Onset of OCD past age 30 should lead you to suspect OCD due to frontal lobe or basal ganglia disease.

Families of persons with OCD have increased risk of OCD and *Gilles de la Tourette's disorder* (tics, grunts, cursing).

Treatment is in vivo exposure and selective serotonin reuptake inhibitors.

## **SOMATOFORM DISORDERS**

Somatoform disorders are conditions that present primarily with general medical symptoms. The somatoform disorders are somatization disorder, factitious disorder, malingering, pain disorder, conversion disorder, hypochondriasis, and body dysmorphic disorder.

### ***Somatization Disorder (SD)***

*Somatization disorder (SD)* is much more common in women, affecting 1–2% of women and 0.4% of the population. In families of persons with SD, there is an increased prevalence of SD in the women and of antisocial personality disorder in the men. The full syndrome is usually diagnosable by age 30, although some symptoms begin in teenage.

It is characterized by multiple medically unexplained symptoms, usually described vaguely or dramatically by the patient, in multiple body systems. To make the diagnosis, the patient must have had four medically unexplained pain symptoms, two medically unexplained gastrointestinal symptoms (e.g., nausea), one medically unexplained sexual symptom (e.g., sexual indifference), and one pseudoneurologic symptom (e.g., blindness). This picture explains why persons with SD are, compared to the general population, more apt to have needless surgery.

Treatment, which requires no medication, includes educating the patient about the disorder, limiting visits to the care of one primary care doctor, and scheduling regular visits to reinforce good health rather than telling the patient to "call if any symptoms arise."

## ***Malingering***

*Malingering* is faking symptoms or signs of illness to avoid an aversive situation like combat, jail or an examination. It is common in persons with antisocial personality disorder or substance abuse. Suspect it if the patient is evasive or intimidating, or if the history is inconsistent. In psychiatry, the most commonly malingered symptoms are hallucinations and suicidal ideation in the absence of observable signs like sad mood or psychomotor slowing.

Unless you observe faking of findings (such as seeing the patient fake fever by heating a thermometer), let the patient save face (patients will not fess up and say "You're right, I'm faking."), but discharge him or her from care.

## ***Factitious Disorder***

*Factitious disorder* is faking symptoms or signs *to enter the sick role* (e.g., to be hospitalized) with no external incentive like avoiding jail. Factitious disorders are more common in persons with Cluster B personality disorders. *Munchausen's syndrome* is a severe example where a patient is hospitalized repeatedly and accepts invasive procedures. *Munchausen by proxy* is a form of child abuse where a parent fabricates illness in a child; this must be reported to the local child and family service agency.

## ***Conversion Disorder***

*Conversion disorder* is characterized by *pseudoneurologic* dysfunction in organs of special sense or the voluntary muscles, such as blindness with a normal neuro-ophthalmologic examination, or hemiparesis without hyperreflexia, clonus, or a Babinski sign. It is common in persons with SD and antisocial personality disorder. Also, many conversion disorder patients have, on further examination, "real" neurologic disease like multiple sclerosis, stroke or epilepsy.

## ***Pain Disorder***

*Pain disorder* is characterized by pain without physical or lab abnormalities, or pain more severe than the findings would suggest. Most persons with pain disorder have a co-existing psychiatric disorder, which should be treated, or a neurologic disorder like reflex sympathetic dystrophy, thoracic outlet syndrome, stroke, multiple sclerosis or thalamic disease.

## ***Hypochondriasis***

*Hypochondriasis* is the fear that one has a serious disease, despite evidence to the contrary. Most persons with hypochondriasis have a co-existing psychiatric illness, most often depression, OCD or SD.

## ***Body Dysmorphic Disorder***

*Body dysmorphic disorder (BDD)* is preoccupation with an imagined defect in appearance (e.g., “my nose is misshapen”), or an exaggeration of a mild anomaly of appearance, that sometimes leads to cosmetic surgery that does not alleviate the preoccupation. BDD may be a variant of OCD.

## **MOOD DISORDERS**

All of us have intense moods. When an intense mood is prolonged and associated with suffering and impaired functioning, we diagnose a mood disorder. The mood disorders are bipolar disorder, major depressive disorder and dysthymic disorder.

### ***Bipolar Disorder***

#### **Definitions**

*Bipolar disorder* is defined by a person having one or more *manic* or *hypomanic* (mildly manic) episodes. Some bipolars never become depressed, although they are predisposed to depression.

Persons with *bipolar I disorder* have had one or more full manic episodes. Persons with *bipolar II disorder* have had one or more hypomanic (but never full manic) episodes and one or more major depressive episodes.

#### **Epidemiology**

Bipolar disorder affects 0.5% of the population, but huge proportions of psychiatrically hospitalized persons have it. Its most common ages of onset are 15–30, but it can begin in childhood or old age. It is more frequent in persons of upper socioeconomic status (SES), and common in writers, artists and musicians. It is strongly familial and highly heritable.

#### **Symptoms and Signs**

Its cardinal signs are *stimulus bound hyperactivity, rapid and pressured speech, and a euphoric, labile or irritable mood*. Also common are grandiosity, racing thoughts, circumstantiality, flight of ideas, clang associations, reduced need for sleep, spending sprees, wearing brightly colored clothing, hypersexuality, singing and dancing, public nudity, delusions (often grandiose), hallucinations and catatonia. Mood disorders, including mania, are the most common cause of *catatonia* (p. 92).

#### **Treatment**

*Lithium* is the treatment of choice of manic episodes and for prophylaxis against recurrence of mania or depression, and is a good antidepressant for depressions in bipolars. Carbamazepine, valproic acid, electroconvulsive therapy (ECT), and neuroleptics are also effective for manic episodes.

## **Prognosis**

About half of bipolars have only one episode, or recurrent episodes with good remissions. The other half, particularly those who have been treated continuously with neuroleptics or have abused substances like alcohol or cocaine, have a chronic course. As time progresses for bipolars, episodes occur more often, are more severe and last longer (*kindling or sensitization hypothesis*). *15% of bipolars eventually commit suicide*; this occurs during depressed or mixed (manic and depressed at the same time) episodes, not during mania.

## **Major Depressive Disorder**

### **Definition**

*Major depressive disorder (MDD)* was formerly termed *unipolar disorder*. Persons with MDD have one or more episodes of major depression, but no manic or hypomanic episodes.

### **Epidemiology**

MDD affects 2–4% of the population, and is more common in women than in men.

### **Signs and Symptoms**

Common features of *major depressive episodes* include a sad or anxious mood, trouble falling asleep (*initial insomnia*) or staying asleep (*terminal insomnia*) or sleeping excessively (*hypersomnia*), reduced or excessive appetite, suicidal ideation or attempts, inability to enjoy pleasurable activities like sex, hobbies or work (*anhedonia*), agitation or psychomotor slowing, and feelings of guilt, self-pity, hopelessness, helplessness or worthlessness.

In *major depressive episodes with melancholic features*, the sad or anxious mood is unresponsive to good news or a sensitively conducted interview (*sustained mood*), *agitation or psychomotor retardation* are common, and terminal insomnia is typical, as is appetite and weight loss, suicidal ideation, anhedonia, and feelings of guilt, hopelessness or worthlessness.

*Major depressive episode with psychotic features* is diagnosed when major depression is accompanied by delusions (most often *delusions of guilt or nihilism or somatic delusions*) or hallucinations (often auditory hallucinations and sometimes hallucinations that command the person to commit suicide). This depression usually shows melancholic features, and is common in depressive episodes in bipolar disorder.

### **Treatment**

Major depression without melancholic features is usually treated with antidepressants and cognitive or interpersonal psychotherapy. Major depression in bipolars is often treated with ECT or lithium. Antidepressants should usually be avoided in bipolars because these can precipitate mania. Severe major depression

with melancholic features, or major depression with psychotic features, is best treated with ECT.

### **Prognosis**

The typical course of MDD is one or several episodes with full recovery. However, MDD is chronic and disabling in 5–10% of cases, and among all persons with MDD, 15% eventually commit suicide.

### ***Dysthymic Disorder***

*Dysthymic disorder* is a chronic, relatively mild type of depression that has lasted at least two years. In contrast to major depression with melancholic features, dysthymic disorder is more apt to show a *mood that is reactive* to a well-conducted interview, *initial insomnia* (trouble falling asleep), and normal motor activity levels. It frequently co-exists with personality disorders, anxiety disorders, substance abuse and eating disorders. It is treated with antidepressants or cognitive or interpersonal psychotherapy.

## **SCHIZOPHRENIA**

*Schizophrenia* is one of the most overdiagnosed conditions in the U.S.

### **Epidemiology**

Using DSM-IV criteria, the population prevalence is 1%. First episodes are equally common in males and females, but chronic hospitalization for schizophrenia is more common for males. It is familial and heritable. Typical age of onset is 15–30. It can begin in childhood, either as schizophrenia or as schizoid or schizotypal personality disorder (p. 104). Onset over 30 suggests another disorder.

### **Signs and Symptoms**

Schizophrenia usually begins insidiously or subacutely with *positive (productive) symptoms*, which include hallucinations (usually auditory), delusions (often persecutory), first rank symptoms, and formal thought disorder. As the illness progresses, positive symptoms are less intense and negative symptoms predominate. *Negative symptoms* include *reduced emotional expressivity*, *paucity of thought and avolition* (reduced motivation, planning and concern).

### **Laboratory Findings**

CT and MRI show increased frequency of mild cortical or cerebellar vermis atrophy. PET and SPECT show increased frequency of frontal lobe hypometabolism and blood flow (*hypofrontality*). None of these is specific for schizophrenia, however.



### **Treatment**

*Neuroleptics* are the treatment of choice. In part because neuroleptics block dopamine, there is a *dopamine hypothesis* that schizophrenia is associated with hyperdopaminergic activity. Patient and family education and support are helpful. Repetitively critical or guilty comments (*excessive expressed emotionality*) by family members are associated with exacerbation of the illness.

### **Prognosis**

Using DSM-IV criteria, prognosis is variable, but schizophrenia is usually chronic and disabling. Schizophrenics rarely marry, maintain a marriage or friendships, or hold a job.

## **DELUSIONAL DISORDER**

### **Epidemiology**

This rare condition affects 0.3% of the population. Typical age of onset is 30–50. At illness onset, the patient is typically married and employed. There is an increased family prevalence of paranoid personality disorder.

### **Signs and Symptoms**

The classic finding is a *well-organized delusion on a mundane subject like love, trust or work*. The delusion is expressed with such clarity that it seems almost believable. The mood responds to the content of the delusion. The only other symptom is hallucinations which, if present, are on the same subject as the delusion; for example, the patient hears voices of his fellow employees conspiring against him.

### **Treatment**

There are no systematic controlled treatment trials. Neuroleptics, including pimozide, are often used.

## **EATING DISORDERS**

The eating disorders are anorexia nervosa and bulimia.

### ***Anorexia Nervosa***

#### **Epidemiology**

Ninety per cent of persons with anorexia nervosa are females between 12 and 30. It is familial and heritable, and there is increased family prevalence of mood disorder. It is more common persons of middle and upper SES.

#### **Symptoms and Signs**

The essential feature is that the patient believes that she is overweight and must diet even after she has lost huge amounts of weight and is gaunt. Despite the term anorexia, appetite is normal and patients often enjoy cooking and often binge-eat

and induce vomiting or take laxatives or diuretics to compensate (*bulimia*). Despite the weight loss, energy is normal. *Amenorrhea* is typical and usually precedes weight loss. Nonmelancholic depression and OCD commonly co-occur.

Other findings include narrowing of shoulders and hips, bradycardia, reduced temperature, dry skin, loss of scalp hair with retained pubic and axillary hair. *Lanugo hairs* (blond, short, downy hairs) grow on cheeks, neck, forearms and thighs. Dissolution of enamel and dental caries occur because of repeated vomiting and gastric acidity. Repeated emesis with hypochloremic alkalosis produces parotid gland enlargement.

### **Treatment**

The treatment is antidepressants and behavior modification.

### **Prognosis**

The condition is fatal in 9% of cases. Full recovery occurs in half of cases.

## ***Bulimia Nervosa***

### **Epidemiology**

As for anorexia nervosa, *bulimia nervosa* primarily affects females 12–30 and it is more prevalent in persons of middle and upper SES. Family members and patients are more likely than the general population to have mood disorders and substance abuse.

### **Symptoms and Signs**

The condition is characterized by binge-eating followed by purging. With frequent vomiting, dental problems and parotid enlargement may occur. Weight loss is usually minimal. *Amenorrhea* is less common than in anorexia nervosa.

### **Treatment**

Treatment includes antidepressants and cognitive and behavioral psychotherapy.

## **PERSONALITY DISORDERS**

In the DSM-IV, personality disorders are diagnosed *categorically*, as if they are discrete illnesses. Unfortunately, most persons meeting criteria for one personality disorder have at least one more personality disorder, yet people have only one personality! In contrast, in a *dimensional system*, diagnosis is based upon the degree (high, average, low) to which a person shows traits like *harm avoidance*, *reward dependence* and *novelty seeking*.

Personality disorders are divided into three clusters. *Cluster A (odd, eccentric)* includes paranoid, schizoid and schizotypal personality disorders. *Cluster B (dramatic, emotional)* includes antisocial, narcissistic, histrionic and borderline personality disorders. *Cluster C (anxious)* includes dependent, avoidant, obsessive compulsive and passive aggressive personality disorders.

## **Cluster A**

### ***Paranoid Personality Disorder***

1. mistrustful and suspicious
2. increased in families of persons with delusional disorder

### ***Schizoid Personality Disorder***

1. withdrawn, a loner
2. increased in families of schizophrenics
3. may be a mild variant of schizophrenia

### ***Schizotypal Personality Disorder***

1. odd ideas (e.g., satanism, magic)
2. mild emotional blunting
3. stilted (manneristic) speech
4. increased in families of schizophrenics
5. may be a mild variant of schizophrenia

## **Cluster B**

### ***Antisocial Personality Disorder***

1. callous, lacks remorse, high novelty seeking, low harm avoidance, low reward dependence
2. fighting, arrests, trouble keeping jobs, lying, using aliases, substance abuse

### ***Borderline Personality Disorder***

1. mood swings, suicidal gestures, high novelty seeking *and* harm avoidance
2. unstable relationships with splitting (p. 33)
3. may be a variant of mood disorder

### ***Histrionic Personality Disorder***

1. theatrical, seductive
2. common premorbid (pre-illness) personality for somatization disorder

### ***Narcissistic Personality Disorder***

1. attitude of self-importance
2. attitude of entitlement
3. requires constant attention
4. hypersensitive to criticism
5. selfish, unempathic

## **Cluster C**

### ***Dependent Personality Disorder***

relies excessively on others for decisions and advice

### ***Avoidant Personality Disorder***

avoids social situations that could be embarrassing

### ***Obsessive Compulsive Personality Disorder***

rigid, meticulous, controlling, perfectionistic

### ***Passive Aggressive Personality Disorder***

expresses anger indirectly by procrastinating, forgetting, coming late, screwing up tasks

## CHAPTER 17. CHILD PSYCHOPATHOLOGY

Child psychiatric disorders consist of four categories: 1) maturational delays, 2) childhood onset of typically adult-onset disorders, 3) pervasive developmental disorders and 4) specific learning disabilities.

*Maturational or developmental delays* are disorders more common in children than adults and for which prevalence decreases with age. These are attention deficit hyperactivity disorder, conduct disorder, elimination disorders, and parasomnias. Although these conditions tend to remit spontaneously as the child becomes older, most children require treatment and respond well (exception: conduct disorder).

Almost all *adult onset disorders* (e.g., mood disorders, anxiety disorders, schizophrenia, secondary behavioral disorders) have a childhood variant. Compared to adult onset of these disorders, childhood onset is associated with a stronger family history and a poorer prognosis, especially if not well treated. Diagnostic criteria and treatments for these conditions are essentially the same as for adults.

*Pervasive developmental disorders* include mental retardation (MR), autism and Rett's disorder (p. 68). MR and autism are lifelong.

*Specific learning disabilities* are diagnosed when a specific ability (e.g., reading, drawing) is impaired despite good or excellent performance of other skills.

### MATURATIONAL DELAYS

#### *Attention Deficit Hyperactivity Disorder (ADHD)*

**Epidemiology:** ADHD is very common, affecting 10% of boys, 2% of girls, and the majority of child psychiatric inpatients. It is the most common reason for referral to child psychiatrists. Like most childhood disorders (exceptions: mood disorders, eating disorders, Rett's disorder), it is more common in boys. It is familial. In families of children with ADHD, there is an increased prevalence of ADHD, conduct disorder, antisocial personality disorder, mood disorders, anxiety disorders and substance abuse.

**Clinical Manifestations:** *Its essential characteristics are an inability to sit quietly, pay attention and complete tasks.* Manifestations include squirming in one's chair, answering out of turn, overresponding to stimuli, and not completing homework or other tasks.

Children with ADHD often have co-existing conditions, including mood disorders, obsessive compulsive disorder, Gilles de la Tourette syndrome, conduct disorder and learning disorders.

**Treatment:** Dozens of double blind controlled studies show the efficacy of stimulants (methylphenidate, dextroamphetamine, pemoline).

**Prognosis:** Prognosis for ADHD is good, so the child's outcome depends largely on what, if any, disorder co-occurs. For example, if the co-existing disorder is conduct disorder, the child's ADHD will respond well to stimulants. But,

because 37% of children with conduct disorder develop adult sociopathy, 37% of children with ADHD plus conduct disorder will develop adult sociopathy.

### ***Conduct Disorder***

***Epidemiology:*** Conduct disorder is the current term for what was once termed juvenile delinquency. It accounts for the majority of children in youth detention centers and jails. In families of children with conduct disorder, there is an increased prevalence of conduct disorder, antisocial personality disorder, criminality and substance abuse.

***Manifestations:*** The child with conduct disorder is callous, insensitive to others, and remorseless. Associated behaviors include everything your parents told you not to do: bullying, fighting, assaulting, being sexually promiscuous, raping, ditching school, running away from home, stealing and being arrested.

***Treatment:*** No treatment works. "Treatment" is based on common decency, not science. Parents and teachers should supervise the child, reward good behavior, set limits, encourage development of talents, and treat co-existing treatable conditions like ADHD.

***Prognosis:*** 37% of children develop adulthood antisocial personality disorder. 63% eventually lead reasonable lives, working and supporting families.

### ***Elimination Disorders: Enuresis and Encopresis***

***Definitions:*** *Enuresis* is frequent urination into clothes and bed past age 5. *Encopresis* is fecal incontinence several times monthly past age 4.

***Epidemiology:*** Both are more common in boys. Both are familial, enuresis dramatically so. If both of one's parents are enuretic, there is a 77% chance that one is or will be enuretic. If one parent is enuretic, the chance is 44%.

***Etiology:*** Both can be due to general medical conditions or "idiopathic." General medical causes of enuresis include a low bladder threshold for emptying, urinary tract infection, spinal cord disease and excessive caffeine intake. Causes of encopresis include hypothyroidism, hypercalcemia, anal fissure, rectal stenosis, lactase deficiency, overeating fatty foods and Hirschsprung's disease.

***Pathophysiology:*** Most "idiopathic" enuresis is due to *reduced nighttime vasopressin production*. Regardless of whether the etiology is secondary or idiopathic, the pathophysiology of encopresis is the same: 1) Constipation with fecal impaction, leading to 2) a distended thin walled bowel with reduced sensory neuron function, reduced sense of rectal fullness and reduced recognition of fecal overflow, 3) habituation to fecal odors to the point of inattention, and 4) being ridiculed.

***Treatment:*** Treat treatable causes of the elimination disorder (e.g., antibiotics for urinary tract infection, thyroxine for hypothyroidism). For idiopathic enuresis, *desmopressin nasal spray* or *imipramine* is combined with behavior modification in which 1) fluids are restricted towards bedtime and 2) the child learns to pair being awake with having a full bladder and urinating. This is accomplished by

1) having the child sleep with a moisture sensitive blanket which triggers a buzzer when urination begins, waking the child, who then finishes urinating in the toilet, or 2) a parent awakening the child to urinate several times nightly.

## **CHILDHOOD ONSET OF DISORDERS WHICH TYPICALLY BEGIN IN ADULTHOOD**

### ***Mood Disorders***

Major depression with and without melancholic features, bipolar disorder, and dysthymic disorder can have childhood onset. Clinical manifestations are the same as for adults, except that the child's chief complaint is less likely to be "I can't concentrate, sleep or enjoy myself" and more apt to be a parent's reporting of a child's behavior such as inattention to schoolwork.

### **Teenage Suicide**

All depressed children and teenagers should be asked about thoughts of suicide, the third leading cause of death in teenage. Epidemiology of teenage suicide is essentially the same as for adult suicide: more common in Native and Euro-Americans than African Americans, in boys than girls, and in Protestants than Catholics. As for adults, gunshots are the most common method for both genders. Also as for adults, the clinical situation (e.g., the child is depressed and has suicidal ideation) is more important than the epidemiology (e.g., the child is Euro-American).

### ***Anxiety Disorders***

Specific phobia, panic disorder, agoraphobia, social phobia and obsessive compulsive disorder can begin in childhood. Separation anxiety disorder has a childhood onset. Interestingly, 20% of adult agoraphobics retrospectively report notable childhood separation anxiety, so separation anxiety disorder might be a precursor of adult agoraphobia or social phobia.

### **Fears and Specific Phobias**

Whether due to inexperience or innate wisdom, all young children fear things like witches, ghosts, dogs, bears, darkness, kidnapers, strangers, separation and things that go bump in the night. When a fear becomes so intense that the child's functioning is impaired, we diagnose a specific phobia. Specific phobias are discussed in chapter 16.

### **Separation Anxiety Disorder**

*Definition:* Between six months and three years, crying and anxiety are normal when parents leave. When severe separation anxiety persists beyond three years and impairs functioning, separation anxiety disorder is diagnosed.

*Epidemiology:* The average age of onset of separation anxiety disorder is nine.

*Manifestations:* It is characterized by fear of being away from one's parents and other familiar adults. Fears include being alone, going to sleep and going to school. 75% of children with the disorder are anxious about going to school and have somatic complaints on school mornings, or refuse to go to school. Not all school refusal, however, is separation anxiety.

*Treatment:* The treatment is in vivo exposure, which includes requiring the child to go to school. Some schools permit parents to bring the child in pajamas. In others, a principal or advisor might drive the child to school.

### **Obsessive Compulsive Disorder (OCD)**

The symptoms and behaviors resemble those of adult OCD, except that 1) although the child usually knows that the obsessions are unrealistic, he or she usually insists (unlike adults) that the compulsions are necessary and 2) the child involves adults in his or her compulsions (e.g., "Dad, please wash my clothes again.") Most adults with OCD know that others dislike participating in the compulsions.

Childhood onset OCD sometimes accompanies Gilles de la Tourette's syndrome. Sometimes, obsessions and compulsions are the first presentation of a childhood mood disorder. Treatment of OCD is exposure therapy and response prevention. If the child is not cured, a selective serotonin reuptake inhibitor is prescribed.

## CHAPTER 18. VIOLENCE: SUICIDE, HOMICIDE AND ACCIDENTS

Violence—suicide, homicide, accidents—is the leading cause of death of Americans under age 40.

### SUICIDE

Suicide is the *ninth leading cause of death* in the US: yearly, 25,000–30,000 Americans kill themselves. Annually, 250,000–300,000 Americans *attempt suicide*. Suicide is the second most preventable cause of death in the US—lung cancer is first—for these reasons: 1) MDs have access to suicidal persons, who often visit MDs before killing themselves; 60% of persons who kill themselves visit an MD during the month before the suicide, and 40% do so during the prior week; 2) the vast majority of suicidal persons talk about suicide spontaneously or if asked; 3) 95% of persons who kill themselves have psychiatric illnesses, most of which respond to treatment; 4) educating community practitioners about suicide lowers local suicide rates.

### *Suicide Assessment*

Ask all persons with behavioral problems if they have suicidal thoughts. To my knowledge, there is no case in medical history where asking about suicide caused suicide. Some persons malingering that they are suicidal to gain hospital admission, but if a malingeringer is hospitalized needlessly, experienced psychiatric inpatient staff figure this out and discharge the patient expeditiously.

### **Diagnoses Associated with Suicide**

*All mental disorders have an elevated risk for suicide, except for mental retardation, dementia and agoraphobia.* The greatest risks are for depression, bipolar disorder, substance abuse (especially sedatives, alcohol and opioids), anorexia nervosa, schizophrenia, borderline personality disorder and panic disorder. *Persons with chronic general medical illnesses also have increased risks, especially those having head and neck neoplasms, AIDS/HIV, temporal lobe epilepsy (especially those needing surgery), and systemic lupus erythematosus; and persons being hemodialyzed.*

### **Mental Status Findings**

Mental status findings with increased risk are *talking about suicide; sustained sadness or anxiety; agitation; and hopelessness.* *For suicide assessment, clinical observations are more important than suicide demographics.*



## ***Suicide Epidemiology***

### **Prior Suicide Attempts**

*Prior suicide attempts* increase suicide risk: 60% of persons who kill themselves have made prior attempts, and 10% of attempters eventually kill themselves.

### ***Family History of Suicide***

Suicide *runs in families* as if it were genetic. This tendency cannot be explained solely by the fact that almost all psychiatric illnesses run in families.

### **Ethnic Group**

*Euro-Americans* are twice as apt as African Americans to commit suicide. Of ethnic groups studied to date, highest rates are for 1) *Native Americans*, next highest for 2) *Euro-Americans*, then 3) *Hispanic Americans*, then 4) *Chinese- and Japanese Americans*, and *least* for 5) *African Americans*. Rates for Arab Americans and Indo- and Pakistani Americans are unknown. Figure 1 covers suicide rates by age, gender and ethnic group.

### **Gender**

Gender carries a higher risk than ethnic group: *Euro-American males* are more likely to commit suicide than *African American males*, who in turn are more apt to kill themselves than *Euro-American females*, who in turn have higher rates than *African American females*.

### **Age**

Suicide is rare in children under age 11. Notable numbers of suicides occur between 11 and 14, and rates increase for older teenagers. For the US population in general, suicide rates are highest among the elderly, but this is due to the high rates for Euro-American men over age 40, who commit the majority of suicides in the US. Except for Euro-Americans and Chinese-Americans, for whom risk increases with age, all other ethnic groups have peak risks during young adulthood.

Though teenage suicide is less frequent than suicide in adulthood, teenage suicide is a huge problem because 1) it is the *third leading cause of death in teenagers*, and 2) it is often preventable.

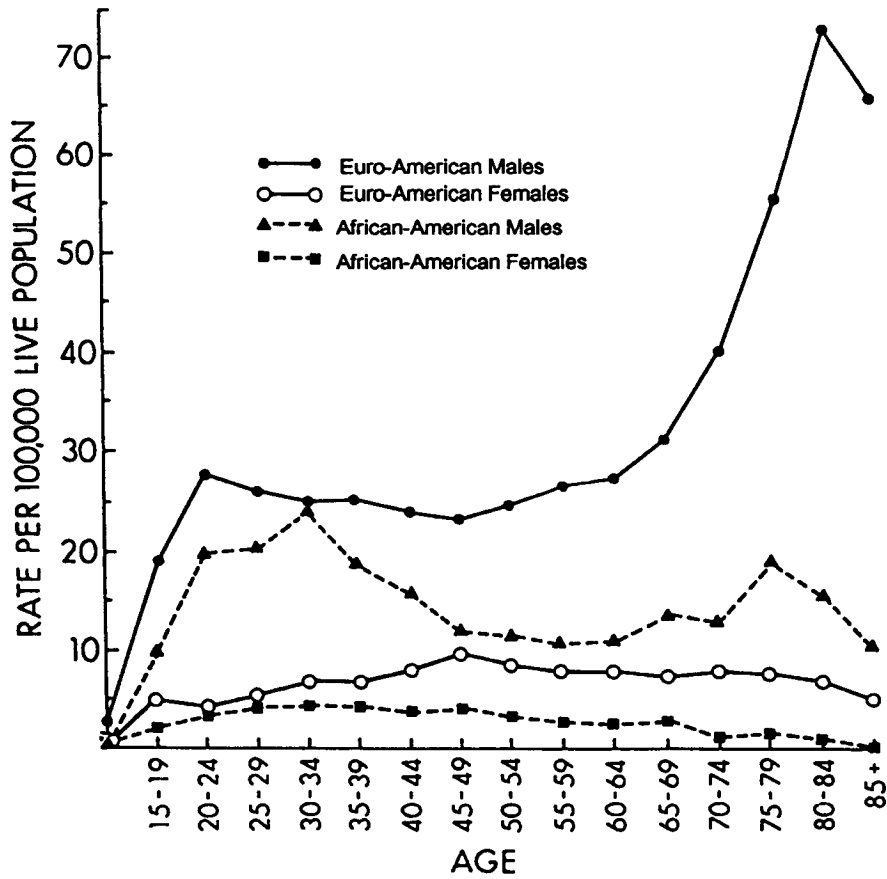
### **Geography**

In the US, the highest suicide rates are for *Western mountain states* like Nevada, Montana and Alaska. In states with large cities, the highest rates are in neighborhoods where many single, separated, divorced and widowed persons live.

### **Marital Status**

Suicide rates are lower for married persons than for *single, separated, divorced and widowed persons*. If you are married, this may be hard to believe.

FIGURE 1. SUICIDE RATES IN U.S.A. BY AGE, SEX & ETHNIC GROUP  
1988



Modified from Murphy GE. Suicide and attempted suicide, in Winokur G, Clayton PJ, eds. *The Medical Basis of Psychiatry*, 2nd Ed. Philadelphia: Saunders, 1994.

Figure 1. Suicide rates in the U.S.A. by age, sex and ethnic group. Modified from Murphy G.E. Suicide and attempted suicide, in Winokur G, Clayton PJ, eds. *The Medical Basis of Psychiatry*, 2nd Ed. Philadelphia: Saunders, 1994.

### Religion

In the US, suicide is *less frequent for Catholics* than for Protestants. Worldwide, Catholic countries (e.g., Ireland, Spain, Mexico) tend to have low rates. The world's highest rates are for Hungary, Austria, Rumania, Sweden and Japan. Mexican

Americans have higher suicide rates than Mexicans. Japanese Americans have lower rates than Japanese, likely because suicide is less stigmatized in Japan.

### **Season**

Worldwide, regardless of climate, suicide is most frequent *in the Spring, especially May*. There is a second—but lower—peak in the fall, especially October. Suicide is not more frequent during holidays, although during holidays, depressed persons often think about how they cannot enjoy themselves like others.

### **Occupation**

Men MDs are not more likely to commit suicide than age-matched men in the general population. In studies conducted before the mid-1970s, *women professionals* like MDs, chemists and psychologists had higher suicide rates than age-matched women. Whether this association still holds today, now that women have more access to professions, is unknown. A review of nine published studies indicates no medical specialty in which suicide is more common. Suicide is probably more common among police, artists, musicians and writers.

### **Smoking**

*Smokers* have higher risks for suicide than non-smokers. The more one smokes, the higher the risk. The increased risk could be due to the association of heavy smoking with alcoholism, other mental illnesses, and chronic general medical illness.

### **Suicide Methods**

For both sexes at all ages, *gunshots* are the most common method for completed suicide. Gun control laws lower teenage suicide rates.

### **Suicide Biochemistry**

Persons who attempt or commit suicide tend to have *lower 5-hydroxy indoleacetic acid (5-HIAA) levels in their CSF* than persons who make no attempts. Among persons who commit suicide violently (e.g., gunshot, jumping), CSF 5-HIAA levels are lower than among persons who commit suicide nonviolently (e.g., pills). What happens in the brain to account for this is unknown. Don't do spinal taps for suicide assessment!

Also, persons who attempt or commit suicide tend to have lower serum cholesterol levels than persons who make no attempts. Regardless, keep your cholesterol down. You're more apt to die from a stroke or heart attack than to kill yourself. And if you're suicidal, you'll know it!

## **HOMICIDE**

Homicide is the 10th leading cause of death in the US. Homicide rates from 1986–96 were the highest in US history. In 1992, the most lethal year ever, there were 26,000 murders. The second most lethal period was the Depression of the 1930s.

Homicide is less preventable than suicide. The diagnoses (antisocial personality disorder, substance dependence, somatization disorder) associated with homicide are less curable than those associated with suicide. Patients are less apt to tell their MD that they are planning homicide than that they are planning suicide.

### ***Homicide Prevention***

Community passage of gun control laws reduces community murder rates for several years.

### ***Risks for Professionals***

Twenty to 40% of mental health professionals have been assaulted. An *assault* is an attempt to injure someone. *Battery* is injury from an assault. No medical professional is immune.

### ***Violence Prediction by Mental Health Professionals***

For a given patient, 100% prediction of violence or suicide risk is impossible. However, statistically-based prediction (e.g., “This person has an increased likelihood of acting violently.”) is possible and necessary.

The best two predictors of assaultive behavior are 1) the clinician’s general impression (e.g., you’ll perceive a risk if a patient is irritable and threatening) and 2) whether the patient has been assaultive recently. Again, *the big behavioral science generalization: past behavior tends to predict future behavior.*

Other clinical predictors are anger, irritability, agitation, pacing, muscle clenching, fist pounding, shouting, making threats and vengeful statements and past assaultiveness.

### ***Homicide Demographics***

In violence assessments of individuals, clinical assessment is more important than epidemiologic factors. But for the population, epidemiology is important for violence prevention.

#### **Age**

The ages of highest likelihood of killing or maiming someone are 15–34. Ages 15–34 are also the ages of highest likelihood of being murdered, but no age group is exempt.

#### **Gender**

In all societies, males are much more likely than females to act violently. However, violence by females is increasing in the US.

#### **Socioeconomic Status, Population Density and Collective Efficacy**

Being violent or a victim of violence is more common among the poor. One of the best demographic predictors of violence is *population density* (e.g.,

overcrowded dwellings with two or three children per room or a family of 12 in a small apartment). Reduced collective efficacy in a neighborhood is also associated with increased violence. *Collective efficacy* is the extent to which neighbors trust each other and are willing to help each other in situations conducive to violence or injury, such as a fight breaking out, children disrespecting an adult, or budget cuts in the fire station.

### **Knowing the Victim**

We tend to kill persons we know and hate. Logical! Why kill someone you like? About 50% of killers know their victims. The most common lethal situation is an argument among acquaintances over money or a love triangle.

### **Kinship**

Within families, persons are less likely to kill biologic relatives than relatives by marriage or adoption. For example, we are more apt to kill a stepparent than a biologic parent. But biologic relatives are not immune.

### **Ethnic Group, Population Density and Collective Efficacy**

The majority of homicides are *intra-racial*. Euro-Americans tend to kill Euro-Americans, Asians to kill Asians and so forth. Why? We are more apt to know, live with and work with persons of similar ethnic background. In the US, African Americans are statistically more likely to commit and be victims of homicide than any other ethnic group. This ethnic difference disappears when population density is accounted for.

### ***The Tarasoff Precedent: The Duty to Protect or Warn***

The 1976 California Supreme Court decision *Tarasoff vs. Board of Regents* set a national precedent that if a clinician believes his or her patient poses a serious threat to harm or kill an identifiable person or group of persons, the clinician must protect or warn the potential victim.

Mr. Poddar told his University of California psychotherapist that he wanted to kill his girlfriend Tatiana Tarasoff. The therapist called the campus police, who checked on Poddar and felt Poddar posed no risk. Soon after, Poddar killed Tarasoff, and her family successfully sued the U. of California on the grounds that the therapist had a *duty to protect or warn* Tarasoff, setting the national precedent.

If a patient who poses a serious threat can be hospitalized and treated successfully enough to eliminate the threat, and then treated as an outpatient absent the threat, the *duty to protect* has been discharged. If the patient does not have a treatable condition, or cannot be hospitalized and treated effectively, or escapes from the hospital, the clinician has a *duty to warn*. Some states require warning the potential victim. Some states require notifying the police. Some require both.

One way to discharge the duty to warn is to tell the patient "If you kill her, you'll be caught, jailed and receive a maximum penalty. You won't be able to cop an insanity plea. I know you don't want to spend your life in jail, right? If you agree, call her now, in my presence, and tell her you're thinking of hurting her, but that you will make every effort not to do so." Alternatively, the patient may let you

make the call in the patient's presence. It is usually best to discuss the warning with the patient, and usually unwise for you to warn the potential victim without the patient's knowledge. For example, in *Hopewell vs. Adebimpe*, a psychiatrist was successfully sued for sending a warning letter, without telling the patient, to the patient's employer, who then fired the patient.

## **ACCIDENTS**

Accidents are our fifth leading cause of death. There are 90,500 accidental deaths annually. Fortunately, accidental death rates have declined for each decade in this century.

Motor vehicles are the leading cause of accidental death, accounting for 44,000 deaths in 1993. Next most common are 2) falls, 3) poisoning, 4) drowning, and 5) fires and burns.

### ***Are Accidents Accidental?***

Most accidents are not totally random or unpredictable. Epidemiologists view accidents as they do diseases; they consider the characteristics of persons and situations. A 17 year old boy driving intoxicated at high speed on an icy road is far more likely to have an accident than you are while you read this book, unless you happen to be driving your car now.

### ***Accident Demographics***

#### **Age**

From birth to age 44, accidents are the leading cause of death. Although accidents are "only" the seventh leading cause of death in the elderly, the accidental death rates for persons over 65 (27,784 deaths annually) exceed the rates for any other age group.

#### **Gender**

Males are twice as likely as females to die in accidents. Accident rates are only slightly greater for males than females. Thus, accidents tend to be more severe in males.

#### **Ethnic Group, Population Density and Collective Efficacy**

At all ages, accidental death rates are higher for Native Americans than for any other U.S. ethnic group. Below age 20 and over age 70, accidental death rates are higher for Euro-Americans than for African Americans. Between ages 20–70, African American rates exceed those for Euro-Americans. Why? Your guess is as good as mine.

#### **Socioeconomic Status**

There is an inverse relationship between SES and accidents: poor persons are more likely to die in accidents than persons of middle or upper SES. Poor persons are more

apt to work in hazardous occupations and to live in unsafe dwellings (e.g., dwellings with space heaters, without fire alarms, without guard rails on upper story windows).

### **Geography**

Rural fatal accident rates exceed urban rates. Speed limits are higher on rural highways, and trauma centers less accessible. Mining and agriculture, the two most dangerous occupations, are rural occupations.

### **Timing**

Accident rates are highest in the summer, especially July. Accidents are more common on weekends, especially Saturdays. Be careful every day!

### **Occupation**

In decreasing order of risk of fatal accidents, the most dangerous occupations are 1) mining 2) agriculture (especially timber cutting and logging), 3) construction and 4) transportation.

### **Using a Cellular Phone While Driving**

Persons who use cellular phones while driving are more apt to have fatal accidents than matched controls who don't use cellular phones when they drive.

### **Accident Risks for Pedestrians**

Increased risks for pedestrians include being a child or being elderly, using streets as play areas, high speed limits, few pedestrian-control devices, and high density of curbside parking.

### **Substance Intoxication**

Alcohol and other drug intoxications are involved in >50% of accidents, both for accident victims and persons "responsible" for accidents.

### **Psychiatric Diagnoses**

Alcoholism, antisocial personality disorder and borderline personality disorder have an increased risk of accidents.

### ***Accident Prevention***

Accident prevention is a responsibility of society, not just our profession. Fortunately, societal interventions reduced accident rates dramatically in the past half century. Life saving interventions include seat belts, air bags, energy-absorbing steering columns, padded car interiors, child seats seating children in rear seats, speed limits, unsafe vehicle lawsuits, helmets for bicyclists and motorcyclists, *traffic calming* (diversion of high-volume, high-speed traffic from the city core and residential areas), swimming pool safety laws, childproof medicine containers, smoke alarms, building and workplace inspections, and efforts by advocacy groups like Mothers Against Drunk Driving.

## CHAPTER 19. DELIRIUM AND DEMENTIA

*Delirium and dementia* are common, serious syndromes often missed by MDs. Both are characterized by diffuse cognitive dysfunction, not just pathology in one or two brain regions. Table 1 compares delirium and dementia.

### DEMENTIAS

Many dementias can be categorized as either cortical or subcortical.

#### *Cortical Dementias*

Cortical dementias are characterized by *four A's: aphasias, amnesias, apraxias and agnosias*. *Amnesias* are memory problems. *Apraxias* are problems with complex movements (e.g., constructional dyspraxia) despite normal sensory functions and strength. *Agnosias* are the inability to recognize familiar information, as in *anosognosia* (nonrecognition of illness). The most common cortical dementias are Alzheimer's disease and Pick's disease.

#### *Subcortical Dementias*

Subcortical dementias are characterized by *three Ms: disturbances of 1) movement, 2) mood and 3) memory*. *Movement disturbances* include *psychomotor slowing* (slow gait, slow speech) in most cases, plus abnormal movements like choreoathetosis, hemiballismus or tremor. The most common mood disturbance is apathy, but atypical depressions and hypomanias (mild manic episodes) also occur.

The most common subcortical dementias are multi-infarct white matter dementias (*Binswanger's disease*), HIV encephalopathy and Parkinson's disease.

#### *Curable and Incurable Dementias*

Although prognosis is poor in 85–90% of dementias, 10–15% of dementias are curable. *Curable dementias* include endocrinopathies, vitamin deficiencies, hematomas, normal pressure hydrocephalus and Wilson's disease.

#### *Severe Behavioral Abnormalities in Dementias and Focal Brain Diseases*

*Lesions of the frontal lobes, temporo limbic system and caudate nucleus are more closely associated with severe behavioral abnormalities, including psychosis, than lesions elsewhere*. This explains why Pick's disease (frontotemporal dementia) is often mistaken for schizophrenia or bipolar disorder, and why Huntington's disease, with its main pathology in the caudate nucleus, usually shows severe behavioral disturbances, whereas Wilson's disease, with its main pathology in the putamen, is much less apt to cause severe behavioral disturbance.



**Table 1. Comparison of Delirium and Dementia**

	<b>Delirium</b>	<b>Dementia</b>
—Presence of diffuse cognitive dysfunction	Always (by definition)	Always (by definition)
—Alertness	Always abnormal (by definition). Clinical tests of alertness (e.g., letter cancellation test) always abnormal.	Normal (by definition) unless a delirium is superimposed on the dementia (e.g., pneumonia with fever causing delirium in an Alzheimer patient)
—Sleep patterns	Usually abnormal, with fitful sleep, reduced nighttime sleep, frequent daytime sleep	Usually normal unless another disorder superimposed
—EEG findings	Most delirious patients have diffuse EEG slowing (exception: delirium tremens, with diffuse fast EEG)	Early in illness, EEG is normal or there is a focal abnormality at lesion site
—Prognosis	15–30% die within a month, especially those not well treated; those who recover usually recover fully with normal life span.	Well treated, short term risk of death small; for 85–90% of demented persons, long term prognosis is progressive deterioration with life span shortened many years

### ***Specific Dementias***

#### **Alzheimer's Disease**

*Alzheimer's disease* (AD) accounts for 50% of dementias. Its incidence increases with age. It is familial. One familial subtype has a *chromosome 21 abnormality* which runs in families along with AD, Down syndrome and myeloproliferative disorders. All persons with Down syndrome develop AD if they live long enough. Another subtype has a *chromosome 19 apolipoprotein abnormality* linked to excessive amyloid deposition. *Chromosome 1 and 14* abnormalities have also been detected. AD has an autoimmune etiologic component.

Histopathology includes *deficient choline acetyl transferase in the nucleus basalis* with high density of *senile (amyloid) plaques and neurofibrillary tangles* there and in the temporoparietal region. The earliest abnormalities are temporoparietal, with *reduced temporoparietal metabolism and blood flow* on single positron emission computed tomography (SPECT) and cognitive abnormalities on digit span (repeating numbers forwards and backwards), digit symbol (ability to link numbers and symbols quickly), and vocabulary testing.

First degree relatives of AD patients, who are at risk for AD, should have cognitive testing yearly starting at age 40. If temporoparietal abnormalities are identified, the relative should take a nonsteroidal antiinflammatory agent daily to possibly slow the onset and progression of the disease.

### **Pick's Disease (Frontotemporal Dementia)**

*Pick's disease (frontotemporal dementia)* is the second most common cortical dementia. Etiology is unknown.

Pathology involves severe atrophy in the frontal convexities and temporal poles. Because lesions in these sites are closely associated with severe behavioral abnormalities, Pick's disease is often mistaken for schizophrenia or bipolar disorder until cognitive testing is done, revealing dementia, and CT or MRI reveal frontotemporal atrophy.

Severe bilateral temporal lobe disease (as in Pick's disease) is sometimes associated with *Kluver-Bucy syndrome*, characterized by dementia, placidity, hyperorality (ingesting, or placing in one's mouth, inedible objects), and inappropriate sexuality (e.g., public masturbation, inappropriate sexual advances).

### **Multi-Infarct Dementia (MID)**

MID is due to multiple small strokes, often in subcortical white matter (*Binswanger's disease*). It is more common in men than women, and in middle aged and elderly persons than young adults. It is associated with arteriosclerosis elsewhere in the body. Risk factors include hypertension, diabetes, hypercholesterolemia, smoking, obesity, sedentary lifestyle, and family history of cardiovascular disease.

Onset may be sudden, followed by mild improvement when edema subsides, followed by consistent impairment and then—unless risk factors are reduced—another sudden deterioration. This decline-stability-decline pattern is a *stepwise progression*.

Its most common clinical presentation is a *subcortical dementia*. Suspect MID in persons with late onset (i.e., age 55–80) atypical depression. MRI is more sensitive than CT in confirming the diagnosis. Treatment includes physical therapy and prevention of future strokes. The depression often responds to antidepressants or ECT.

### **Parkinson's Disease (PD)**

*Parkinson's disease* is common, and 20–40% of PD patients develop subcortical dementia. Typical age of onset is 55–65. The main pathology is degeneration of dopaminergic neurons in the substantia nigra. There are many cytoplasmic *Lewy bodies* in the substantia nigra and other structures. PD has a hereditary subtype associated with an *autosomal dominant chromosome 4 mutation*, but most cases are idiopathic. Head trauma, carbon monoxide poisoning, viral encephalitis, arteriosclerosis and *MPTP* (an illicit synthetic opiate) *toxicity* all cause a Parkinson-like disease.

Parkinson's findings are slow or festinating (starting slow and progressing rapidly as the trunk tilts forwards) gait, expressionless face, pill-rolling resting

tremor (rhythmic, thumbs apposing forefingers, more intense with arms at rest), oily skin, drooling, rigidity, and micrographia (small handwriting). Although substantia nigra degeneration is visible on MRI, brain imaging is not needed for diagnosis.

Treatments include L-DOPA, amantidine, bromocriptine, anticholinergic antiparkinson agents (e.g., benztropine) and ECT.

### **Huntington's Disease (HD)**

*Huntington's disease (HD)*, a rare disorder, is caused by an *autosomal dominant abnormality on chromosome 4*. Penetrance is complete; persons with the HD genotype who live to age 50 will develop the disease. The main pathology is *caudate nucleus atrophy*, appearing on MRI as a *butterfly pattern* with concavity of the lateral aspects of the lateral ventricles.

Onset is usually between 30–50, with choreoathetoid movements and severe behavioral abnormalities, including subcortical dementia, depression with high suicide rate, psychosis and violence. HD is incurable. Deterioration is progressive with shortened life span. Neuroleptics like haloperidol reduce behavioral symptoms somewhat, but do not change HD's progression.

Genetic counseling is mandatory. Prenatal screening by amniocentesis or chorionic villus sampling is available, as is chromosome analysis of blood for children or adults at risk. Given the complete penetrance and awful prognosis, some parents or children prefer not to know the child's genotype.

### **Wilson's Disease**

Wilson's disease has a rapidly progressive juvenile type with onset between ages 7 and 15 and a slowly developing adult type with onset between 19 and 35. An *autosomal recessive abnormality on chromosome 13* reduces 1) copper excretion into the gut and 2) ceruloplasmin production, causing copper deposition in the cornea, liver and basal ganglia, especially the *lentiform nuclei* (putamen and globus pallidus). In the cornea, it produces a pathognomonic *Kayser-Fleischer ring*, seen by slit lamp. In the liver, it causes hepatitis or cirrhosis.

In the lentiform nuclei, it causes dystonia and mental retardation (in a child) or subcortical dementia and wingbeating (shoulder) tremor (in an adult). Serum ceruloplasmin <20 mg/ml and increased 24 hour urinary copper excretion are specific for the disease. CT, MRI and PET usually show lentiform nucleus abnormality.

Prompt diagnosis with treatment including a low copper diet and a chelating agent like d-penicillamine are so helpful that the patient can lead a reasonable, long life. Missed diagnosis leads to neuropsychiatric impairment and shortened life span.

### **Normal Pressure Hydrocephalus (NPH)**

*Normal pressure hydrocephalus* has many causes, including infection (e.g., general paresis), subarachnoid hemorrhage and vitamin deficiency. Reduced CSF absorption—the hydrocephalus is communicating, not obstructive—causes hugely dilated ventricles, obvious on CT or MRI, with normal CSF pressure.

Clinical presentation is a *triad of dementia, urinary incontinence and gait impairment*. Management includes 1) treating the underlying cause and 2) ventriculo-peritoneal neurosurgical shunting. Outcome is usually good.

### **Wernicke's Encephalopathy**

*Wernicke's encephalopathy* is a heritable problem of thiamine utilization. The disease (the phenotype) is precipitated by malnutrition with thiamine deficiency in alcoholism, anorexia nervosa or fad diets. Pathology includes punctate hemorrhages in the periaqueductal gray matter and thalamus.

Clinical presentation includes 1) delirium or dementia and 2) eye signs, either a) nystagmus or b) *abducens muscle (sixth nerve) palsy*. Untreated, it may be fatal. Promptly recognized and treated with thiamine, outcome is usually good. Untreated, it may lead to alcoholic amnestic disorder or may be fatal.

### **Alcoholic Amnestic Disorder (Korsakoff's Psychosis)**

In *alcoholic amnestic disorder (Korsakoff's psychosis)*, pathology is in the limbic system, including the *mammillary bodies*. The patient has severe deficits in learning new information, leading to severe recent memory deficits. This often leads to inaccurate guessing at answers to questions (*confabulation*). An example: Doctor: "Where are you now?" Patient: "I'm in the train station." Doctor: "Are you sure?" Patient: "No, I'm in the house . . . I'm in the MacDonaldis." Confabulation is guessing, not malingering. Treatment includes thiamine and multivitamins.

## CHAPTER 20. SUBSTANCE ABUSE

### DEFINITIONS

Society defines what is a substance of abuse. For example, caffeine is addicting, but we caffeine addicts aren't labeled that way.

#### *Drug Dependence*

*Drug dependence* is the use of substances with sufficient frequency to produce tolerance, withdrawal and characteristic behaviors:

*Tolerance* occurs when (1) increasing doses of the drug are needed to obtain the same effect or (2) continued use of the drug yields a lesser effect.

*Withdrawal* occurs when stopping the drug produces a characteristic withdrawal syndrome for that drug and, if withdrawal occurs, that drug is taken to relieve the symptoms.

*Other characteristic behaviors* are (1) considerable time spent obtaining the drug; (2) reduced social and occupational functioning; and 3) continued drug use despite knowing of dangerous consequences.

#### *Drug Abuse*

All drug-dependent persons are drug abusers, but not all abusers are dependent. The distinction between dependence and abuse is prognostically unimportant because, on followup evaluation, outcomes for persons diagnosed with drug abuse are the same as for persons diagnosed with drug dependence.

*Drug abuse* is defined when the following are present in the absence of dependence:

- (1) *social* (e.g., marital discord), *occupational* (e.g., absenteeism) and *legal* (e.g., arrests for driving under the influence) *consequences* of drug use
- (2) using substances in *hazardous situations* (e.g., using machinery, driving a car)

### PREVALENCE OF SUBSTANCE ABUSE

In the U.S. the *prevalence of alcohol abuse* is 11–16%, and of abuse of other substances (not including caffeine or nicotine) is 5–6%. Substance abuse is *seriously underdiagnosed* in medical settings, partly for lack of detailed and non-judgmental questioning by clinicians and partly because many substance abusers *deny that they have a problem*. Many persons use *multiple substances* (e.g., alcohol and cocaine).

## SUBSTANCE INTOXICATIONS

Intoxicated, or possibly intoxicated, persons should have *urine screening for drugs of abuse* and *blood studies for alcohol levels and presence of phen-cyclidine*. In most states, blood alcohol of *0.1 grams/100 cc. or greater* means that the patient is legally intoxicated. About 10 states lowered their limit to *0.08 gm./100 cc.*

### ***Alcohol and Sedative Hypnotic Intoxication***

#### **Manifestations**

The manifestations of alcohol and sedative-hypnotic (e.g., barbiturates, benzo-diazepines) intoxications are essentially the same, consisting of

- (1) *a euphoric, irritable or sad mood,*
- (2) *slurred speech,*
- (3) *ataxia,*
- (4) *nystagmus,*
- (5) *disinhibition, and*
- (6) *combativeness.*

Acute alcohol intoxication can mimic mania and can induce nonmelancholic depression. Thirty per cent of patients with bipolar disorder are alcoholic.

#### **Management**

Most alcohol and sedative-hypnotic intoxications are managed by rest and supportive conversation in a quiet room and require no antidote. Patients should be observed for withdrawal symptoms. Severe intoxications require support of vital functions.

### ***Inhalant Intoxication***

*Volatile organic solvents* (e.g., airplane glue, hair spray, spot remover, lighter fluid), can be *purchased legally by minors* and inhaled to produce symptoms resembling those of alcohol or sedative-hypnotic intoxications, including *euphoria, disinhibition, ataxia and slurred speech*. Visual hallucinations may occur. These solvents affect *GABA receptors*. Inhaling from a soaked rag is termed *huffing*. Sometimes this produces a *rash around the nose and mouth*. The *odor of the inhaled substance* can be detected on the breath, skin or clothes.

Effects of intoxication wear off within an hour and heavy users may inhale hourly. Acute treatment is the same as for alcohol intoxication. Complications include pulmonary, renal, hematopoietic and neurologic disease, the latter including cerebellar and pyramidal tract disease and peripheral neuropathy.

Addiction is rarely a complaint of inhalant abusers, many of whom abuse other substances.

## ***Opiate and Opioid Intoxication***

### **Manifestations**

- (1) *euphoria*
- (2) *decreased body temperature*
- (3) *pinpoint pupils*
- (4) *nausea*
- (5) *drowsiness*, leading to *sleep (nodding)* with *cigarette burns of the chest* because the patient's responsiveness to pain is reduced by the opiate
- (6) if severe, *respiratory depression with cyanosis*

Opiate and opioid intoxication do not induce combativeness.

### **Management**

If the intoxication is severe or the patient is comatose, the antidote is *intravenous naloxone (Narcan)*.

## ***Cocaine Intoxication***

Although many more persons are addicted to caffeine or nicotine than cocaine, cocaine is probably the most physiologically addicting of all drugs. Its pleasurable effects result from its action on dopamine 2 receptors. *Crack cocaine and free-based cocaine*, produced by splitting of the HCl molecule from regular cocaine, are more addicting and physiologically deadly than regular cocaine.

### **Systemic Manifestations**

- (1) *tachycardia*
- (2) *hypertension*
- (3) *hyperthermia*
- (4) *dilated pupils (mydriasis)*
- (5) *sweating*

There is a *risk of death from a single dose*, even in a person in normal general health, from *hyperthermia, seizures, stroke or myocardial infarction*. For pregnant women, there is a risk of fetal death from *abruptio placentae*.

Often, substance abusers drink alcohol and inhale or inject *cocaine* simultaneously. This produces *cocaethylene*, which produces an intense "rush" and is physiologically more intense and dangerous than cocaine or alcohol alone.

### **Behavioral Manifestations**

- (1) *euphoria or emotional lability*
- (2) *tactile hallucinations*, also called *formications, cocaine bugs*, or *Magnan's sign*
- (3) *geometric visual hallucinations* (resembling the visual experiences of migraine)

## **Management**

- (1) As for the other intoxications, the patient should be in a *calm, supportive setting*.
- (2) *Urine should be acidified with ammonium chloride or cranberry juice* to enhance excretion.
- (3) If the patient is psychotic, a *neuroleptic (haloperidol is best)* should be prescribed.
- (4) For severe hypertension, the *alpha blocker phentolamine (Regitine)* should be prescribed.

## ***Amphetamine Intoxication***

The manifestations, complications and management of amphetamine intoxication is the same as for cocaine intoxication.

## ***Anticholinergic Intoxication***

Anticholinergic substances include atropine, scopolamine, antihistamines, over-the-counter cold or sleeping medications, antidepressants, low potency neuroleptics, and antiparkinson agents.

## **Manifestations**

- (1) *Red as a beet*: reddening of the skin, especially the face
- (2) *Dry as a bone*: lack of salivation, sweating and vaginal lubrication
- (3) *Blind as a bat*: reduced accommodation with blurred vision
- (4) *Mad as a hatter*: delirium

## **Management**

Subcutaneous *physostigmine*

## ***Lysergic Acid Diethylamide Intoxication***

*Lysergic acid diethylamide (LSD)* is a prototype hallucinogen. It acts on *serotonin receptors*.

## **Systemic Manifestations**

- (1) dizziness
- (2) weakness
- (3) tremor
- (4) nausea
- (5) cognitive impairment
- (6) slightly dilated pupils



### **Behavioral Manifestations**

- (1) *euphoria*
- (2) *multi-modal hallucinations, especially visual and sometimes kaleidoscopic*
- (3) *double perceptions, such as hearing a smell (synesthesias)*

### **Management of Mild Intoxication**

- (1) supportive environment
- (2) one dose of a benzodiazepine or neuroleptic

### **Management of Severe Intoxication**

- (1) hospitalize
- (2) neuroleptic medication (haloperidol is best)
- (3) if not better in three days, ECT

### ***Marijuana Intoxication***

The active ingredient in marijuana is *tetrahydrocannabinol (THC)*, which links to *G protein receptors*.

### **Systemic Manifestations**

- (1) *conjunctival injection*
- (2) *craving for sweets*

### **Behavioral Manifestations**

- (1) *euphoria*
- (2) *distorted time sense*
- (3) *impaired cognitive functioning*; in experiments, pilots who smoked a "joint" of marijuana could not properly fly planes in flight simulations several hours later.
- (4) can cause *hallucinations and delusions*

### **Management**

Supportive conversation in a friendly, calm environment usually suffices. No antidotes are necessary.

### ***Phencyclidine Intoxication***

*Phencyclidine (PCP, angel dust)* was first used as a human anesthetic until anesthesiologists noticed their patients were delirious and combative. PCP is still used as an animal anesthetic. PCP acts on *glutamate NMDA receptors*.

### **Systemic Manifestations**

PCP affects the *brainstem and vestibulocerebellar system*. Systemic manifestations include the following:

- (1) *dizziness*
- (2) *nystagmus*
- (3) *analgesia*
- (4) *narrow or normal-width pupils*

### **Behavioral Manifestations**

- (1) *irritability and combativeness*
- (2) *cataplexy* (motionlessness) and other signs of *catatonia* (p. 92)
- (3) *delirium*

### **Management**

- (1) hospitalization often required
- (2) restraints often required (restrained patients must be supervised carefully)
- (3) *acidify urine with ammonium chloride or cranberry juice*
- (4) *benzodiazepines*

## **WITHDRAWAL STATES**

### ***Alcohol and Sedative-Hypnotic Withdrawal***

#### **Manifestations of Alcohol or Sedative-Hypnotic Withdrawal**

Just as alcohol and sedative-hypnotic intoxication are similar, withdrawals from these substances are similar and include the following:

- (1) *tachycardia*
- (2) *hyperthermia*
- (3) *sweating*
- (4) *coarse tremor (the shakes)*
- (5) *delirium*: Delirium plus tremor is *delirium tremens (DTs)*, which is much more common with alcohol withdrawal than with sedative-hypnotic withdrawal. Alcoholics who were drinking daily before admission to a hospital should be observed carefully for early signs of DTs, or may be given a benzodiazepine (e.g., chlordiazepoxide) to prevent DTs.
- (6) *grand mal seizures*, which occur more quickly with sedative-hypnotic than with alcohol withdrawal
- (7) *death* can result from untreated DTs, seizures or hyperthermia.

Although manifestations are similar, management of alcohol withdrawal differs from that of sedative-hypnotic withdrawal, as follows:

#### **Management of Alcohol Withdrawal**

There are two common treatments of alcohol withdrawal:

- (1) *long-acting benzodiazepines* like *chlordiazepoxide (Librium)*
- (2) *carbamazepine (Tegretol)*

## **Management of Sedative-Hypnotic Withdrawal**

Although most sedative-hypnotics have *cross-tolerance* with each other (e.g., benzodiazepines can be substituted for barbiturates without withdrawal symptoms), and although progressive decrements in dose of the drug to which the patient is addicted seems logical, the best treatment is *substitution of a long acting barbiturate like pentobarbital* as follows:

- (1) Give *test doses of pentobarbital* enough to produce mild intoxication.
- (2) Starting with the dose required to maintain mild intoxication in the prior 24 hours, reduce the dose by 10% per day in 10 days.

## **Opiate and Opioid Withdrawal**

### **Manifestations**

- (1) *tachycardia*
- (2) *rhinorrhea* (can be faked by pulling nasal hairs)
- (3) *lacrimation* (can be faked by pulling nasal hairs)
- (4) *pain*: joint, abdominal, anywhere else (can be malingered)
- (5) *nausea, vomiting or diarrhea*
- (6) *piloerection (gooseflesh)*, which cannot be faked)
- (7) *leukocytosis*
- (8) *spontaneous ejaculation* (there are better ways to ejaculate)
- (9) *fetal death* during a pregnant woman's withdrawal; death of a teenager or adult rarely if ever occurs from opiate withdrawal alone

### **Management**

- (1) Patients (exception: pregnant women) can withdraw "*cold turkey*," meaning without medication, without risk of dying. ("Cold turkey" refers to piloerection resembling turkey skin.) Nevertheless, untreated opiate withdrawal is uncomfortable, and most opiate withdrawals are treated with *dolophine (Methadone) substitution* or by a combination of *clonidine and a benzodiazepine*.
- (2) For opiate addicts with painful diseases, stab wounds or surgical wounds, treatment is to substitute Methadone to cover the addiction and prescribe whatever analgesic (anything from aspirin to morphine) a non-addicted person would require for pain.

## **Cocaine and Amphetamine Withdrawal**

### **Manifestations**

Cocaine and amphetamine withdrawal are characterized by *hypersomnia, non-melancholic depression and hyperphagia*, symptoms opposite to cocaine and amphetamine intoxications.

**Management**

Cocaine and amphetamine can be abruptly discontinued safely. Prescription of *desipramine, naltrexone, amantidine, or bromocriptine*, the latter three being dopamine agonists, reduces cocaine craving.

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# SECTION 7. BEHAVIORAL NEUROSCIENCE

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## CHAPTER 21. BRAIN ORGANIZATION AND BEHAVIOR

*by Frederick S. Sierles, M.D., and Michael Alan Taylor, M.D.*

### NEUROANATOMIC SITES AND CIRCUITS AND NOR- MAL AND ABNORMAL BEHAVIORS

Since the time of Broca and Wernicke we have learned many links between brain anatomy and behavior. However strong a brain-behavior correlation, this does not always prove a given region “controls” a function. For example, 1) brainstem, thalamic or frontal lobe disease causing inattention can make a patient err in copying the outline of a geometric shape, a “function” of the non-dominant parietal lobe and the corpus callosum. 2) Severe limbic dysfunction can cause the frontal lobes to “shut down,” reducing frontal functioning and implying mistakenly that frontal dysfunction was the main problem. The brain works in systems.

### FUNCTIONS OF THE BRAIN

*The brain's functions* are 1) behavior and 2) integrating the functions of other organs. Through behavior, the brain mediates between the internal physiologic milieu (e.g., endocrine secretion) and the outside world. Behavior is observable action in response to internal (e.g., thoughts and emotions) and external cues.

### WAYS TO VIEW THE BRAIN

There are three ways to view the brain: 1) left to right, meaning dominant and nondominant hemispheres, 2) front to back, meaning an anterior brain system for thought and action and a posterior data base brain and 3) bottom to top, meaning reptilian, paleomammalian and neomammalian brains (*MacLean's triune brain*). Left to right elucidates the brain's language, visual-spatial and perceiving functions. Front to back elucidates an acting-planning-thinking brain and a sensing-perceiving brain.

## ***The Brain from Left to Right: Hemispheric Dominance***

### **Dominant Hemisphere**

The *dominant hemisphere* is the one that handles *propositional language*, which consists of grammar, syntax and semantics. *Grammar* is the rules of language. *Syntax* is organizing words into sentences. *Semantics* is the meaning of words. For 95% of persons, the dominant hemisphere is the left. Right dominance is most likely in pure left handers for whom left-handedness runs in the family. Pure left handers are persons who perform all difficult functions with the left hand, foot or eye: writing, using a knife, pouring liquids, pushing objects with one leg, looking in a microscope.

### **Nondominant Hemisphere**

The *nondominant hemisphere* is the “other hemisphere.” It handles *prosody* (the emotional and gesturing part of language) visual-spatial information, and recognizing familiar information that people take for granted. We take for granted that we 1) are aware of the left side of our bodies and the left side of space (the left environment), 2) recognize our friends and family, and 3) know where we are seriously ill. This is because our nondominant hemispheres are functioning well. On the other hand, some persons with nondominant parietal lobe disease don’t notice their left hemiparesis, repeatedly try to walk, and suffer injuries as a result. Table 1 compares the dominant and nondominant hemispheres.

### **Left and Right Hemispheres**

The *left hemisphere*, dominant in 95% of persons, processes sensory information from the right side of space and the right side of the body, and controls movement on the right side of the body. The *right hemisphere* handles sensory information from the left side of space, and controls left sided movement.

### ***Connecting the Left and Right Hemispheres: The Corpus Callosum***

The *corpus callosum* links the left and right hemispheres, letting each hemisphere know what the other is doing. One test of callosal integrity is having the patient, eyes closed, tie his or her shoelaces. Another is having the patient, eyes closed, mimic with one hand a position in which you place the fingers, palm, and wrist of the other hand. For example, you might place a patient’s left hand in a “thumbs up” position and ask her to mimic this with her right hand. Findings suggesting callosal damage include 1) inability to tie one’s shoelaces with one’s eyes closed; 2) inability to mimic with one hand the position in which the other hand is placed; 3) inability to copy the outline of a simple geometric shape (*constructional dyspraxia*) with one’s preferred hand (e.g., right) and then copying the shape correctly with the other hand (i.e., left) correctly; 4) inability to pantomime an action (e.g., turning an imaginary key) with one’s left hand and then correctly

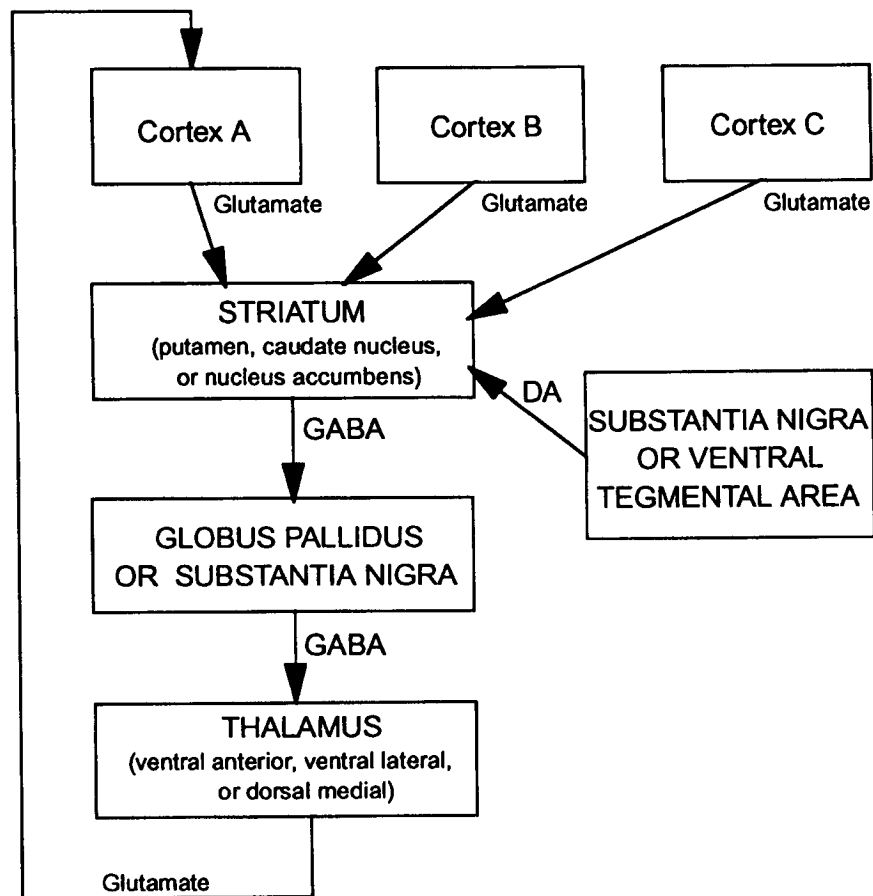
**Table 1. Comparison of the Dominant and Nondominant Hemispheres**

	<b>Dominant</b>	<b>Nondominant</b>
Gestalts vs. details	Detail oriented. When you look at a forest, your dominant hemisphere sees individual trees.	Gestaltic (holistic). Your nondominant hemisphere sees the forest, not individual trees.
Information processing	Handles information in steps and sequences. Processes one item at a time.	Handles information in parallel. Processes multiple items simultaneously.
Logic and creativity	Uses rules and logic.	Creative. Solves problems without knowing how.
Emotionality	Handles social emotions like polite smiling. Less emotionally expressive.	Handles primary emotions like anger and fear. More emotionally expressive. Left side of face more expressive than right side of face.
Speed of information processing	Handles high frequency information, like the contents of this chapter	Handles low frequency information, like the outline of this page
Anatomy	Larger planum temporale (part of temporal lobe that includes Wernicke's area). Larger temporo-parieto-occipital area. Larger Sylvian fissure and lateral ventricle. More gray matter.	Larger frontal area. More white matter. More dendritic arborization.
Neurotransmitters	Greater concentration of dopaminergic neurons.	Greater concentration of serotonergic neurons.

pantomiming the action with one's right hand; and 5) being able to write but unable to read, including being unable to read one's own writing (*alexia without agraphia*).

### ***The Brain from Front to Back: Anterior and Posterior Brain Systems***

The *anterior brain system* consists of 1) the frontal lobes, 2) frontal-basal ganglia-thalamic circuits (Figure 1), 3) part of the posterior limb of the internal capsule, and 4) cerebellar input to the frontal lobe. The anterior brain is the *action*



**Figure 1.** Prefrontal-striato-pallido-thalamo-cortical loops. Cortex A represents the prefrontal cortex specific to a particular loop, while Cortex B and Cortex C represent postrolandic areas. Only the prefrontal cortical area receives thalamic projections. Modified from Crosson B. *Subcortical Functions in Language and Memory*. New York, Guilford, 1992.

*part of the brain, controlling movement, thinking and planning. It is blind and deaf. It asks the back of the brain, perhaps literally, "What's going on out there? We must know before I act."*

### ***Posterior Brain System***

The *posterior brain system* obtains the data from which the anterior brain acts. The posterior brain is the temporal, parietal and occipital lobes and their associated subcortical structures, including the thalamus and part of the posterior limb of the internal capsule.



## **Receiving Information and Organizing It Into Patterns**

The posterior brain receives unformed, primitive sensations from sense organs via the thalamus and the posterior limb of the internal capsule, and organizes these into recognizable patterns, such as feeling a quarter in one's hand, seeing a door, and hearing a specific word.

## **Consciousness and the Thalamus**

Every 12–13 milliseconds, the thalamus sweeps across the sensory cortices like a beacon to form multiple-millisecond sensory pictures, and sends this information to the inferior parietal lobule. *Each thalamic sweep provides the awareness of consciousness.*

## **Initial Interpretation and the Inferior Parietal Lobule**

Having received this information from the sensory cortices and the thalamus, the inferior parietal lobule of the dominant hemisphere interprets this information. For example, "The man with the blue facial warpaint is directing his troops." *The inferior parietal lobule* (Brodmann's areas 39,40,43) consists of the angular gyrus (area 39), the *supramarginal gyrus* (40) and the *parietal operculum* (43). *Operculum* means adjacent to the insula. Then, the inferior parietal lobule sends efferents to the prefrontal cortex, which directs thoughts and actions in response; for example, "Let's signal to the man in the warpaint that we want to negotiate."

## ***The Brain from Bottom to Top***

The brain viewed from bottom to top consists of the reptilian brain, the paleomammalian brain and the neomammalian brain (Maclean's triune brain). The neomammalian and paleomammalian brains comprise the *cerebral cortex*.

### **Reptilian Brain**

The *reptilian brain* is the brainstem, the vestibular system related to the brainstem, part of the cerebellum, part of the amygdala, and the thalamus, hypothalamus, basal ganglia and hippocampus. Its functions are arousal, activation, balance, information input and output, memory, homeostatic and procreative drives, and flight and fight. The brainstem contains nuclei of cells that produce much of the brain's serotonin (pontine median raphe nuclei), norepinephrine (locus ceruleus) and dopamine (substantia nigra).

### **Paleomammalian Brain**

The *paleomammalian brain* is the limbic system: the septal nuclei, mammillary bodies, fornix, hippocampus, parahippocampus, entorhinal cortex, part of the amygdala, and the hypothalamus and cingulate gyrus. Its functions are appetitive drives, emotions (including a fight or flight response), learning, and memory.

## Neomammalian Brain

The *neomammalian brain* is the cerebral hemispheres, cerebellar cortices, and corpus callosum. Its functions are action (frontal lobe and cerebellum), comprehension and thought. The cerebellum, which has cognitive as well as motor functions, is highly developed in humans.

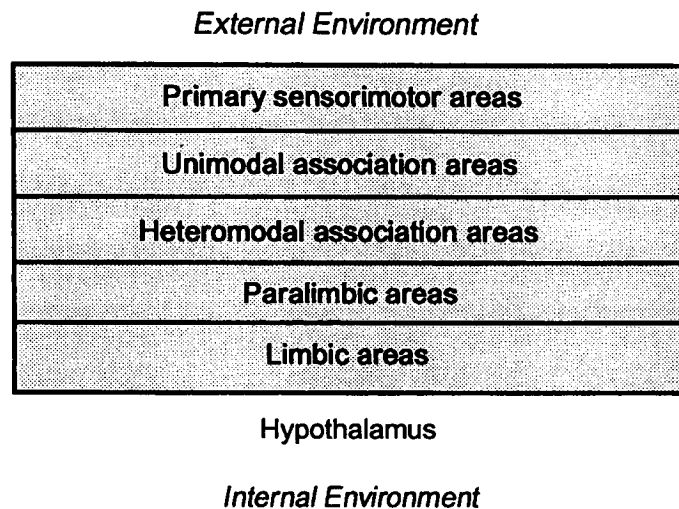
## Cerebral Cortex: Neomammalian and Paleomammalian Brains

The *cerebral cortex* consists of the neomammalian and paleomammalian brains. The cerebral cortex includes the primary sensorimotor cortex, the unimodal association areas, the heteromodal association areas, the limbic system, and the paralimbic areas (Figures 1–3).

## Primary Sensorimotor Areas: Sensory Component

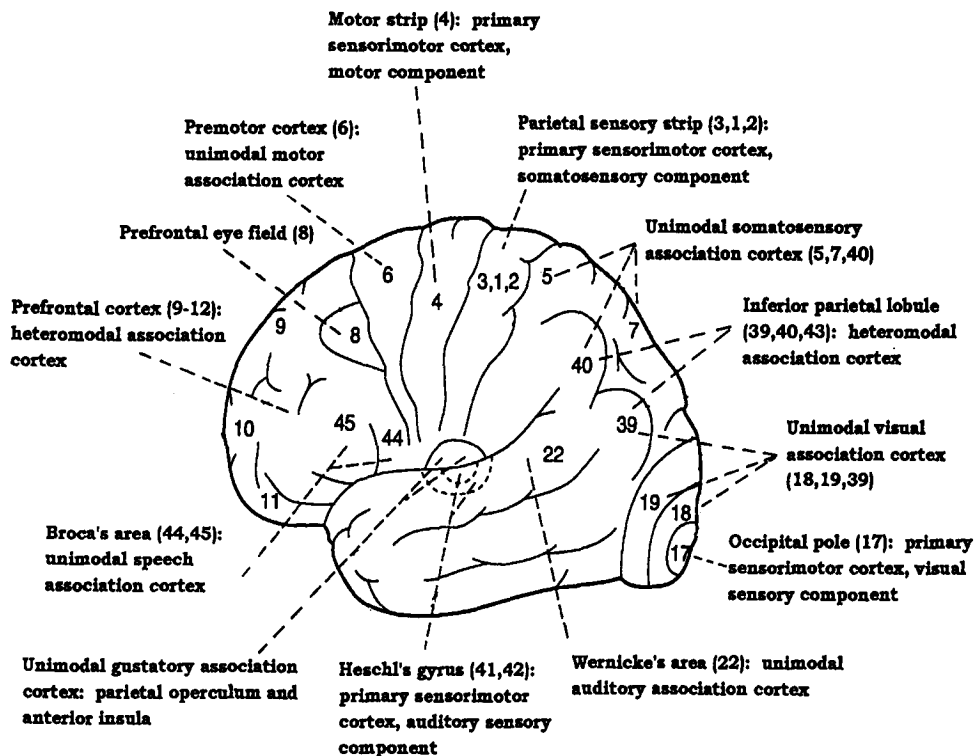
The *sensory component of the primary sensorimotor areas* (Figures 2, 3) receives primitive sensations including sound, taste, light, color and touch, all of which pass first through the thalamus and internal capsule. One exception: smell bypasses the thalamus.

*Heschl's gyrus* (Brodmann's area 41) receives sound. The *occipital pole* (area 17) receives light and color. The primary olfactory cortex (area 34) receives smell. The *parietal sensory strip* (areas 3, 1 and 2) receives pain, temperature, touch and information about position and vibration. It contains a sensory homunculus (actually, overlapping homunculi for different sensations) that begins inferolaterally



**Figure 2.** Cortical zones and their connections. Modified from Mesulam M-M. *Principles of Behavioral Neurology*. Philadelphia: FA Davis, 1985.

with the abdominal viscera and mouth and moves superomedially to the trunk, hip, leg, foot and toes.



**Figure 3.** Cerebral cortex of the left hemisphere (dominant in 95% of persons. Modified from Fix J. *High Yield Neuroanatomy*. Baltimore: Williams and Wilkins, 1996.

### Primary Sensorimotor Areas: Motor Component

The *motor strip* (area 4) begins the corticospinal and corticobulbar tracts. It contains a motor homunculus with anatomic parallels to the sensory homunculus. It is not a control center. It is a relay station with relatively few pyramidal cells that send messages that are generated in the prefrontal and premotor cortices and mediated by subcortical structures.

### Unimodal Association Areas: Sensory Component

*Unimodal association areas*, which have sensory and motor components, facilitate responsiveness to the outside world. The *sensory component of the unimodal association areas* organize primitive sensations from a single (hence, "unimodal") sensory modality (e.g., vision) into recognizable patterns and link these patterns

to limbic system memories in the same modality and to multimodal or symbolic memories. *Memory lends meaning to perception.*

Brodmann's areas 18, 19 and 39 handle visual pattern recognition. The gustatory association cortex likely lies in the parietal operculum and anterior insula. The olfactory association cortex is in the parahippocampal gyrus (area 28). Complex sensations like position sense, stereognosis and graphesthesia (p. 146) are processed in the *parietal somatosensory association cortex* (areas 5, 7, 40).

*Wernicke's area* (area 22) forms words. Words formed in Wernicke's area are linked to limbic system memories, which give meaning to the words. When Wernicke's area is linked to 1) Heschl's gyrus, and 2) other unimodal association areas (via the angular gyrus), we can a) comprehend what we hear and b) name what we see, taste, smell or touch.

### **Unimodal Association Areas: Motor Component**

The *motor component of the unimodal association areas* is the *premotor cortex* (area 6). It contains motor programs, analogous to computer software, that direct actions in the corticospinal and corticobulbar tracts. These motor programs give, for each action, automatic directions such as the muscles to be used, the strength of the contractions, the number of repetitions, and the rhythm. Simultaneously, subcortical structures *ensure that these directions are correct, akin to a traffic cop ensuring a safe flow of traffic at a busy intersection.*

*Broca's area* (areas 44 and 45), the inferolateral portion of the premotor cortex in the dominant hemisphere, contains the motor programs for the smooth articulation of speech by the mouth, lips, tongue, jaw, palate, cheeks, and uvula. It is also a *grammar center for functor words*, meaning small words like prepositions and articles. Examples: to, a, an, the.

### **Heteromodal Association Areas**

The *heteromodal association areas* include 1) *the inferior parietal lobules* (areas 39, 40 and 43) which in turn include the *supramarginal gyrus* (area 40) and the *angular gyrus* (area 39) and 2) the *prefrontal cortex* (areas 9–12). Heteromodal association areas synthesize information from many sources. The *inferior parietal lobules* handle cross-modal associations. For example, through the *angular gyrus*, visual perceptions (e.g., the image of a flower) from areas 18 and 19 are linked with words formed by Wernicke's area ("That's a flower.") If this information is to be acted upon (e.g., the person wants to say "That's a flower"), it is transmitted anteriorly 1) in the *arcuate fasciculus* within the *superior longitudinal fasciculus* to premotor areas 2) through the parahippocampal gyrus to the prefrontal cortex. These posterior-to-anterior transmissions yield a response.

The *prefrontal cortex* is heteromodal association cortex with executive functions such as planning, reasoning, deciding whether to act, and initiating action. To perform these functions, it synthesizes information from the posterior brain (e.g., the inferior parietal lobule) and—by way of the cingulate and parahippocampal gyri—from the limbic system, which generates and stores memories and generates mood states associated with memories and drives.

### **Limbic System: Relationship with the Hypothalamus**

*Limbic structures* connect to the *hypothalamus*, the head ganglion of the internal milieu and a major generator of drives and instincts which promote the survival of the self and the species. The hypothalamus helps to coordinate electrolyte balance, blood glucose, body temperature, metabolic rate, autonomic tone, sexual phases, circadian rhythms and immunoregulation. The drives generated by the hypothalamus are transmitted to the limbic system and then, via the parahippocampal and cingulate gyri, to the prefrontal cortex, which can act—or decide not to act—in response to drives.

### **The Limbic System: Relationship to the Parahippocampal Gyrus**

All limbic system structures also connect to the *parahippocampal gyrus* (areas 27, 28, 34 and 35) which receives sensory information and perceptions from unimodal association cortices and the thalamus. The parahippocampal gyrus contains the hippocampus and links to other structures associated with new learning and prior memories. Thus, the parahippocampal gyrus is a meeting point where perceptions of the outside world link with data from inside the organism.

### **Limbic System: Generating Moods**

Data from inside the organism and the outside world is linked with memories of prior experience to generate moods, which are then expressed by the cortex and the autonomic system.

### **Paralimbic Areas**

In addition to the parahippocampal gyrus, the *paralimbic areas* include the cingulate gyrus (areas 23,24,31,33), the retrosplenial area (26,29,30), and (D) the paraolfactory regions (32,35). The paralimbic areas link the brain's heteromodal cortices with the limbic system. Thus, paralimbic structures link 1) sensory experience from the outside world (via the inferior parietal lobule and the thalamus) with 2) limbic system memories and mood states associated with the sensory experience and 3) drives generated by hypothalamic-limbic connections. Consequently, paralimbic structures 1) give emotional coloring to current sensory experience, information upon which the prefrontal cortex can act and 2) give emotional coloring to thought and sensation, which cause limbic-hypothalamic generation of autonomic responses.

## **PROPOSITIONAL LANGUAGE: A FUNCTION OF THE DOMINANT HEMISPHERE**

### ***Anatomy of Propositional Language***

Key structures for propositional language (p. 131) include the dominant prefrontal cortex, Broca's area, Heschl's gyrus, Wernicke's area, the angular gyrus,

limbic system memory for the meaning of words (all discussed previously) the arcuate fasciculus within the superior longitudinal fasciculus (all discussed previously), and watershed areas deep to the lateral convexity of the cortex.

### **Dominant Prefrontal Cortex**

The *prefrontal cortex of the dominant hemisphere* generates spontaneous speech in response to thought, perception and mood.

### **Arcuate Fasciculus**

The *arcuate fasciculus within the superior longitudinal fasciculus* transfers information from the posterior to the anterior brain, particularly the transfer of words formed in Wernicke's area to Broca's area to be spoken.

### **Watershed Areas**

*Watershed areas* are poorly vascularized areas especially susceptible to hypotension, hypoperfusion and hypoxemia. Watershed areas include hemisphere portions of the frontal lobe deep to Broca's area and portions of the temporal lobe deep to Wernicke's area. The watershed area deep to Broca's area connects Broca's area to prefrontal cortex responsible for spontaneous speech. The watershed area deep to Wernicke's area connects Wernicke's area to limbic structures that "remember" meanings of words, analogous to a dictionary.

### ***Aphasias: Speech and Language Abnormalities***

*Aphasias* are speech or language disturbances due to dominant hemisphere disease. Stroke is the most common cause, but any lesion (e.g., Alzheimer's disease, traumatic injury) affecting the perisylvian region (areas around the Sylvian fissure in the hemispheric convexity) or related subcortical structures can cause aphasia.

### **Assessing Language**

When a patient has abnormal speech or language, assess 1) spontaneity, 2) fluency, 3) auditory comprehension, 4) verbal repetition, 5) whether speech is paraphasic (with serious misuse of words) and 6) whether neighborhood signs (signs produced by extension of the lesion to a nearby brain area) are present. Most of this requires only that you listen carefully while you interview the patient.

*Spontaneity* is whether the patient speaks on his or her own, when you ask no question, or elaborates on answers when you do pose a question. It is a prefrontal function. If an aphasic patient's spontaneity is normal, the lesion is in the posterior brain. If spontaneity is abnormal, the lesion is anterior.

*Fluency* is the ability to articulate words smoothly and clearly with no slurring or struggling. It is a premotor (Broca's area) function. If an aphasic person's speech is fluent, the lesion is posterior. If speech is nonfluent, the lesion is anterior.

*Auditory comprehension* means understanding what is said. If your patient answers questions relevantly, auditory comprehension is normal. If you cannot understand the patient's comments, test comprehension by giving directions like "Raise your left hand" and "Point to a picture." If an aphasic person's comprehension is normal, or mildly abnormal, the lesion must be anterior. If comprehension is moderately or severely abnormal, the lesion must be posterior.

*Verbal repetition* is repeating what is said. During most interviews, patients repeat one or more of your questions, revealing normal repetition. If a patient repeats no questions, ask the patient to "Repeat after me; Say 'No ifs ands or buts.' Say 'The boy went to the store.'" If repetition is normal, the lesion must be deep to or distant from the perisylvian region. Normal repetition is a mechanical action that requires an intact Wernicke's area, arcuate fasciculus and Broca's area. It does not require normal comprehension or spontaneity.

*Paraphasic speech* is characterized by severe misuse of words. *In-class paraphasias* are errors in which the mistake is on the topic that is being discussed. For example, you display a pen and say "Tell me what this is" and the patient replies "It's an ink pencil." *Out-of-class paraphasias* are errors in which the erroneously used words are irrelevant to the discussion. For example, you hold out the pen and ask "Tell me what this is," and the patient replies "That's the horse house." If the patient's speech is not paraphasic, the lesion is anterior. If speech is paraphasic, the patient has either 1) a posterior lesion, 2) an arcuate fasciculus lesion, 3) schizophrenia, or 4) a chronic drug induced psychosis.

*Neighborhood signs* occur when a lesion extends to a nearby anatomic site. For example, many Broca's aphasic patients have right upper extremity paresis or right lower facial weakness because the lesion spread into the motor strip (Brodmann area 4).

*Writing by Aphasic Patients:* Aphasic patients cannot compensate by writing, because their writing contains the same errors as their speech, and sometimes, the lesion causes additional problems like right hemiparesis or dyspraxic writing. *Dyspraxia* is inability to perform movements despite normal strength and sensation.

### **Broca's Aphasia**

*Broca's aphasia* is due to disease of Broca's area. As Broca's area is 1) that portion of the premotor cortex for smooth articulation of words and 2) a grammar center for functor words, Broca's aphasia presents with slow, labored, dysarthric, telegraphic speech. *Telegraphic* means leaving out small words, as in a telegram. The patient cannot repeat fluently what the examiner says. Comprehension is usually good, with an occasional mild deficit for complex sentences with many words like "Put the red circle on top of the blue square."

*Neighborhood signs* may include 1) contralateral upper extremity or lower facial weakness; 2) *depressed mood*; the more anterior the lesion, the greater the likelihood of depression; 3) *buccolingual dyspraxia* (inability to pantomime an action like blowing out an imaginary candle); and 4) *impaired conjugate gaze* when

the frontal eye field (area 8) is affected; if the lesion is destructive, gaze is ipsilateral; if irritative, gaze is contralateral.

### **Wernicke's Aphasia**

As Wernicke's area forms words, and as words take meaning when Wernicke's area links with limbic system memories for word meanings, Wernicke's area lesions cause the following: 1) impaired comprehension, 2) impaired repetition and 3) paraphasic speech. Spontaneity and fluency are normal. Neighborhood signs include occasional quadrantanopia in the contralateral visual field, and occasional hemisensory loss due to extension of the lesion into the sensory strip.

*Paraphasic errors* include the following:

1. *Nonsequiturs*: Nonsequiturs are statements irrelevant to the question asked or the topic discussed. This comes from 1) not understanding the question and 2) not recognizing one's comprehension problem. Example: "How are you doing today?" "Grass isn't always green."
2. *Private word usage*: Private word usage is insertion of irrelevant words. Example: "I enjoy sports house like tennis."
3. *Perseveration*: needless repetitive insertion of a previously spoken word, phrase or sentence. For example, "Needless insertion of a previous word. Like needlessly inserting a prior word. Like putting in words needlessly."
4. *Word approximations*: using words or phrases similar in meaning to the correct word, omitting the correct word. For example, you hold out a pen and say "What is this?" and the patient replies "That's an ink pencil." or "That's a writer."
5. *Neologistic paraphasias (neologisms)*: words with no meaning; words which are not in the dictionary or in common slang usage. For example, "I'm giving a talk on traskiths."
6. *Driveling speech*: Combinations of the above cause driveling speech (meaningless doubletalk) spoken spontaneously and fluently, with good balance of essential and nonessential words (normal syntax) and with normal melody, emotionality and gesturing (normal prosody). For example, in response to your question "What's the date?" the patient glibly and fluently responds "I won't do it if it's precise. There aren't too many features that give it to me. I take four, take three, that's very nice." If you didn't speak English, you would think the patient spoke normally!

### **Global Aphasia**

*Global aphasia* is caused by wide lesions in the perisylvian speech area, injuring both Broca's and Wernicke's areas. Global aphasia combines all aspects of both Broca's and Wernicke's aphasias. The patient has slow, labored, dysarthric, telegraphic and paraphasic speech with poor comprehension, inability to repeat,



and some neighborhood signs. Example: Doctor: "How are you feeling?" Patient (struggling to speak): "They . . . houses . . . take . . . city."

### **Conduction Aphasia**

*Conduction aphasia* is caused by a lesion in the parietal lobe or the arcuate fasciculus which, noted above, connects 1) Wernicke's area and the angular gyrus (posterior structures) to 2) Broca's area and the premotor cortex (anterior structures). Other language-related structures are intact. Thus, spontaneity, prosody, comprehension and reading comprehension are intact, and fluency is good except for occasional stoppage due to a word-finding problem.

However, words correctly "thought" and comprehended cannot be transmitted anteriorly for articulation by Broca's area or for writing. Thus, 1) the patient's speech resembles Wernicke's aphasic speech with fluently spoken jargon, 2) the patient cannot read aloud and cannot repeat what you say, and 3) the patient cannot compensate by writing. This is very troubling to the patient, who understands the problem but cannot overcome it.

### **Transcortical (Extrasylvian) Aphasias**

*Transcortical aphasias* are caused by 1) lesions in the prefrontal cortex or in watershed areas with 2) normal repetition. Repetition is normal because Heschl's gyrus, Wernicke's area, the arcuate fasciculus and Broca's areas are intact.

In *transcortical motor aphasia*, there is a lesion of the prefrontal cortex or of the watershed area between 1) Broca's area and 2) the prefrontal cortex. Because the posterior brain is intact, the patient comprehends what the examiner says. Because the perisylvian region in the cortical convexity is intact, repetition is normal. However, the patient cannot speak spontaneously. Asked an open-ended question like "Tell me what was the problem that brought you to the hospital," the patient may 1) remain silent, 2) repeat the question, or 3) by habit based on experience, say "I don't know."

In *transcortical sensory aphasia*, a watershed lesion isolates Wernicke's area from limbic system memories for correct word meanings. Consequently, the patient cannot comprehend your speech and speaks paraphasically. However, since the perisylvian region is intact, and since spontaneity and fluency are intact, the patient can repeat what the examiner says, and often does so spontaneously.

In *isolation of the speech area*, a wide lesion damages the entire watershed area deep to the perisylvian region. Heschl's gyrus, Wernicke's area, the arcuate fasciculus and Broca's area stay intact. Consequently, the patient lacks spontaneity and doesn't comprehend others' speech. The only intact language function is repetition. The patient's only speech is repeating what he hears (*echolalia*).

### **Aphasias Due to Subcortical Disease**

Subcortical structures contribute to language. The anterior and posterior cortices transmit efferents to the corpus striatum (caudate, putamen and nucleus accumbens), which transmits to the globus pallidus and substantia nigra, which send efferents to the thalamus, which sends messages to the anterior cortex (Figure 1).

According to *Crosson-Wallisich lexical decision-making theory*, the corpus striatum selects among alternative thoughts generated in the cortex, and sets in motion the final spoken words via thalamic stimulation of the anterior cortex. *The basal ganglia can be viewed as a traffic cop at the brain's crossroads, letting some words and movements pass, and stopping others.* Subcortical disease can cause aphasia.

*Thalamic Aphasia:* Disease of the dominant thalamus sometimes causes aphasia with fluent paraphasic speech, and normal comprehension and repetition. Of all the aphasias, this one most closely resembles the language disorder (termed formal thought disorder) of persons with schizophrenia and chronic drug-induced psychosis.

*Aphasia Due to Basal Ganglia Disease:* Disease of the basal ganglia of the dominant hemisphere sometimes causes aphasia. In general, if *anterior basal ganglia structures* are damaged, the aphasia is nonfluent, and if *posterior structures* are damaged, the aphasia is fluent.

### **The Cerebellum in Language and Speech**

The *cerebellar neocortex* is better developed in humans than in other primates. The cerebellum is part of the motor system, and hence part of the anterior brain system. It works in concert with the prefrontal cortex, connected by brainstem motor pathways and the thalamus. Its role in speech and language (the nondominant cerebellum is involved) includes word finding for long, complex words and action verbs and coordination of speech and thought. Lesions can cause dysarthria, mild verbal fluency problems, word finding problems for complex words or action verbs, or scanning speech. In scanning speech, each syllable has an odd inflection. For example: "I DID not GIVE any TOYS TO my son FOR CHRISTmas."

### **PROSODY: A FUNCTION OF THE NONDOMINANT HEMISPHERE**

*Prosody, a nondominant hemisphere function,* is the emotionality, inflection, melody, emphasis and gesturing that accompany propositional language. Prosodic abnormalities are termed *dysprosodias*. Dysprosodias are common, perhaps as common as aphasias. However, as dysprosodias were first "discovered" during World War II, and first described systematically in 1981, few physicians know about prosody.

Prosody is served by areas of the nondominant hemisphere that are homologous to areas in the dominant hemisphere that serve propositional language. Influenced in part by the limbic system and paralimbic cortex, the nondominant prefrontal cortex spontaneously generates prosody. The frontal premotor areas homologous to Broca's area contain "motor programs" for the competent production of prosody (expressive prosody). Comprehension of the prosody of others is handled by the areas homologous to Wernicke's area and adjacent parietal structures.

## ASSESSING ANTERIOR BRAIN SYSTEMS

### *Alertness and Attention*

*Alertness and attention* require an intact brainstem reticular activating system, thalamus and prefrontal cortex. The thalamus activates the cerebral cortex. Alertness and attention can be tested with the *letter cancellation test (A test)* which works as follows: Tell the patient “I will read you 52 letters. Every time I say the letter ‘A,’ tap the table with this pencil.” If the patient makes five or more errors (either of omission or commission), attention is severely impaired, which may invalidate many further tests of cognitive function.

### *Prefrontal-Basal Ganglia-Thalamus-Frontal Circuits*

There are several circuits in which the prefrontal cortex sends efferents to the corpus striatum, which sends efferents to the globus pallidus and substantia nigra, which send efferents to the thalamus, which completes the loop by sending efferents to the prefrontal cortex (Fig. 1). Two of these circuits are the medial orbitofrontal circuit and the dorsolateral prefrontal circuit.

#### **Medial Orbitofrontal Circuit**

The *medial orbitofrontal circuit* begins with prefrontal area 10 and projects to the ventromedial caudate nucleus, which sends efferents to the globus pallidus and substantia nigra. The globus pallidus and substantia nigra send efferents to the dorsomedial and ventral anterior nuclei of the thalamus, which send feedback to area 10. Lesions in this circuit cause the orbitofrontal syndrome with 1) a silly, mood termed *Witzelsucht*, 2) *irritability*, 3) echopraxia or echolalia and 4) utilization behaviors. *Echopraxia* is copying the examiner’s movements despite instructions to the contrary. For example, you say “When I touch my nose, you touch your chin.” Then you touch your nose and the patient touches his nose. In a *utilization behavior*, the patient repeatedly and needlessly responds to an object’s function. For example, a patient repeatedly flushes the toilet. Another frequently pulls a fire alarm.

#### **Dorsolateral Prefrontal Circuit**

The *dorsolateral prefrontal circuit* begins with prefrontal areas 9 and 10 and projects to the head of the caudate nucleus. The caudate head sends efferents to the globus pallidus and substantia nigra. The globus pallidus sends efferents to the ventral anterior and medial dorsal thalamic nuclei, which send feedback to prefrontal areas 9 and 10. This circuit has executive functions like organizing one’s work and generating new ideas. Patients with abnormalities of this circuit appear apathetic, do not generate new ideas, and have trouble presenting a well organized, sequential history.

## **Assessing Frontal Lobe Functions**

Assessing frontal function includes testing 1) working memory, 2) motor functions and 3) executive functions.

### **Working Memory and Concentration**

The prefrontal cortex “holds” new sensory information presented to it for perhaps 30 seconds and permits decisions about what to do next (*working memory*). *Working memory* includes immediate recall and concentration. Ask the patient to repeat three unrelated words after you state the three words. For example: “Repeat after me, ‘book, house, candle.’ ” One test of *concentration* is to have the patient spell a five letter word (e.g., earth or world) forwards (tests spelling ability) and then backwards (tests concentration), or to subtract seven serially from 100.

### **Frontal Motor Functions**

Noted above, the anterior brain handles motor functions. In addition to observing gait, listening to speech and performing a general neurologic examination, you may assess for 1) motor persistence, 2) motor sequencing and 3) echopraxia. To *assess motor persistence*, have the patient extend his or her arms parallel to the floor and hold the position for 20 seconds. To *assess motor sequencing*, demonstrate and ask the patient to repeat the following sequence: “Touch the tabletop with the side of your fist, then with the side of your outstretched hand, then with the palm of your hand.” To *assess for echopraxia* (copying actions based on sensory experience despite instruction to the contrary), tell the patient “When I touch my nose you touch your chin.” Then you touch your nose. If the patient then touches his nose, this is *echopraxia*. Echopraxia is a common finding in *catatonia* (p. 92), which has numerous causes including mood disorder and anterior brain system lesions.

### **Frontal Executive Functions**

Assessing *frontal executive functions* includes testing for 1) convergence/similarities, 2) divergence and 3) planning. To test *convergence*, have the patient state the *similarities* between “an orange and a grapefruit,” “a table and a chair,” and “an airplane and a bicycle.” To *assess divergence*, have the patient state multiple possible uses, conventional and unconventional, for ordinary objects. For example, “Tell me different ways you might use a spoon.” To *assess planning*, listen how the patient describes solving problems in his or her own life.

### **A Mnemonic Device for Remembering Frontal Functions**

To remember frontal functions use the mnemonic AWOSAJM: 1) alertness, 2) working memory, 3) orientation to time, place and person, 4) speech, 5) abstracting ability, 5) judgment and problem solving and 6) motor functions.

## ASSESSING POSTERIOR BRAIN SYSTEMS: THE PARIETAL LOBES

*Parietal lobe functions* include 1) sensations and perceptions of pain, temperature, touch, vibration, and position, 2) complex perceptions such as graphesthesia and stereognosis (discussed below), 3) recognition of familiar things like the left side of one's body or the left side of space; family and friends; one's illness; left from right; and directions; and 4) perceptions that guide movements, including dressing, construction, kinesthetic praxis and ideokinetic praxis.

### ***Pain, Temperature, Touch, Position and Vibration***

The *parietal sensory strip* (areas 3, 1, and 2) receives pain, temperature touch, position and vibration sensations. *Pain, temperature and touch sensations* enter the local nerve root of the spinal cord and immediately cross to the other side of the spinal cord. Areas 3, 1, and 2 also receive position and vibration sensation from the contralateral dorsal columns by way of the decussation of the medial lemniscus, the thalamus, and the posterior limb of the internal capsule.

### ***Graphesthesia***

*Graphesthesia* is the ability to recognize, with eyes closed, a number traced on the palm of one's hand. Tell the patient "I am going to draw numbers, one at a time, on the palm of your left hand with the cap of this pen. The numbers may be 2, 3, 4, 5, 7, or 8, not 6 or 9. (Why not 6 or 9?) They may be drawn upside down or right side up. Each time, tell me what the number is." Then "draw" numbers in the palm of the left hand and ask the patient to identify them. Follow this by drawing numbers in the palm of the right hand. Inability to recognize the numbers is *graphanesthesia*. Inability to recognize numbers "drawn" on the right palm (in right handers and most left handers) reflects disease of the left parietal lobe. Inability to recognize numbers drawn on the left palm is due to disease in the right parietal lobe or the corpus callosum. If the lesion is in the corpus callosum, information about the correctly perceived object cannot cross from the right (nondominant) parietal lobe to the left (dominant) parietal lobe for naming.

### ***Stereognosis***

*Stereognosis* is the ability to recognize, with eyes closed, an object placed in the palm of the hand. The objects (e.g., screw, button, coin, paper clip) should be noiseless and easily recognizable. For right handers and most left handers, test the left hand first. Inability to recognize the objects is *stereoagnosia*. If the patient cannot name the object in the left hand, the lesion is either in the right parietal lobe or the corpus callosum. If the patient cannot name the objects placed in the right hand, the lesion is likely in the left parietal lobe.

### ***Ideokinetic Praxis***

*Ideokinetic praxis* is the ability to *pantomime* actions on request. The dominant parietal lobe plays a crucial role in this function. For right handers and most left handers, test the left hand first. Say “Without speaking, using your left hand, show me how you would . . . turn an imaginary key in an imaginary lock . . . comb your hair as if you had a comb in your hand . . . hit an imaginary nail with an imaginary hammer.”

Inability to perform any of these tasks is called *ideokinetic dyspraxia*. A common dyspraxic error is using one’s hand as the imaginary object, such as combing one’s hair with one’s fingers, or hammering the imaginary nail with one’s fist. If the patient cannot perform the task with the left hand, ask him or her to perform the task with the right hand. If the patient cannot do the task with either hand, the lesion is in the dominant parietal lobe. If the patient cannot perform the task with the left hand, but can do so with the right hand, the lesion is in the corpus callosum.

### ***Kinesthetic Praxis***

*Kinesthetic praxis* is the ability to mimic movements of the examiner’s upper extremities, particularly hand movements. Tell the patient “Copy what I do. When I use my left hand, you use your left hand. When I use my right hand, you use your right hand.” Do not say aloud the type of gesture you are making. Then demonstrate 1) the “peace sign”; 2) a fist; 3) using scissors; 4) sawing wood; 5) pointing upward; 6) using a fork; or any variation.

Either hand may be tested first. Kinesthetic praxis is primarily a function of the contralateral parietal lobe. Errors are *kinesthetic dyspraxias*.

### ***Dressing***

Relatives, friends, caretakers, or the patient may report that the patient has trouble dressing. Observe the patient putting on a shirt or pants or a hospital gown. Inability to dress (*dressing dyspraxia*) may be due to 1) *body-clothing disorientation*, which is almost always due to nondominant parietal lobe disease, or 2) *unilateral neglect*. *Left spatial neglect* is due to right parietal disease in right handers and most left handers. *Right spatial neglect* is often due to left frontal lobe disease.

### ***Constructional Praxis***

*Constructional praxis* is the ability to copy the outline of a geometric shape like a Greek cross or a triangle without lifting one’s hand from the paper. This function is necessary for drawing, sketching and other artistic tasks. Test the patient’s preferred hand first. If the patient cannot perform the task with the preferred hand, ask him to perform the task with the nonpreferred hand. If the patient cannot

perform the task with either hand, the dysfunction, termed constructional dyspraxia, is due to nondominant parietal disease. If the patient cannot perform the task with the preferred hand but can do it with the nonpreferred hand, the dyspraxia is due to corpus callosal disease.

## CHAPTER 22. NEUROTRANSMITTERS

by Frederick S. Sierles, M.D. and Michael Alan Taylor, M.D.

Like the Simpson trials, neurotransmitters receive a ton of attention. Unlike the trials, (1) neurotransmitters are tested on USMLE, and (2) we are relatively ignorant about them.

One reason for our ignorance is that the brain has ten billion neurons and a hundred trillion synapses (we counted them personally). Neurotransmitters act at each synapse.

Despite our ignorance, or perhaps because of it, we neuroscientists make striking oversimplifications about the behavioral effects of neurotransmitters; for example, the oversimplifications that “Obsessive compulsive disorder is associated with too much serotonin” and “Schizophrenia is associated with too much dopamine.”

### NEUROPHYSIOLOGY AND NEUROTRANSMITTERS

Some neurophysiology of neurotransmitters is clear:

- 1) The brain sends messages by way of neurotransmitters acting at synapses. Neurotransmitters from presynaptic neurons excite or inhibit the next neurons in the message chain.
- 2) Excitation results from action potentials generated by sodium or calcium entering cells. Inhibition results from chloride entering cells or potassium leaving cells.
- 3) Ion channel excitation or inhibition is termed *gating*.
- 4) Whether excitation or inhibition occurs depends on which neurotransmitter is released from vesicles in the presynaptic membrane, which receptor responds to the neurotransmitter, and whether a drug or toxin is affecting the process.
- 5) Gamma amino butyric acid (GABA) is the main inhibitory transmitter in the brain and spinal cord. GABA receptors are located on, and can inhibit, many neurons that produce other neurotransmitters.
- 6) L-glutamate is the brain's main excitatory neurotransmitter. It is present in all brain neurons and acts at 60% of brain synapses.
- 7) Most receptors don't “gate” ion channels directly. They act on *G-proteins* which open or close nearby channels.
- 8) Neurotransmitters stop working when they are degraded intracellularly by enzymes (e.g., monoamine oxidase), taken back into the presynaptic neuron (reuptake), or blocked by substances (e.g., curare) that occupy receptor sites.

Some neurotransmitters are discussed below.



## NOREPINEPHRINE (NE)

- 1) Precursor: tyrosine.
- 2) Metabolite: MHPG (3-methoxy-4-hydroxyphenylglycol).
- 3) Main source: locus ceruleus
- 4) Distribution: neocortex and its subcortical structures (basal ganglia, thalamus), limbic system, hypothalamus, cerebellum and spinal cord.
- 5) Receptors:
  - A) Alpha 1 (acts on the cardiovascular system)
    1. Agonist: *Phenylephrine* elevates blood pressure and decongests the nose.
    2. Antagonist: *Prazosin* (Minipress): antihypertensive
  - B) Alpha 2 (decrease firing of NE neurons and decrease release of NE from presynaptic terminals). Alpha 2 NE receptors are on the cell bodies of presynaptic neurons.
    1. Agonist: *Clonidine* (Catapres): antihypertensive agent that reduces NE production and curtails locus ceruleus firing.
    2. Antagonist: *Yohimbine* (Dayto Himbin, Yocon, Yohimex) decreases adrenergic and increases cholinergic activity. Probably because of its cholinergic properties, it promotes penile erection in men with erectile dysfunction.
  - C) Beta 1: found in heart, kidney, fat cells and brain
  - D) Beta 2: lung, blood vessels, uterus and brain
    1. Agonist: *Isoproterenol* (Isuprel), a bronchodilator, is also used as a cardiac stimulant in heart block and cardiogenic shock. Not a psychotropic agent.
    2. Antagonist: *Propranolol* (Inderal) reduces myocardial contractility and is used to treat angina, arrhythmia, myocardial infarction (after the acute phase), hypertension, migraine, essential tremor, lithium-induced tremor, neuroleptic-induced akathisia, and impulsive aggressive behavior due to brain diseases like stroke. Some authors (not us) recommend it for "stage fright."
- 6) Reuptake blockade: The antidepressants *desipramine* (Norpramin), *nortriptyline* (Pamelor) and *protryptiline* (Vivactil) are partially specific NE reuptake inhibitors. *Venlafaxine* (Effexor) selectively inhibits reuptake of both NE and serotonin.
- 7) Enzymatic degradation: Monoamine oxidase inhibitors like *phenelzine* (Nardil) cause intracellular deamination of NE, other catecholamines, and indoleamines.

## DOPAMINE (DA)

- 1) Precursor: tyrosine
- 2) Metabolite: homovanilic acid

- 3) Sources and distribution:
  - a) Substantia nigra sends axons to the caudate, putamen (nigrostriatal system) and frontal cortex.
  - b) Ventral tegmentum sends axons to the limbic system (mesolimbic DA system) and neocortex (mesocortical DA system).
  - c) Hypothalamic DA2 neurons send axons to pituitary venous sinuses (tuberoinfundibular system), inhibiting prolactin production.
- 4) Receptors:
 

DA2 (the most important receptor)

  - a) Abnormalities in the nigrostriatal system are associated with movement disorders like tremor and choreoathetosis.
  - b) Abnormalities in the mesolimbic and mesocortical system are associated with schizophrenia and other psychoses.
  - d) Reduced DA2 activity in the tuberoinfundibular system is associated with hyperprolactinemia (amenorrhea, lactation, pseudopregnancy).
  - e) DA2 receptors are part of the brain's reward system, associated with novelty-seeking (excitement-seeking, risk-taking), including the exciting effects of cocaine.

DA1, DA3, DA4, DA5: Little is known about these receptors. Don't worry about them.
- 5) Agonists:
  1. *Bromocriptine* (Parlodel)
    - a) Suppresses lactation after stillbirth or for mothers not wanting to breastfeed
    - b) Treats hyperprolactinemia syndromes including those due to pituitary adenomas, acromegaly (alone or after pituitary irradiation)
    - c) Treats Parkinson's disease
    - d) Treats neuroleptic malignant syndrome and cocaine withdrawal.
  2. *Amantidine* (Symmetrel)
    - a) Treats Parkinson's disease
    - b) Treats neuroleptic-induced Parkinsonism
    - c) Treats neuroleptic malignant syndrome
    - d) Prevents influenza A infections.
  3. *Electroconvulsive therapy* releases multiple neurotransmitters including DA. Besides its usual uses in neuropsychiatry, it is also used for neuroleptic malignant syndrome and Parkinson's disease.
- 6) Antagonists
  - a. Most neuroleptics antagonize DA2 considerably. Clozapine (Clozaril) antagonizes DA2 modestly, probably explaining why it is less likely to cause tardive dyskinesia.
  - b. Several neuroleptics also antagonize DA1, DA3 and DA4.
- 7) Reuptake blockade
  - a. *Bupropion* (Wellbutrin) is a DA reuptake blocking antidepressant.
  - b. Cocaine and amphetamines block reuptake of DA, NE and 5-HT.

## SEROTONIN (5-HYDROXYTRYPTAMINE, 5-HT)

- 1) Precursor: Tryptophan
- 2) Metabolite: 5-hydroxy-indoleacetic acid (5-HIAA).
- 3) Source: Pontine median raphe nuclei
- 4) Distribution: Wide distribution similar to NE
- 5) Receptors: Many
- 6) Agonists:
  - a) The anxiolytic *Buspirone* (Buspar) may be agonistic.
  - b) *Sumatriptan* (Imitrex), an agonist, alleviates migraines.
  - c) *LSD*, *mescaline*, and other *hallucinogens* may be agonistic.
    - 5-HT-2A: The experimental drug *alpha-methyl-serotonin* is a 2A agonist, and *clozapine* and *olanzapine* are antagonists.
    - 5-HT-2B: Alpha-methyl-serotonin is an agonist.
    - 5-HT-2C: Alpha-methyl-serotonin is an agonist, and *clozapine* and *olanzapine* are antagonists.
- 7) Antagonists:
  - a) *clozapine* (Clozaril)
  - b) *risperidone* (Risperidol)
  - c) *olanzapine* (Zyprexa)
  - d) *ondansetron*, an antiemetic
- 8) Reuptake blockade: The selective serotonin reuptake blockers (SSRI) *fluoxetine* (Prozac), *fluvoxamine* (Luvox), *sertraline* (Zoloft), and *paroxetine* (Paxil), and the partially selective 5-HT reuptake blocking agent *clomipramine* (Anafranil) have antidepressant, anti-anxiety, anti-obsessional and anti-bulimic properties.
- 9) Behavioral associations:
  - a) The trait of harm avoidance (cautiousness, worrying about consequences) is associated with 5HT.
  - b) Low CSF 5-HIAA levels are associated with violence- and suicide-proneness, especially violent suicide (firearms, jumping). However, this fact is clinically useless, and nobody knows what causes the CSF 5-HIAA changes.

## ACETYLCHOLINE (ACh)

- 1) Precursors: choline and acetyl coenzyme A.
- 2) Metabolites: choline and acetic acid.
- 3) Source: basal forebrain (nucleus basalis of Meynert, diagonal band of Broca, medial septal nucleus), cranial nerve nuclei 3, 7, 9 and 10 (the vagus nerve), autonomic ganglia, and the corpus striatum.
- 4) Distribution:
  - a) In all autonomic ganglia, the presynaptic neuron produces ACh.
  - b) In the parasympathetic nervous system, ACh is the main postganglionic transmitter.

- c) In the sympathetic nervous system, most postganglionic neurons are adrenergic.
  - d) In the corpus striatum, ACh circuits are short and in equilibrium with DA neurons. Increased striatal DA activity inhibits ACh release. Increased ACh activity, acting through gamma amino butyric acid (an inhibitory neurotransmitter), inhibits DA firing.
  - e) At the neuromuscular junction (NMJ), ACh acts on the motor end plate.
- 5) Receptors: The brain and autonomic nervous system both contain muscarinic (M1-M4) and nicotinic ACh receptors. At the NMJ, receptors are nicotinic.
- a) Muscarinic receptors
    - 1. Agonists: *Bethanechol* (Urecholine), which promotes salivation, bladder contraction and erection, does not cross the blood-brain barrier.
    - 2. Antagonists: *Atropine* antagonizes all muscarinic receptors.
  - b) Nicotinic receptors: Antagonists of nicotinic receptors include *curare*, *botulinum toxin* and some *snake venoms*.
- 6) Reuptake: Choline is taken back into neurons, not ACh.
- 7) Behavioral and other clinical associations:
- a) Alzheimer's disease: degeneration in the nucleus basalis with diminished ACh production.
  - b) Myasthenia gravis: auto-immune inhibition of NMJ ACh transmission.

## **GAMMA AMINO BUTYRIC ACID (GABA)**

- 1) Precursor: glutamate. Synthesis of GABA from glutamate requires the enzyme glutamic acid decarboxylase and the coenzyme Vitamin B6.
- 2) Metabolite: glutamic acid.
- 3) Source: Unlike the cell bodies of NE, DA and 5-HT, located mainly in the "reticular core," cell bodies of GABA-producing neurons are located in many brain regions, including stellate cells of the neocortex; caudate, globus pallidus and substantia nigra; and cerebellar Purkinje cells. GABA coexists with other neurotransmitters in many cells. GABA receptors are located on, and can inhibit, many neurons that produce other neurotransmitters.
- 4) Distribution: projections to the globus pallidus, substantia nigra and thalamus. GABAergic neurons in the cerebellar Purkinje cells project to the brainstem.
- 5) Receptors: contain sites for *benzodiazepines* and *barbiturates*. Benzodiazepines attach to GABA receptors, which open chloride channels. Barbiturates and ethyl alcohol (EtOH) attach to GABA receptors and open chloride channels directly. Benzodiazepines, barbiturates and EtOH each increases the affinity of the GABA receptor for the other, so these drugs potentiate each other. The anticonvulsants valproic acid and vigabatrin act on GABA receptors.

## L-GLUTAMATE

- 1) Precursor: Glutamate
- 2) Metabolite: Glutamic acid
- 3) Source: Neocortical and hippocampal pyramidal cells; cerebellar granule cells; sensory nerves.
- 4) Receptors: Glutamate receptors are named for their agonists: kainate, AMPA (alpha-amino-3-hydroxy-5-methyl-4-phosphonobutyric acid), and NMDA (N-methyl-D-aspartate).
  - A. Activation of kainate and AMPA receptors causes excitation by opening sodium and calcium channels.
  - B. NMDA permits entry of sodium and calcium and is blocked by magnesium under resting conditions. To activate an NMDA receptor, glutamate must bind to it and magnesium must leave the ion channel.
    1. *Phencyclidine* (PCP) blocks NMDA channels, and this may account for its psychosis-generating effects.
    2. The antitussive *dextromethorphan* and its metabolite dextrophan block NMDA channels, but less potently than does PCP. In excess doses, dextromethorphan produces ataxia and neurobehavioral side effects, including a PCP-like psychosis.
- 5) Behavioral associations: In cardiogenic shock, stroke, Huntington's disease, psychomotor epilepsy (degeneration of limbic neurons due to persistent seizures) and amyotrophic lateral sclerosis, excessive local glutamate production may cause excessive depolarization, neuronal excitation and cell death. Perhaps NMDA receptor blockers could minimize neuronal death for persons at risk.

## ENDORPHINS

### *Enkephalins*

- 1) Precursors: proenkephalin, prodynorphin
- 2) Sources: caudate, putamen and globus pallidus, and in interneurons in dorsal horns of the spinal cord, the raphe nuclei of the pons, and in the periaqueductal gray matter.
- 3) Metabolites: amino acids
- 4) Distribution: frontal cortex, limbic system, thalamus, hypothalamus
- 5) Receptor functions:
  - a) Movement
  - b) Analgesia
  - c) Release of ventral tegmental DA neurons from inhibition. This may contribute to the DA-mediated reward system associated with opiate addiction.

## ***Beta-endorphin***

- 1) Precursor: proopiomelanocortin
- 2) Metabolite: amino acids and other peptides
- 2) Source: hypothalamus and anterior pituitary
- 3) Distribution: pituitary, limbic system
- 4) Functions:
  - a) production of corticotropins such as ACTH and luteinizing hormone releasing hormone
  - b) ACTH and beta endorphin are co-localized and cause stress-related analgesia. ACTH and beta endorphin are produced in excess in major depression with melancholia.
  - c) Beta endorphin is 48 times more potent than morphine as an analgesic.

## CHAPTER 23. PSYCHOPHARMACOLOGY

Diagnosis should precede treatment. Premature treatment masks symptoms and impedes diagnosis. Except for dire emergencies (e.g., the patient is combative) or court-ordered treatment, informed consent must be obtained from the patient or a guardian. Pharmacotherapy and psychotherapy can be given concurrently.

### NEUROLEPTICS

#### *Definitions and History*

Neuroleptics used to be termed *antipsychotics*. “*Neuroleptic*” means “grabs the neuron,” referring to frequent extrapyramidal side effects (EPS). The first neuroleptic, *chlorpromazine* (Thorazine), discovered in France in 1952, reduced symptoms so much that tens of thousands of persons were discharged from chronic mental hospitals in the U.S. in the 1960s and 1970s, a *deinstitutionalization revolution*. Unfortunately, many discharged persons became homeless.

#### *Indications*

Neuroleptics are the drug of choice for *schizophrenia*. They (particularly haloperidol) may be the drug of choice for *cocaine and amphetamine psychosis*. They are effective in *acute mania* if the patient is combative or extremely hyperactive, but they do not prevent recurrence of mania. They are useful for *LSD psychosis, delusional disorder, psychoses due to brain disorders like brain injury* (treating the primary cause, like neurosurgery for the brain injury, is the best treatment), *Huntington's disease*, and *Gilles de la Tourette syndrome*. They can be combined with antidepressants for major depression with psychotic and melancholic features, but ECT is much better for psychotic depression.

#### *Mechanisms of Action, Therapeutic Effects and Side Effects*

The main therapeutic action of neuroleptics is *dopamine 2 (DA2) blockade in the mesolimbic system*. Blocking of other DA receptors, like DA4, may also be therapeutic.

#### **Extrapyramidal, Endocrine, Anticholinergic, Alpha Adrenergic Blocking and Antihistaminic Side Effects**

Neuroleptics also cause DA2 blockade in 1) the *nigrostriatal system*, causing *extrapyramidal side effects (EPS)* like Parkinsonism, and 2) the *tuberoinfundibular system*, causing endocrine effects like breast engorgement or lactation.

Neuroleptics often produce *anticholinergic effects* like dry mouth; may produce *alpha adrenergic blockade* with postural hypotension; and may produce sedating *antihistaminic effects*.

Neuroleptics with high potency per milligram (*high potency neuroleptics*) are more apt to produce EPS, and less apt to produce the other side effects, than other neuroleptics. *Low potency neuroleptics* are more apt to cause anticholinergic, alpha blocking and antihistaminic effects, and less apt to cause EPS.

### ***Choosing a Neuroleptic***

Once the right dose is reached for a given patient, all neuroleptics—both high-potency and low-potency—have *equal antipsychotic efficacy*. For example, for Mr. Brown, 30 mg. of haloperidol daily and 400 mg. of clozapine daily have equal antipsychotic efficacy. Thus, neuroleptics are chosen in part because of their side effect profiles and the patient's past experience with the drug. Reportedly, the new neuroleptics *clozapine*, *risperidone* and *olanzapine* are unique because they reduce the *negative symptoms* (deficit symptoms) like reduced motivation and spontaneity) of schizophrenia in addition to reducing *positive symptoms* ("productive" symptoms like hallucinations and delusions). Equally likely, these three drugs do not *cause* negative symptoms, as do the other neuroleptics. For example, all other neuroleptics can cause *akinesia*, meaning reduced gestures, facial expressivity, spontaneity and motivation.

### ***Specific Neuroleptics***

#### **Chlorpromazine (Thorazine, Largactil)**

While its historical significance is great, *chlorpromazine* is rarely used today because of its postural hypotensive and sedative effects. It is low potency. Prescribe it only for patients who have successfully taken it for years and swear by it.

#### **Fluphenazine (Prolixin)**

*Fluphenazine* is popular because it has an oral form, an intramuscular (IM) form for emergency use, and a *long-acting IM decanoate depot form*, where one injection lasts 2–4 weeks, for outpatients who don't comply with taking daily neuroleptic tablets. It is high potency.

#### **Thioridazine (Mellaril)**

In the early 70s, *thioridazine* was the most popular neuroleptic because of its low frequency of EPS (low potency) and because it had some antidepressant effect. Nowadays, its use is limited because doses cannot exceed 600 mg./day, a dose above which can cause irreversible *retinitis pigmentosa*. Prescribe it only for patients who have taken it for years and "swear by it."

#### **Haloperidol (Haldol)**

*Haloperidol* is widely used because of its versatility and low frequency of cardiovascular side effects. It has an oral form, an IM form for emergency use, a long-acting decanoate form, and even an intravenous (IV) form which, although rarely used, may be effective for intractable psychoses. It is high potency.



**Clozapine (Clozaril)**

*Clozapine*, which blocks DA<sub>2</sub> and DA<sub>4</sub> receptors, is the only neuroleptic which does not cause (at least, no cases reported yet) tardive dyskinesia. A low potency drug, it has a low frequency of EPS. Of the neuroleptics, it has the highest incidence (1–2% of patients) of *agranulocytosis*, requiring weekly monitoring by the manufacturer of the patient's WBC count, which makes it very costly. Of the neuroleptics, it has the highest incidence of seizures. It only has an oral form, reducing its versatility. It should be prescribed only for patients who require a neuroleptic but have *tardive dyskinesia (TD)*, or for patients who require a neuroleptic but have not responded to good trials of other neuroleptics.

**Olanzapine (Zyprexa)**

*Olanzapine* resembles clozapine, but it causes agranulocytosis much less often.

**Risperidone (Risperidal)**

*Risperidone*, a new high potency drug, blocks DA<sub>2</sub>, DA<sub>4</sub> and some serotonin receptors. It does not cause negative symptoms. It only has an oral form.

**Pimozide (Orap)**

*Pimozide* may be the best neuroleptic for delusional disorder. Pimozide and haloperidol are the best neuroleptics for Gilles de la Tourette syndrome. Pimozide is probably the least effective neuroleptic for acute psychosis.

**Molindone (Moban)**

*Molindone's* uniqueness is that, of the neuroleptics, it is least apt to lower the seizure threshold. It has high potency.

**Thiothixene (Navane)**

Thiothixene has neither unique features nor unique risks. It has oral and IM (short-acting, not depot) forms and a high potency.

***Neuroleptic Side Effects***

Neuroleptics are not to be trifled with. They can cause permanent (e.g., tardive dyskinesia) or fatal (e.g., agranulocytosis, neuroleptic malignant syndrome, pharyngeal dystonia) side effects. Because of DA<sub>2</sub> blockade, EPS are the most common side effects.

One treatment of neuroleptic side effects is discontinuing the neuroleptic or reducing its dose. In the discussions below, assume for each case that—except for a patient with a potentially fatal side effect—continued use of the neuroleptic is required.

The EPS are Parkinsonism, rabbit syndrome, akinesia, dystonia, akathisia, neuroleptic malignant syndrome and tardive dyskinesia.

### **Parkinsonism**

The signs of *neuroleptic induced Parkinsonism* are the same as those of Parkinson's disease. As Parkinson's disease typically begins after age 50, Parkinsonism in a younger person on neuroleptics is usually a neuroleptic side effect. One exception: the street drug *MPTP* causes an incurable Parkinson syndrome. The treatments of neuroleptic-induced parkinsonism (again, assuming continued neuroleptic use is in order) include *amantidine* (an antiviral, dopaminergic antiparkinson agent), or an *anticholinergic antiparkinson agent* like *benztropine* (*Cogentin*) or *trihexphenidyl* (*Artane*). After 3–6 months, the antiparkinson agent should be discontinued; usually, the Parkinsonism does not recur when the agent is withdrawn.

### **Rabbit Syndrome**

*Rabbit syndrome* is a variant of Parkinsonism with rhythmic puckering of the mouth. Treatment is the same as for Parkinsonism.

### **Akinesia**

Resembling emotional blunting, akinesia is treated the same as is neuroleptic induced Parkinsonism, usually with good results.

### **Dystonia**

*Dystonia* is involuntary spasm of specific muscles. It can present as *opisthotonus* (total body extension, as in tetanus), *oculogyric crisis* (immobility of the eyes), *torticollis* (head turning due to neck spasm), *tongue spasm* (tongue immobility with reduced ability to talk and feeling like the tongue is swollen), and *pharyngeal spasm* (impaired swallowing with risk for aspiration). Prompt treatment with IV (preferable) or IM *benztropine* (*Cogentin*) or *diphenhydramine* (*Benadryl*) yields prompt relief with patient gratitude.

### **Akathisia**

*Akathisia* is an inner restlessness, experienced as “like I’m jumping out of my skin.” The patient may rock or pace. Akathisia differs from agitation in that 1) akathistic patients cannot say why they are restless, whereas most agitated persons can do so (e.g., “I have a big exam,” or “The aliens are going to kill me.”) and 2) almost all akathistic patients are on neuroleptics, whereas most agitated persons are not on neuroleptics. Treatment is a low dose of *propranolol* (*Inderal*). Anticholinergic agents or benzodiazepines are less effective.

### **Neuroleptic Malignant Syndrome**

*Neuroleptic malignant syndrome (NMS)* is due to DA blockade. Its main features are fever and rigidity. Delirium, catatonia and myoglobinuria (a poor-prognosis sign) may occur. It is most common in recently admitted hyperactive inpatients who drink insufficient fluids and are treated on overheated units. Untreated, it is often fatal. The neuroleptic should be stopped, fever should be reduced

by supportive measures (e.g., cool, moist blankets), and a dopaminergic agent (*bromocriptine*, amantidine or ECT) and *dantrolene* (*Dantrium*, a muscle relaxant) should be prescribed.

### **Tardive Dyskinesia**

*Tardive dyskinesia (TD)* results from taking neuroleptics for months or years. Its main feature is *choreoathetoid movements* of the mouth, trunk, pelvis and extremities. It can cause cognitive dysfunction or dysphagia. Unless diagnosed right after onset, it is incurable. Thus, patients receiving neuroleptics should 1) give informed consent documented in the chart and 2) be checked routinely for TD with a systematic physical screening test like the *Abnormal Involuntary Movements Scale (AIMS)*. TD has variants called *tardive akathisia* (incurable) and *tardive dystonia* (effectively treated as for acute dystonia).

### **Endocrine Side Effects**

There is an inverse relationship between DA levels and *prolactin* production. DA blockade by neuroleptics increases prolactin production, causing *lactation*, *breast engorgement* (men as well as women) and *amenorrhea*, the combination of which can mimic pregnancy (*pseudopregnancy*). These side effects are treated with amantidine (Symmetrel).

### **Alpha Adrenergic Blockade and Postural Hypotension**

Alpha adrenergic blockade with *postural hypotension* is most common with low potency neuroleptics. It tends to disappear over time, but it is distressing and causes falls. Patients should rise slowly from lying or sitting. They should wear support stockings and an abdominal binder. If this isn't enough, they should take high salt diets—if medically safe—or the salt-retaining steroid *fludrocortisone*.

### **Anticholinergic Effects: Dry Mouth, Blurred Vision, Delirium, GI Atony, Sexual Dysfunction**

Anticholinergic side effects are common with low potency neuroleptics. Their management is discussed on page 166.

### **Agranulocytosis**

About 20% of persons taking neuroleptics have mild, benign asymptomatic WBC reductions. *Agranulocytosis*, a life threatening fall in WBC, most common with clozapine, can occur with any neuroleptic. Anyone on a neuroleptic who develops fever, especially with sore throat, must have a "stat" WBC and differential count. If the WBC are less than 3000/cu. mm., or the polymorphs fewer than 1000/cu. mm., the neuroleptic must be discontinued, and the infection causing the fever must be treated promptly or the patient will die.

**Photosensitivity**

Tell patients receiving a neuroleptic for the first time that their first outdoor excursions should be brief. If brief exposure causes sunburn, the patient should use a sun block >15 SPF. If sunlight causes eye pain, the patient should wear sunglasses.

**LITHIUM**

*Lithium salts* are found in the earth's crust. The element lithium is in the periodic table in the same column as sodium, potassium and rubidium. It acts on phosphoinositide second messenger systems and inhibits cyclic AMP.

***Indications***

*Lithium* is the treatment of choice for manic episodes if the patient is not combative or excessively hyperactive. It is a fine antidepressant for depression in bipolars, and an excellent maintenance treatment to prevent of recurrence of mania or depression in bipolars. It is a decent treatment for major depressive disorder and a decent maintenance treatment in recurrent major depressive disorder. It is effective for anorexia nervosa and bulimia, used to treat abstinent alcoholics with mood disorders, and used in managing impulsive aggression.

***Strategies of Use***

Because lithium affects the heart, thyroid, bone marrow and kidneys, and because lithium-induced diabetes insipidus can be confused for diabetes mellitus because both present with polyuria and polydipsia, the following should be ordered before lithium treatment: baseline ECG, thyroid function, CBC, urinalysis and serum electrolytes and glucose. While neuroleptics and most antidepressants should be prescribed by increments, lithium should be started in a full therapeutic dose of 600–900 mg. twice daily with meals. Eventually it can be prescribed in one daily dose.

Blood levels are usually monitored. For acute treatment, the serum lithium level should be 1.0–1.5 meq./liter; the maintenance level should be 0.8–1.2.

***Side Effects***

Side effects at therapeutic blood levels include postural tremor, nausea, drowsiness, weight gain, nephrogenic diabetes insipidus, and hypothyroidism. With blood levels between 1.5–3.0, lithium toxicity presents with GI and CNS symptoms.

**Postural Tremor**

*Postural tremor* is accentuated by extending one's arms parallel to the floor. The tremor can be treated with small doses of a beta adrenergic blocker like propranolol.

**Nausea**

Sometimes, lithium irritates the stomach and causes nausea. Unlike most psychotropic agents, which are best prescribed at least an hour before or after meals, lithium is best prescribed with meals to counteract nausea.

**Drowsiness**

Mild drowsiness sometimes occurs early in lithium treatment. It is a good prognostic sign, and the patient should be told 1) that it will diminish spontaneously with time and 2) to bear with it because it shows that lithium is working.

**Weight Gain**

Weight gain with obesity is a common with all psychotropic medications. It is treated the same as for obesity of any cause.

**Nephrogenic Diabetes Insipidus**

Lithium's effect on cyclic AMP can cause nephrogenic diabetes insipidus or hypothyroidism. In *nephrogenic diabetes insipidus*, the kidney doesn't respond to antidiuretic hormone (vasopressin), yielding large volumes of dilute urine and causing polyuria and polydipsia. Urine specific gravity is  $< 1.005$  and the urine contains no sugar. Serum sodium is elevated, and fasting blood sugar is normal. Treatment is with *hydrochlorthiazide* (Hydrodiuril) or *chlorthiazide* (Diuril). As these cause lithium retention, serum lithium must be monitored.

**Hypothyroidism**

*Lithium-induced hypothyroidism* is like hypothyroidism from any cause: cold intolerance, bradycardia, anemia, myxedema, coarse voice, cognitive dysfunction, elevated TSH and reduced T3 and T4. It is treated with thyroxine (T4).

**Cardiac Repolarization Abnormalities**

Lithium can cause cardiac repolarization abnormalities, especially in persons with heart disease. Lithium should be prescribed cautiously—if at all—in persons with active heart disease.

**Cardiac Teratogenicity**

There is a debate about whether lithium causes cardiac teratogenicity. The early literature suggested a propensity for cardiac anomalies, particularly *Ebstein's tricuspid anomaly*, when lithium is prescribed in the first trimester. Recent research challenges this.

**Lithium Intoxication**

When the serum lithium exceeds 1.5, lithium toxicity occurs, characterized by GI symptoms like nausea, vomiting and diarrhea, and CNS symptoms like rigidity, coarse tremor, seizures, delirium, and coma. When blood level approaches or exceeds 3.0, hemodialysis is lifesaving.

### **Lithium and Sweating**

When people on lithium sweat, they lose more lithium than sodium in their sweat. Consequently, closer monitoring of general health, and of serum lithium and sodium, are required for persons whose lives entail much sweating.

### **Drug-Drug Interactions**

Lithium has some important drug-drug interactions.

### **Osmotic Diuretics**

When lithium is given with osmotic diuretics like theophylline (e.g., in a bipolar patient with COPD) or mannitol, considerable lithium is lost in the urine. Consequently, higher doses of lithium, and close monitoring of serum lithium, are required.

### **Nonsteroidal Anti-Inflammatory Agents**

Never prescribe lithium and indomethacin together. *Indomethacin* causes severe life threatening lithium retention. *Naproxen*, *phenylbutazone* and *ibuprofen* also cause lithium retention and can be prescribed only with great caution. *Aspirin*, *sulindac*, and *acetaminophen* can be prescribed safely for persons taking lithium.

### **Low Sodium Diets and Sodium-Losing Diuretics**

Lithium may be prescribed cautiously to patients on diets in which they can't use extra salt. It cannot be prescribed for persons on *low-sodium diets* or sodium losing diets, both of which cause lithium retention. It should not be prescribed for persons on *sodium losing diuretics*. This, too, causes lithium retention. In persons with lithium-induced nephrogenic diabetes insipidus, hydrochlorothiazide or chlorothiazide should be prescribed, but serum lithium must be watched carefully. *Furosemide* (Lasix) can be prescribed safely with lithium.

## **ANTIDEPRESSANTS**

### ***Mechanisms of Action***

Antidepressants work by *inhibiting reuptake of monoamines* into the axon (*monoamine reuptake blocking antidepressants, MARIs*) or *blocking the intracellular deamination of monoamines (monoamine oxidase inhibitors)*.

### **Non-Specific Monoamine Reuptake Inhibitors**

The earliest MARIs were tricyclics that were *non-specific*; that is, they blocked the uptake of multiple monoamines. These non-specific reuptake blockers, which included *amitriptyline* (Elavil) and *imipramine* (Tofranil), have more side effects than partially specific or specific reuptake blockers.

### **Partially Specific Monoamine Reuptake Inhibitors**

Partially specific MARIs inhibit reuptake of one monoamine primarily and other monoamines minimally. Examples include *desipramine* (Norpramin, Pertofrane) and *nortryptiline* (Pamelor), which are *partially specific norepinephrine reuptake blockers*, *clomipramine* (Anafranil), a *partially specific serotonin reuptake blocker*, and *bupropion* (Wellbutrin), a *partially specific dopamine reuptake blocker*.

### **Specific Monoamine Reuptake Inhibitors**

Specific monoamine reuptake blockers block reuptake of one transmitter exclusively. The best known are *selective serotonin reuptake inhibitors fluoxetine* (Prozac), *fluvoxamine* (Luvox), *paroxetine* (Paxil) and *sertraline* (Zoloft).

### **Monoamine Oxidase Inhibitors**

The monoamine oxidase inhibitors (MAOI) include *phenelzine* (Nardil) and *tranlycypromine* (Parnate).

### **Indications**

Antidepressants are indicated for mild major depression with melancholic features, major depression without melancholic features, dysthymic disorder, panic disorder, agoraphobia, social phobia unresponsive to cognitive-behavioral therapy, anorexia nervosa, bulimia, migraine, and chronic pain unresponsive to other treatments. They can be combined with neuroleptics for major depression with melancholic and psychotic features, but several other treatments, notably ECT, are preferable.

### **Strategies of Use**

#### **Nonselective or Partially Selective Monoamine Reuptake Inhibitors (MARIs)**

Low doses (e.g., 25 mg. BID of imipramine) should be started, and increased gradually (say, by 25 mg. every day) until a full therapeutic dose is reached. The most common error made by clinicians is underdosing. For example, many clinicians prescribe only 75–150 mg./day of imipramine, although the full therapeutic effect occurs at 200–300 mg./day.

#### **Selective Serotonin Reuptake Inhibitors (SSRI)**

The *SSRIs* are unique in that in many patients, the starting dose (e.g., fluoxetine 10 mg./day or sertraline 50 mg./day) can eliminate symptoms within 2–4 weeks without raising the dose. If significant improvement does not occur by two weeks, the dose should be increased.

### **Monoamine Oxidase Inhibitors (MAOI)**

Low doses (e.g., phenelzine 15 mg. BID) should be started, and the dose should be raised gradually until a therapeutic dose of 60–120 mg./day is reached.

The patient should be instructed (verbally and in writing) about what foods (high-tyramine-containing foods) and drugs (sympathomimetics, opioids, and barbiturates) must be avoided. High-tyramine foods include red wine (white wines and white Zinfandel are OK), beer, all cheeses except fresh cream cheese, liver, aged meats (fresh meat is OK), Italian broad beans, yeast extract for flavoring (cooked yeast is OK), and the flavorings Bovril and Marmite. Chocolate and caffeine in moderate quantities are OK. The patient should carry several *nifedipine* (Procardia, an alpha adrenergic blocking agent) capsules in case of accidentally ingesting a high tyramine food or a sympathomimetic drug or if symptoms of hypertension (e.g., occipital headache and neck pain, nausea, photophobia occur). An MAOI-induced *hypertensive crisis* should be treated with *phentolamine* (Regitine, an alpha adrenergic blocker) in the hospital.

*MAOIs and SSRIs must never be given together.* The combination causes a fatal serotonin syndrome, as can combining an MAOI and a synthetic narcotic like *meperidine* (Demerol). However, a person receiving a nonspecific MARI such as amitriptyline can then receive an MAOI in combination. The reverse is not true: Persons taking MAOIs should not subsequently receive a nonspecific MARI.

### **Managing Side Effects**

#### **Weight Gain and Obesity**

All psychotropic drugs, antidepressants included, cause *weight gain* and predispose to *obesity*. (An exception: fluoxetine sometimes reduces appetite and is used in weight reduction programs.) Managing weight gain due to antidepressants is the same as managing obesity in general (p. 40).

#### **Sexual Dysfunction**

Because depression often causes sexual dysfunction, take a sexual history before prescribing antidepressants to determine whether dysfunction is due to depression or the antidepressant. If the latter, ask the patient to bear with the side effect until the depression is relieved, and then add an “antidote” (e.g., cyproheptidine, neostigmine, bupropion) specific for the dysfunction.

#### **Precipitating Mania**

Thirty per cent of the time, if the patient is bipolar, treatment with antidepressants will cause mania. Avoid antidepressants in bipolars. If you must prescribe an antidepressant to a depressed bipolar patient, do so along with a mood stabilizer like lithium.

#### **Side Effects of Nonspecific Monoamine Reuptake Inhibitors**

*Anticholinergic side effects* are common with the nonspecific MARIs Each is treated as follows:



1. *Anticholinergic delirium* is treated with *physostigmine*.
2. *Dry mouth* is treated several ways:
  - a. Keeping sugarless fluids or sugarless hard candy available. Sugar predisposes to *oral candidiasis (thrush)*
  - b. *Moi-stir* or other mouth spray
  - c. *Pilocarpine* or *bethanechol* tablets
3. *Sexual dysfunction* is treated with *neostigmine* (Prostigmin) 1 hour prior to intercourse.
4. *GI atony with constipation* is treated with fiber or bulk laxatives and exercise.
5. *Postural hypotension* is treated the same as when due to alpha adrenergic blocking neuroleptics.
6. *Blurred vision* will usually subside spontaneously, but it is treated with *pilocarpine* eye drops.

### **Side Effects of Selective Serotonin Reuptake Inhibitors**

Common side effects of SSRI include insomnia, anxiety, decreased libido, anorexia, dry mouth and headache. A full serotonin syndrome may occur with too high a dose, when an MAOI is concurrently prescribed with an SSRI, or when a synthetic narcotic is concurrently prescribed with an MAOI.

1. Insomnia can be managed by prescribing the SSRI in the AM, or prescribing *cyproheptidine* (Periactin, a serotonin antagonist) at bedtime.
2. *Somnolence* can be managed by prescribing the SSRI at bedtime.
3. *Anxiety* is usually well tolerated and best managed by prescribing the SSRI in the AM.
4. *Sexual dysfunction* can be managed by prescribing *cyproheptidine* or *bupropion* one hour before sex.
5. The *serotonin syndrome* consists of restlessness, sweating, insomnia, nausea, diarrhea, abdominal cramps, myoclonus, and delirium. The cause should be found and treated. The syndrome can be managed by removing the cause, reducing the SSRI dose, or prescribing cyproheptidine.

### **Side Effects of Monoamine Oxidase Inhibitors**

1. *Anticholinergic side effects*: For complex reasons MAOIs have anticholinergic side effects like the nonspecific MAOIs. These are treated the same as anticholinergic effects of MAOIs.
2. *Sexual dysfunction*: With MAOIs, sexual dysfunction could be due to *anticholinergic effects* (treated with neostigmine) or *serotonergic effects* (treated with cyproheptidine). Which of the two effects is occurring depends on which side effects co-exist.
3. *Increased libido*: Patients often welcome this side effect.
4. *Amphetamine-like psychosis*: Treated by discontinuing the MAOI and prescribing haloperidol.

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