ALCOHOLISM IN A SHOT GLASS

SOMATIC ILLNESSES ASSOCIATED WITH ALCOHOL ABUSE

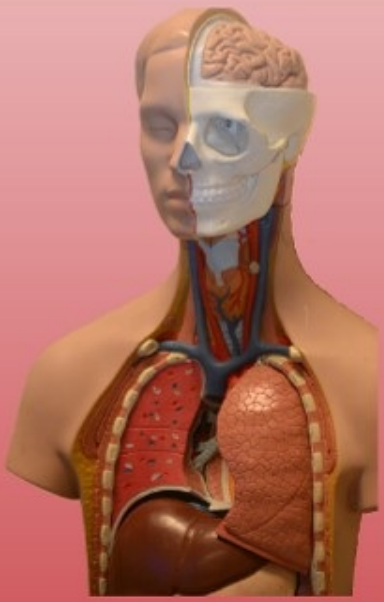

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Table of Contents

Somatic Illnesses Associated With Alcohol Abuse

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From *Alcoholism in a Shot Glass: What You Need to Know to Understand and Treat Alcohol Abuse* by Jerome D. Levin

Somatic Illnesses Associated With Alcohol Abuse

Alcohol abuse can damage any part of the body. The damage may be direct, the consequence of alcohol's effect on a particular cell, tissue, organ, or system, or it may be indirect, the consequence of alcohol's profound alteration of the body's internal chemical environment. The most common sites of damage are the nervous system, liver, and blood. The gastrointestinal system, heart, muscles, and reproductive organs also may be damaged.

Although the mechanisms by which alcohol destroys cells and damages organs are multiple, complex, and only partly understood, it is believed that alcohol's ability to penetrate membranes and disrupt membrane phenomena leads to cell death and is one of the most important pathways to somatic damage secondary to alcohol abuse.

THE NERVOUS SYSTEM

The structure of the nervous system is extremely complex. For our purpose a simple description will suffice. The nervous system consists of two main divisions: the *central nervous system* and the *peripheral nervous system*. The central nervous system consists of the brain (Figure 3.1) and the spinal cord. The peripheral nervous system consists of the nerves connecting the brain and spinal cord to the muscles, glands, and sense organs. It has two

parts: the nerves supplying the “voluntary” muscles and those supplying the “involuntary” muscles known as the *somatic* peripheral nervous system, and the glands of the internal organs, the *autonomic* nervous system.

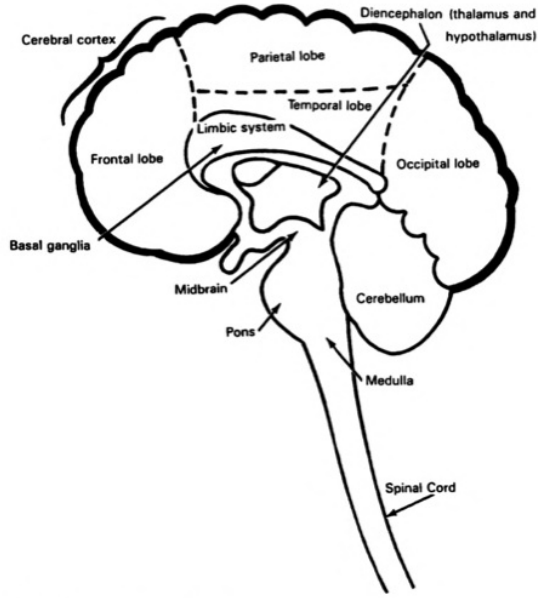

Figure 3.1 The human brain.

The brain and spinal cord are hollow structures filled with fluid, called *cerebral-spinal fluid*. The brain is organized in a hierarchical manner. The higher centers, known as the *cerebral cortex*, are responsible for voluntary muscle control, interpretation of sensory input, memory, language, abstract thought, and rational planning. It is divided into two *hemispheres* that are somewhat specialized in function. Each hemisphere is divided into four lobes:

frontal, parietal, temporal, and *occipital*. Folded beneath the cortex is the *limbic system*, an interconnected group of structures involved in the experience of emotion, among other things. Beneath the limbic system are the *basal ganglia*, clusters of nerve bodies that relay motor and sensory information to the cortex.

The next lower part of the brain is the *diencephalon*. It consists of two main structures: the *thalamus* and *hypothalamus*. The thalamus is primarily a sensory relay; the hypothalamus has many important regulatory functions and is involved in the expression of emotion. The *midbrain*, which is beneath the diencephalon, receives and processes auditory and visual sensory input. The *medulla*, or brain stem, lies between the midbrain and the spinal cord. It controls involuntary (vegetative) activities, such as respiration and heart rate. The *cerebellum*, or little brain, is behind the medulla. It coordinates fine motor activity. The *pons*, which is involved in sleep regulation, is in front of the medulla. The spinal cord begins where the central nervous system leaves the skull. All of these structures have more functions than have been noted here.

Alcohol affects *every* part of the nervous system. Alcohol abuse can damage any or all of them. Alcohol first depresses the cerebral cortex, resulting in disinhibition. This depression is indirect. The state of arousal—the level of activity—of the cortex is controlled by structures in the brain stem. It is believed that alcohol depresses the arousal system, which in turn

depresses the cortex. As dosage increases, alcohol directly depresses cortical activity. In higher doses it depresses the cerebellum, resulting in slurred speech and staggering gait. In very high doses alcohol can depress the respiratory centers of the medulla, resulting in death.

Functionally, the nervous system can be viewed as a mechanism for the reception, transmission, and interpretation of information from the external and internal environments. It then originates and transmits responses to that information. Alcohol abuse can derange all of these functions.

As discussed in chapter 1, alcohol's primary pharmacological action is the depression of synaptic transmission in the central nervous system. This is what makes a person "high." Although asymptomatic social drinking may have some enduring effects on the nervous system, they are usually assumed to be of little practical significance. (Some recent research has questioned this.) This is not necessarily the case when drinking turns into problem drinking or alcoholism. The possible deleterious neurological effects of prolonged heavy drinking are manifold. Some of them are the result of the addictive properties of alcohol, including the development of tolerance, requiring progressively larger amounts to "get the same high"; development of psychological dependence; development of physiological dependence; and withdrawal symptoms of varying degrees of severity. Other possible neurological sequelae—consequences—of chronic alcoholism include diffuse

damage to the brain, degeneration of specific structures in the brain, damage associated with the nutritional deficiencies often concomitant with alcoholism, and damage to the peripheral nervous system. Additionally, alcohol abuse impairs both cognitive functioning and normal sleep patterns. These impairments may occur in the absence of gross neurological changes. The neurological sequelae of alcoholism are sometimes transient and reversible; unfortunately, they are also sometimes permanent and irreversible. Let us examine each of the possible neurological complications of alcoholism, beginning with the phenomenon of tolerance.

Tolerance, Dependence, and Withdrawal

The nervous system accommodates, or becomes less sensitive to, the effects of alcohol. The mechanism of this accommodation is unknown. Its practical consequences may include an increase in alcohol consumption by the drinker as he or she seeks to reexperience his or her old high. Since alcohol induces (increases the production of) the MEOS enzymes, prolonged heavy drinking also results in the liver metabolizing alcohol more rapidly. These nervous system and metabolic effects are additive. They set up an addictive cycle in which the drinker must drink more to get the same effect, while the increased alcohol consumption builds further neuronal tolerance and induces more MEOS enzymes. The alcoholic drinker finds him or herself in a situation analogous to that of the laboratory rat who must run ever faster

on the treadmill to receive the same reward. The chronic heavy drinker may require three or four times the amount of alcohol initially needed to get equally high. (This is a modest multiplier compared to heroin and cocaine tolerance. Those addicted to those drugs may need 100 times the initial dose to get the same high.) There are limits, however, to this cyclical process. In advanced alcoholism, a point is reached when the integrity of the nervous system is compromised and tolerance to alcohol decreases. At that point, liver damage may also intervene, lowering the rate of metabolism of alcohol. Advanced chronic drinkers frequently report such a loss of tolerance. This loss of tolerance is pathognomonic (characteristic) of advanced alcoholism.

Long before becoming *physiologically dependent* on alcohol a drinker may come to rely on it to relax, assuage anxiety, self-medicate depression, speak in public, have sex, or function comfortably in a social situation. When a drinker cannot do something without alcohol, he or she has become *psychologically dependent* on it. To that extent, the drinker is "hooked." I have heard several patients say, without irony, "I'm so nervous I just can't drive my car without having a drink." Psychological dependence may or may not progress into physiological dependence; the reverse, however, is not true. Physiological dependence by definition entails psychological dependence, because the physiologically dependent drinker cannot function at all without alcohol. Psychological dependence on alcohol may be limited and of little practical significance, as in the case of the nervous public speaker who must

get a “little high” before a semiannual speaking engagement; or it may be all encompassing, as in the case of the alcoholic who cannot love, work, or play without alcohol. Highly specific psychological dependencies on alcohol do not necessarily progress, but like any chemical dependency they are indicative of a failure to deal with a conflict in a more adaptive way.

Physiological dependence is defined rather simply. If withdrawal symptoms accompany the cessation of drinking, the drinker is physiologically dependent. Alcohol is a central nervous system depressant. Chronic heavy alcohol use chronically depresses the central nervous system; it gets used to functioning in a depressed state. When the depressant is removed, there is a *rebound* effect, and the central nervous system becomes hyperactive. It is as if a coiled spring were suddenly released. Withdrawal symptoms range from anxiety and tremulousness to hallucinations and convulsions. Withdrawal from alcohol can be fatal. The physiologically dependent drinker must continue to drink to prevent the occurrence of withdrawal symptoms. The drinker’s position is the reverse of that of the man who banged his head against the wall because it felt so good when he stopped; the physiologically dependent drinker drinks because it feels so awful when he stops.

Withdrawal reactions are difficult to predict. Generally speaking, the longer the duration of the binge and the greater the quantity of absolute alcohol consumed, the greater the risk of a serious withdrawal reaction.

General health, nutritional status, age, and a history of previous difficulties during alcohol withdrawal are also of prognostic significance. Although mild withdrawal symptoms may occur even after a few days of steady drinking, serious withdrawal symptoms are not common until the drinker has consumed at least a pint of whiskey (10 drinks) or its equivalent per day for at least 10 days (Butz, 1982). It is good to bear in mind, however, that atypical reactions do occur.

Withdrawal symptoms begin several hours to several days after the last drink. They tend to peak between the second and fourth days, the third day being most commonly reported as the worst, and they generally abate, at least in their acute manifestations, within a week. Three stages of withdrawal are generally recognized by physicians. They are of progressively greater severity. Withdrawal syndromes do not, of course, come in neatly labeled packages, and a continuum of symptom severity is probably closer to reality than the conventional trichotomy.

The first stage, the mildest, is characterized by tremulousness, restlessness, appetite loss, insomnia, anxiety, and intense feelings of apprehension. Patients describe themselves as “ready to jump out of their skins.” Pulse and heartbeat are rapid. Most withdrawals do not progress beyond this stage, which is very uncomfortable but not in itself dangerous. The main danger is that the patient will drink to relieve the pain.

The second stage is marked by intensification of symptoms: the tremors become more severe; the patient feels that he or she is "shaking inside"; pulse, heart rate, respiration, and blood pressure continue to elevate; and anxiety and dread become more intense. During this stage the patient may suffer *alcoholic hallucinosis*. As one of my patients put it, "Doc, I had the audio-visuals." These hallucinations do not have any prognostic significance; they are not indicative of schizophrenia or other psychoses and should not be diagnosed as such. I have seen hospital records of patients who experienced a single episode of alcoholic hallucinosis many years ago but who still carried a diagnosis of schizophrenia. Patients experiencing alcoholic hallucinosis are usually oriented as to time, place, and person and are in fairly good contact with reality. That is, they realize that their hallucinations have no basis in external reality, even though they are powerless to stop them. They are, inevitably, extremely frightened. They require, and usually respond well to, reassurance.

The presence of hallucinations is a consequence of the hyperactivity of the nervous system; it is a physiological phenomenon. The content of the hallucinations is, at least in part, expressive of emotional and intrapsychic conflicts; it is a psychological phenomenon. Alcoholic hallucinations, like all hallucinations, can be analyzed in much the same manner as dreams. Although this is rarely possible, or therapeutically desirable, during detoxification, the clinician should attend to and try to and understand what

the hallucinations mean to the patient. Convulsive seizures of the grand mal (epileptic) type may also occur during alcohol withdrawal. They too are a rebound phenomenon and should not be misdiagnosed as epilepsy. They are dangerous mostly because of the risk of self-injury during a seizure. In rare instances, *status epilepticus*, a life-threatening condition of continuous seizures, develops. Seizures, when they occur, usually develop 48 hours after the last drink. Unfortunately, brain damage from prolonged excessive drinking *can* lead to epilepsy, but withdrawal seizures in and of themselves do not indicate the kind of structural changes in brain tissue that would render a person epileptic.

The third and most dangerous stage of withdrawal is known as *delirium tremens* (DTs). Most withdrawals do not progress to this stage. During DTs, the withdrawal symptoms become even worse. The alcoholic enters a state of abject terror. Hallucinations, often of small crawling animals or insects, become persecutory and are now tactile as well as visual and auditory. Psychomotor agitation becomes intense. Pulse becomes even more rapid, blood pressure continues to rise, and fever develops. Orientation and contact with reality are lost, and confusion and paranoia set in. Nausea, vomiting, explosive diarrhea, and drenching perspiration are also common. Sometimes the patient must be restrained to prevent self-injury or injury to others. Even with the best care, there is significant mortality during DTs.

I recall a patient who finally achieved stable sobriety after an excess of 20 attempts. He told me how de-humanized he felt when he was first restrained during DTs. But he went on to say, "You know it was funny, Doctor, after a while I looked forward to being put into a straitjacket on the 'flight deck' (AA slang for closed psychiatric ward). When the cops brought me in they all knew me. An aide would say, 'Shit, Jack, you back again? You better get in the jacket before you get too bad like you always do,' and I would feel kind of warm and safe and secure when they laced me in."

Some researchers consider an ordinary hangover to be a mild, self-limiting withdrawal syndrome. Others consider it to be a direct consequence of the toxicity of alcohol. It is both. The proportion of toxicity and withdrawal vary with the hangover.

The medical treatment of withdrawal from alcohol is called *detoxification*. It is accomplished in a variety of ways, but all involve the use of sedative drugs, which are *titrated* downward (systematically reduced) to zero over several days to a week. Currently, the most popular drug used for this purpose is the tranquilizer Librium. Other sedative-hypnotic drugs are also used. These drugs can themselves be addicting, and their use must be time-limited. Treatment of vitamin, mineral, and other nutritional deficiencies is vital. Anticonvulsive medication also may be used. Close supervision, nursing care, and emotional support are important during detoxification. Many

medical complications can occur. The technical aspects of detoxification from alcohol, such as choice and dosage of withdrawal drug, are medical issues and will not be discussed here.

Withdrawal from alcohol can be dangerous. Assessment of how and where detoxification should be carried out is a medical decision. It should be made by a physician experienced in and knowledgeable about alcoholism and the management of withdrawal. It should *never* be made by a nonmedical therapist or counselor. Referral for medical evaluation is always appropriate for the alcoholic patient, who has been using a highly toxic substance indiscriminately. In many cases detoxification can be carried out in an outpatient setting, with or without medication. If the patient has an intact social support system, the risk associated with outpatient detoxification is lessened. However, if the alcoholic is debilitated, if the intoxication has been prolonged, or if there is a history of seizures or DTs, detoxification should be accomplished in an inpatient setting. Hospitalization offers the additional advantage of providing external controls and an opportunity for educational interventions, if the patient is capable of hearing them. Detoxification is a medical procedure, not a treatment for alcoholism. Therefore, it is vital that counseling and alcohol education, which *are* treatments for alcoholism, be provided by the detoxification facility.

Wernicke's Syndrome and Korsakoff's Psychosis

As noted, alcohol's interaction with the human nervous system is complex. The slowing of synaptic transmission, first in inhibitory and then in excitatory synapses, is responsible for both the subjective inner experience, the high, and the objective behavioral effects of drinking. Furthermore, the neurons become acclimated to the presence of ethanol, possibly resulting in physiological dependence, and withdrawal symptoms when the chronic user attempts to stop drinking. The nervous system can become damaged by alcohol abuse in at least three additional ways: (1) from the toxic effects of the alcohol itself, (2) from the poisoning of brain cells by toxins circulating in the blood as a result of the failure of a diseased liver to metabolize them, and (3) as a consequene of the nutritional deficits concomitant with alcoholism. Wernicke's syndrome and Korsakoff's psychosis result from such a nutritional deficiency—explicitly, from a lack of *thiamine*. Thiamine is also denoted vitamin B_1.

Wernicke's syndrome is an acute condition initially characterized by confusion, delirium, and hyperactivity. The patient frequently is also suffering from *peripheral neuropathy*, a condition resulting from damage to the peripheral nerves by alcohol and/or nutritional deficits. (Peripheral neuropathy will be discussed more fully in the next section.) As Wernicke's syndrome develops, double vision (*diplopia*) ensues, and, if treatment is not instituted, the patient becomes quiet and sinks into a terminal stupor. There is strong evidence that Wernicke's syndrome is a nutritional deficiency

disease. If caught in time, it is rapidly and dramatically reversible by the intravenous (IV) administration of thiamine. Complete recovery is possible but not usual. Although Wernicke's syndrome will occur whenever there is severe thiamine deficiency from any cause, its occurrence is overwhelmingly associated with advanced alcoholism.

Thiamine serves several vital metabolic functions. It is required for the process by which neurons "bum" glucose in order to obtain energy, and it is needed to build *myelin*, a fatty substance that serves as an "insulator" for some axons. Although all cells require thiamine to help meet their energy requirements, brain cells require a great deal of energy to perform their functions and are therefore highly sensitive to oxygen deprivation or thiamine deficiency. The primary lesions in Wernicke's syndrome occur in the midbrain. However, other brain structures are damaged too.

Korsakoff's psychosis is a chronic condition believed to be a residual of Wernicke's syndrome. It may also result from an insidious subclinical process —that is, a process without manifest symptoms. In either case, Korsakoff s psychosis is the long-term result of brain damage caused by a thiamine deficiency not caught in time. Korsakoff patients have severe short-term memory deficits, which they attempt to conceal by filling in the gaps. This is called *confabulation* and is primarily an unconscious process, a defense against catastrophic anxiety. Korsakoff patients lack insight and suffer from

impaired judgment. There may also be general intellectual deterioration. Various sensory and motor impairments resulting from damage to the peripheral nerves also commonly accompany Korsakoff's psychosis. Some patients fully recover with thiamine therapy and abstinence from alcohol; partial recovery, however, is much more common, and some patients do not improve at all. Although thiamine deficiency is definitely etiological in Korsakoff's psychosis, some authorities believe that alcohol abuse per se is also involved in the etiology of this syndrome. In street parlance, Korsakoff's psychosis is called "wet brain." (The term wet brain may also refer to alcoholic dementia.)

Thiamine must be processed by the liver before the body can use it. Therefore, liver disease complicates the treatment of Wemicke-Korsakoff syndrome. The mineral magnesium is required for the liver to convert thiamine into usable form. Unfortunately, *hypomagnesemia* (magnesium deficiency) is also common in advanced alcoholism, and it too must be treated. Other nutritional deficiencies, particularly of niacin, are common as well. Extreme niacin deficiency causes *pellagra*, which is characterized by skin lesions ("wine sores"), psychiatric symptoms, and brain damage. Vitamin, mineral, and other nutritional deficits are so common in alcoholism that the nutritional status of all alcoholic patients, including functional middle-class ones, should be evaluated and appropriate remediation instituted.

Peripheral Neuropathy

Just as demyelinization of nerve tracts in the central nervous system (the brain and spinal cord) can occur as a result of nutritional deficiencies associated with advanced alcoholism, demyelinization can also occur in the peripheral nerves, which transmit sensory and motor information to and from the muscles and central nervous system. The loss of myelin is believed to be primarily the effect of thiamine deficiency, as thiamine is a coenzyme required for the synthesis of myelin. However, the process is probably more complicated, involving deficiencies of all B vitamins. Some authorities believe that the direct toxic effects of alcohol itself also play a role in the etiology of *peripheral neuropathy*. Loss of myelin slows the transmission of information through the nerves (peripheral nerves are axons or bundles of axons). This slowing results first in various sensory abnormalities, usually in the lower extremities, including burning, tingling, and prickling sensations; pain; and eventually numbness. If the drinking continues and the thiamine deficiency is not corrected, the degenerative process continues and the axon itself, rather than just its sheath, degenerates. At this point motor symptoms occur, ranging from gait disturbances to foot drop to paralysis. Since the distance from the feet to the spinal cord is greater than the distance from the hands to the spinal cord, symptoms in the feet and legs usually occur first. Later the hands and arms become involved. The final stage of degeneration involves the destruction of the nerve cell body. Muscle wasting may also occur. Advanced

cases may involve the autonomic as well as the somatic peripheral systems.

If damage is restricted to demyelinization, abstinence and nutritional repair lead to rapid recovery. If the axons have been damaged, recovery will be slow, taking months or even years. If the *neuron somata*, the cell bodies located in the anterior horns—horn-shaped gray matter on the belly side—of the spinal cord, have been destroyed, the damage will be permanent, and normal function will not be restored. Since alcoholic peripheral neuropathy is not uncommon, alcoholism counselors and therapists will encounter clients suffering from this disorder. *Alcoholic polyneuropathy*, as this condition is also known, is treated by abstinence from alcohol and massive dosages of B vitamins, initially intramuscularly. If drinking continues, further nerve degeneration is the rule. The clinician should remember that peripheral neuropathy can have many causes and that it is a rare side effect of the drug Antabuse.

Degenerative Diseases of the Brain

In addition to brain damage caused by avitaminosis (disease resulting from vitamin deficiency) associated with alcoholism, alcohol itself can damage nervous tissue, including brain tissue. How does alcohol damage the nervous system? Researchers are not quite sure, but several factors seem to be operative.

First, alcohol may induce a form of *autoimmune* response in the brain. There is evidence that it does so by releasing *sequestered* (chemically bound) *antigens*. Antigens are proteins whose normal function is to stimulate the production of antibodies, which attack invaders such as viruses. They are part of the body's defense (immune) system. Unfortunately, the cells' defense signals can get mixed up, and the defensive halfback, so to speak, tackles the defensive guards instead of the offensive ball carrier, causing an autoimmune response. It is thought that alcohol mixes up the defense signals, releasing antigens when there is no need for them. The antigens and/or the antibodies produced by them react with brain proteins. Such an autoimmune response destroys neurons.

Second, alcohol causes red blood cells to *agglutinate*—clump or sludge — and these agglutinated cells have trouble getting through the tiny blood vessels, the capillaries of the brain. The agglutinated cells block and sometimes break the capillaries of the brain. The result is a series of microstrokes. Cumulatively, they may result in considerable *necrosis* (death of brain tissue).

Third, alcohol, by its deleterious effects on neuronal membranes, disrupts protein synthesis in the brain, which if severe enough also results in the death of neurons. Since central nervous system neurons do not regenerate, they are lost forever. Some necrosis is an inevitable

accompaniment of normal aging. In people with good health this does not result in significant functional loss. Alcohol abuse can greatly accelerate the process, and the result is premature senility.

When these processes result in diffuse damage to the cerebral cortex, *alcoholic chronic brain syndrome* develops. This is a form of dementia and is known as *alcoholic dementia* or sometimes *alcoholic deterioration*. The syndrome is marked by confusion, memory loss, and general intellectual deterioration. Personality changes, including the development of emotional lability (instability) and paranoia, are common. The patient is moody and suspicious. The syndrome may progress to motor involvement, stupor, and death. As the disease progresses, the hollow spaces of the brain, the *ventricles*, enlarge and the cortex shrinks. There is considerable evidence that subclinical cortical shrinkage is common in alcoholics. Thus, the neurological requisites of recovery from alcoholism, the capacities to learn and to control impulses, may be compromised by alcoholism itself. If the alcoholic continues to drink, deterioration usually continues. If the alcoholic's dementia is mild, abstinence and rehabilitation may result in a social recovery. Many of these cases, however, require institutional care.

Other parts of the brain may also degenerate. When the damage is to the pons, the result is *central pontine myelinolysis*. This relatively rare condition is found in chronic alcoholics who are also severely malnourished. It is marked

by rapid deterioration and death. When the damage is primarily to the *corpus callosum*, the nerve fibers connecting the right and left cerebral hemispheres, the result is *Marchiafava-Bignami disease*. This condition is usually fatal. When the damage is to the cerebellum, the result is *cerebellar degeneration*. Symptoms include progressive unsteadiness of gait and slurred speech. If drinking continues, the condition worsens. With abstinence, however, improvement is usually possible. Nutritional factors may play a role in this condition. The optic nerve and other parts of the visual system also may be affected. When this happens the result is known as *alcoholic amblyopia*, a condition characterized by blurred vision and blind spots. Again, treatment consists of abstinence from alcohol and remediation of nutritional deficits. Some cases improve; others do not.

Alcohol's Effect on Learning

As noted, alcohol interferes with the synthesis of proteins in the neuron. It is believed that *messenger RNA* (ribonucleic acid) a cytoplasmic protein, plays a role in encoding new information. It is precisely the normal biochemical processes by which RNA is built and modified that are deranged by alcohol. The synthesis and processing of other cytoproteins are also negatively influenced by alcohol. When alcohol abuse is mild, the derangement is of function only, and no cell destruction takes place. Nevertheless, there are cognitive deficits; learning is impaired. There is

evidence that mice and other experimental animals require more trials to learn a maze following alcohol ingestion than they did before. This effect persists for some time after the mouse's last drink. Psychological testing has demonstrated that similar effects can, and frequently do, occur in humans. DeLuca and Wallace (1981) found that cognitive deficits in alcoholics are more common, more severe, and more enduring than previously believed. Although there is some evidence and considerable theoretical speculation that minimal brain dysfunction may be etiological in the development of some forms of alcoholism, the kinds of cognitive dysfunction researchers have recently found in alcoholics are almost certainly consequences, not antecedents, or causes, of alcoholism. This means that restitution of cognitive capacities will require a prolonged period of abstinence and will be less than complete for many alcoholics who do achieve stable sobriety. As they say in AA, "It takes five years to get your marbles back and the rest of your life to learn how to use them." Verbal abilities return more quickly and completely than do perceptual ones, and most recovering alcoholics with sobriety of any duration compensate so well for whatever residual damage they have that no functional impairment is noticed.

Nonetheless, these alcohol-related learning decrements and cognitive impairments have obvious and important practical significance in counseling active or recently sober alcoholics. It is prudent for the alcoholism counselor to assume that the active alcoholic, however socially intact, is suffering from

some degree of cognitive deficit. The counselor must take into account the fact that his or her client, no matter how well dressed and well educated, is not playing with a full deck. Neurological impairment and psychodynamic denial combine to interfere with the alcoholic patient's capacity to hear the counselor. Interventions, therefore, should be short, simple, clear, and redundant.

There is an interesting phenomenon called *state-dependent* learning. When experimental animals learn a task under the influence of a drug (such as ethanol), they will therefore perform the task better under the influence of the drug than without it. It is as if the total organismic state is part of the "learning set." This is also true of people, so an alcoholic who has "learned" to make love, write a poem, give a speech, or ride a bike under the influence of alcohol will find it difficult to do these things without it. He or she must learn them all over again when sober. When the recently sober person comes to counseling upset that he or she cannot function in some way, it is wise to inquire whether or not the particular skill in question had ever been exercised while the person was sober. If not, the counselor can explain that one can do anything sober that one can do while drinking but that it may take some time to be able to do so, since new learning is involved. There are exceptions. I knew a man who had been a highly skilled "second story man" while drunk, but who complained that he had never regained his abilities as a "cat burglar" in sobriety.

It is interesting to note that if a subject has learned a task under the influence of one sedative-hypnotic drug, such as alcohol, he or she will "remember" it just as well under the influence of another sedative- hypnotic drug, such as phenobarbital. The same is true of other classes of drugs.

Sleep Disturbances

Sleep disturbances are extremely common in both active alcoholism and recovery from alcoholism. Alcoholics, both active and recovering, frequently complain of an inability to fall asleep; or restless or tormented sleep; of disturbing or anxiety-ridden dreams; and, less frequently, of early morning awakening. Although psychological factors undoubtedly have a role in these disturbances, it is known that the pharmacological effect of alcohol itself is a powerful determinant of these abnormalities.

Sleep is neither a uniform state nor a passive happening. On the contrary, the sleep cycle is characterized by a regular, rather complex, sequence of discrete sleep states, and falling asleep is an active process mediated by the neurotransmitter *serotonin*. The sleep cycle has been studied by monitoring the electrical activity of the brain using an *electroencephalograph* (EEG). The EEG records composite pictures of the electrochemical activity of the cortex; these composite pictures are brain waves. A calm, resting normal young adult shows a brain wave pattern of 8 to

12 cycles per second while awake with his or her eyes closed. This is the *alpha* rhythm. As one becomes drowsy, the alpha rhythm is replaced by lower-voltage waves of 4 to 6 cycles per second. These are the *theta* waves of *stage 1* sleep. After a few minutes the EEG waves slow in frequency and increase in amplitude. This is *stage 2* sleep. It is marked by "sleep spindles" of 12 to 15 cycles per second and occasional bursts of high-voltage patterns. After several more minutes, slower (1 to 4 cycles) higher-voltage waves called *delta* waves begin to appear. This is *stage 3* sleep. When the slow waves predominate, *stage 4* sleep is reached. This slow-wave sleep appears to be a biological necessity, although it is not known why. The sleeper remains in stage 4 for about half an hour and then enters a stage of *rapid eye movement* (REM) sleep. REM sleep is associated with dreaming, which is also a biological necessity. The cycle then repeats itself. Slow-wave and REM sleep make up about 40% of a night's sleep.

Alcohol disturbs and disrupts the sleep cycle. It reduces the amount of slow-wave and REM sleep, and it distorts the normal brain wave patterns of stage 2 sleep. When a person is deprived of slow-wave and/or REM sleep, there is a "rebound effect" on subsequent nights, and the sleeper tries to make good the lost slow-wave and REM sleep. This happens when alcohol abuse ceases. At first REM rebound predominates, and the newly sober alcoholic complains of restless, tormented sleep. Complaints of insomnia and frequent awakening during the night are common. There is evidence that the

neurotransmitter serotonin is used by a part of the brain stem called the *raphe nuclei* to induce and maintain sleep. Alcohol is known to profoundly alter the metabolism of serotonin. Additionally, the metabolism of other neurotransmitters in the *biogenic amine family*, which are believed to mediate REM sleep, are also affected by alcohol. The metabolism of one of these, the neurotransmitter norepinephrine and of the enzyme *monoamine oxidase* (MAO), both of which play a vital role in sleep phenomena, are profoundly altered by alcohol.

The practical meaning of all of this is that there is a physiological basis for the wide-ranging sleep disturbances of alcoholism. Of equal importance is the fact that these abnormalities continue long into sobriety. AA takes a hard line on early-recovery sleeplessness, telling its members, "Nobody ever died of lack of sleep." The counselor can assure the sleepless early recovery patient that his or her sleep disturbance is a symptom of withdrawal and of neurological recovery and that it will correct itself in time, as long as the recovery process is not disrupted by drinking.

Subacute Alcohol Withdrawal Syndrome

Subacute alcohol withdrawal syndrome, also known as the *attenuated* or prolonged withdrawal syndrome, consists of the cognitive deficits, sleep disturbances, and concomitant emotional dysphoria that are characteristic of

at least the first year of sobriety. The syndrome does not include any unmistakable pathology (structural neurological damage) that may have resulted from alcoholism. One might view it as a shakedown period during which the nervous system is reestablishing its health. The emotional dysphoria that sometimes accompanies this healing process can serve as a "drink signal." If the drink signal is acted on, the recovery is aborted and active alcoholism resumes. The duration and severity of the subacute alcohol withdrawal syndrome vary widely. Usually, the heavier and more prolonged the drinking has been, the greater and more intense the attenuated withdrawal syndrome will be. Recent evidence suggests that prolonged withdrawal symptoms are far more common, more disabling, and longer lasting than previously suspected. Psychotherapeutic interventions during the first year of sobriety must take into account this syndrome and its possible effects on the recovering alcoholic's feelings and behavior. The counselor should tell the client that he or she is likely to have mood swings and otherwise inexplicable emotional discomfort during the first year of sobriety and that we will try to find an emotional or interpersonal reason for (or determinant of) that discomfort, but that often we will not find a reason because what you will be experiencing is neurochemical, not psychological, part of the recovery process during which your nervous system is readjusting itself.

Hepatic Encephalopathy

Liver disease is a common complication of alcoholism. If damage to the liver is severe, it may not be able to do its metabolic work. This can result in the presence of toxins in the bloodstream, which carries them to the brain. Although other toxins are probably involved, ammonia seems to be the main culprit. Therefore, *hepatic* or *portosystemic* encephalopathy (brain disease) is also known as *ammonia intoxication*. Its effects include confusion, drowsiness, and, in severe cases, unresponsiveness. The patient may develop a characteristic flapping tremor. Treatment includes addressing the underlying liver pathology and restriction of protein intake. Except in severe cases, ammonia intoxication is intermittent, often occurring when the patient eats too much protein. From a counseling viewpoint, it is important to be aware that ambulatory alcoholics with liver disease may be subject to period confusion from ammonia intoxication. They may appear to have been drinking, although they have not.

Alcoholic Hypoglycemia

As noted, the metabolism of alcohol profoundly alters the biochemical environment of the hepatocyte. Specifically, the supply of NAD is depleted as it is converted to NADH. NAD is necessary for the liver to convert stored carbohydrate *(glycogen)* into sugar *(glucose)*. If an alcoholic has not been taking in sufficient sugar during a prolonged drinking bout, his or her liver may not be able to maintain an adequate level of blood glucose. Since the

brain requires a constant supply of both oxygen and glucose to meet its energy needs, coma may ensue. If intravenous glucose is not administered in time, brain damage and even death may occur. Although brain damage from alcoholic *hypoglycemia* is not common, it is seen in clinical practice. Its clinical manifestations depend on the site and extent of the damage.

Trauma

Alcoholics are far more subject to head injury from automobile and other accidents, falls while drunk, and fights than the average person. Therefore, alcoholics presenting themselves for treatment of alcoholism may be suffering from slow intracranial bleeding resulting from a head injury. Such a *subdural hematoma* may cause brain damage and can be fatal. The confusion and other symptoms of such an injury are often mistaken for withdrawal symptoms or the effects of intoxication. The possibility of such an injury must always be considered in a detoxification setting.

Blackouts

Blackouts, or *alcoholic amnesias*, are memory losses during drinking episodes. In effect, the drinker has a temporary loss of the capacity for recent memory. Such loss can be partial ("grayouts") or total. Presumably, memory losses during drinking episodes are caused by alcohol's power to disrupt protein synthesis in the neuron. The encoding and storage of information—

the memory process itself—are theorized to require just such intraneuronal synthetic processes. Blackouts are also called *alcoholic palimpsests*. Palimpsest means *sand writing* in Greek. In a blackout, whatever is written in the memory bank is wiped out like writing in the sand because it cannot be converted into a more permanent *engram* (that is, it cannot be permanently stored in neural tissue).

Blackouts are a rather common symptom of problem drinking. Social drinkers also have been known to experience them occasionally. They are often casually dismissed by the drinker; many authorities, however (such as Jellinek, 1960) consider them to be *prodromal* (precursory) signs of alcoholism. This casual dismissal may or may not conceal profound apprehension on the part of the drinker. Psychologically, blackouts can be understood as failures of the synthetic powers of the ego; experientially, they are disruptions in the experience of the self. They are often a source of great anxiety and guilt for the drinker, who does not know what he or she may have done during the blank period. Unfortunately, such guilt and remorse may have a basis in reality, since auto accidents and serious crimes have been known to occur during blackouts. Additionally, the disruption in the sense of continuity of self and the experience of fragmentation are uncanny, causing great anxiety apart from concern over unknown acts. Frequent blackouts are pathognomonic of alcoholism.

THE LIVER

Alcoholic liver disease is a complex phenomenon. It was once believed to be a consequence of the poor nutrition so frequently associated with severe alcoholism. It is now know that this is not the case. Alcohol abuse itself can cause both functional and structural derangement of the liver, because the metabolism of alcohol greatly alters the biochemical milieu of the hepatocyte, the liver cell. This is not to say that poor nutrition does not exacerbate this pathogenic (disease-causing) process, or that nutritional therapy is not a vital part of the treatment of alcoholic liver disease.

Heavy drinking alters the inner environment of the hepatocyte in two fundamental ways: (1) it reduces the availability of NAD, replacing it with NADH, and (2) it induces enzymes in the MEOS, resulting in a thickening of the *endoplasmic reticulum*, which contains these enzymes. The first change can be conceptualized as a decrease in the concentration of available hydrogen ion acceptors or, in electrochemical terms, as a decrease in the "oxidizing" capacity of the cell. The second change, which can be observed under the microscope, alters the way the liver metabolizes many substances, including drugs, since these substances are also acted on by the enzymes of the MEOS. This is the basis of the alcoholic's cross-tolerance for barbiturates and other drugs.

The decreased availability of acceptors results in an increase in the

concentration of hydrogen ions in the liver cells which in turn increases the production of fat, while the lack of NAD inhibits the metabolism of that fat. The liver, in effect, now begins to use hydrogen as its fuel instead of fat, its normal fuel. This leads to a buildup of fat in the hepatocyte. There is also an accumulation of extracellular fat. This overall increase in hepatic fat results in the *first* stage of alcoholic liver disease—*alcoholic fatty liver*. The energy-producing organells of the hepatocyte, the mitochondria, are also damaged by this alteration in the normal oxidation of fatty acids. These intracellular changes may persist for months after the cessation of drinking. Accumulation of fat in the liver per se is initially asymptomatic, although laboratory tests may show abnormalities in the levels of liver enzymes. Later there is abdominal pain or discomfort. As the liver swells, it may grow large enough to press against the ribs and to be felt by the drinker. An enlarged liver can easily be palpated (that is, felt) during a medical examination.

Secondary consequences of the increased production and reduced oxidation of lipids (fat) by the liver are also possible. The mitochondria may try to bum off some of the excess lipids by utilizing abnormal biochemical pathways, which causes a class of organic chemicals known as *ketones* to be released into the bloodstream. This condition, which also occurs in untreated diabetes, is called *ketoacidosis* and can be dangerous. The level of fat in the bloodstream may also increase, mostly in the form of an excess of *triglycerides*. This condition is known as *hypertriglyceridemia* and increases

the risk of cardiovascular disease.

As discussed earlier, excess hydrogen also interferes with the synthesis of sugar from the liver's stores of glycogen, making the alcoholic subject to hypoglycemia, especially if he or she has not been eating.

Finally, the excess of hydrogen ions leads to a buildup of *lactic acid* in the blood, which decreases the excretion of *uric acid*. The resulting high blood level of uric acid is called *hyperuricemia*. High levels of lactic acid are associated with anxiety; high levels of uric acid are associated with *gout*.

Prolonged drinking thus alters the cofactor balance in the liver, which increases the concentration of hydrogen ions, leading to accumulation of fat. These changes greatly alter the overall chemistry of the liver and indeed the entire body. Fatty liver in itself is not a serious condition. If drinking ceases, the liver will repair itself and return to functional and structural normality.

Unfortunately, the disease process does not necessarily stop here. An active inflammatory process may ensue. This is the *second* stage of alcoholic liver disease and is called *alcoholic hepatitis*. Alcoholic hepatitis should be distinguished from hepatitis in general, which simply means liver inflammation. Liver inflammation can have many causes, the most common being viral infection. When alcoholic liver disease progresses to the stage of alcoholic hepatitis, cell destruction occurs, and the liver is, to some extent,

permanently damaged. It is not understood why fatty liver is universal in chronic alcoholics while only a minority develop alcoholic hepatitis. Duration and severity of drinking, genetically determined susceptibility, and associated malnutrition may play a role. At this stage the alcoholic experiences liver swelling, jaundice, hepatic pain, and fever. This is the active phase of alcoholic liver disease, the phase in which the damage is actually done. The disease process may be slow and insidious or rapid and dramatic. Alcoholic hepatitis is extremely serious. If its progress is not reversed, it can be fatal. Treatment consists of total abstinence from alcohol and vigorous supportive medical care.

The active inflammatory process of alcoholic hepatitis kills off a varying number of liver cells. These cells do not regenerate; instead they are replaced by fiber and scar tissue. This infiltration of the liver by scar tissue is called *alcoholic cirrhosis* or *Laeannec's cirrhosis*. In this *third* stage of alcoholic liver disease, the ability of the liver to perform its wide-ranging metabolic functions is impaired, and irreversible structural changes compromise the architecture of the liver. The liver now has a hard time doing its job. The chemistry of the body may be deranged in a variety of ways. Additionally, the fibrous nature of the cirrhotic liver interferes with the flow of blood and other fluids through it. Thus, chemical change and mechanical blockage occur. As the liver's capacity to perform its metabolic tasks becomes marginal, the level of toxins, especially ammonia, in the blood increases, causing hepatic

encephalopathy (ammonia intoxication). This condition often comes and goes, depending on the condition of the liver and the patient's diet. When hepatic encephalopathy is mild, the alcoholic may act as if he or she has been drinking, when in reality the liver is diseased. Mechanical blockage may result in fluid accumulation in the abdomen (*ascites*) or in backflow of the hepatoportal circulatory system, causing pressure on the veins of the esophagus (*esophageal varices*). This may result in *esophageal hemorrhage*, which is a medical emergency. If the hemorrhage is not stopped, the patient will die. *Edema*, which is fluid accumulation in other parts of the body, is another possible complication of cirrhosis.

Treatment of alcoholic cirrhosis consists of total abstinence from alcohol; a carefully controlled diet; and a variety of medical and possibly surgical interventions to reduce the ascites, alleviate pressure on the esophageal varices, and minimize or eliminate ammonia intoxication. If drinking continues, further episodes of alcoholic hepatitis usually occur, worsening the cirrhosis. Unabated, this process is fatal. If the alcoholic stops drinking, the outcome is variable. Some cases of cirrhosis can be managed medically. Others cannot, and death results either from liver failure or esophageal hemorrhage.

Alcoholics are also at increased risk for liver cancer. Unfortunately, this is the case even for recovered alcoholics.

Liver disease also has secondary consequences. Among its many functions, the liver regulates the supply of many substances essential for the body's metabolic economy. This can be called this *homeostatic* function of the liver. The liver accomplishes this regulation by storing nutrients, that have been derived from food and absorbed by the small intestine, that pass through it by way of the hepatoportal circulatory system, the liver's direct link with the intestinal tract. As discussed earlier, the liver helps maintain a relatively constant level of blood glucose by converting excess glucose into glycogen, storing the glycogen, and reconverting it into glucose as needed. The liver performs a similar regulatory function for many vitamins and minerals. It may also modify these substances so the body can use them. This is the case with thiamine. The liver also has an important role in regulating the supply and use of *magnesium, zinc*, and *folic acid*, all of which perform vital metabolic functions. Magnesium is involved in muscle function, including that of the heart muscle; zinc is a component of many enzymes; and folic acid is required for the formation of red blood cells. Cirrhotic liver disease compromises all of these liver functions. Additionally, the liver produces the hormone *erythropoietin*, which activates the production of red blood cells. This process, too, is interfered with by liver disease.

THE DIGESTIVE SYSTEM

The digestive system is essentially a hollow tube in which food is

digested and from which it is absorbed. The tube has various sections called the mouth, pharynx, esophagus, stomach, small intestine, and large intestine. Two outlying organs, the pancreas and liver, also have digestive functions.

Alcohol is an irritant, and constant irritation can damage tissue, including the lips, tongue, mouth, and pharynx, through which or over which alcohol passes. The chronic alcoholic is at increased risk of cancer of all these organs. The heavy smoking so frequently concomitant with alcoholism undoubtedly also plays a role in the etiology of these cancers. In general, the effects of heavy smoking and nutritional deficits confound the role of alcohol in the pathogenesis of many conditions. However, as already shown, whatever the role of these other factors, alcohol alone can do more than enough damage.

The zinc deficiency sometimes associated with chronic alcoholism may lessen the taste acuity of the tongue, further diminishing appetite and worsening an existing malnutrition. The esophageal varices that may have resulted from backwards pressure created by the blocked hepatoportal circulation that is secondary to cirrhosis may hemorrhage, or even be ruptured by violent vomiting. Vomiting may also cause blockage of the air passages and/or aspiration pneumonia—that is, pneumonia caused by foreign particles in the lungs. More than one intoxicated drinker has died in this way. Alcoholism is also associated with increased risk of esophageal

cancer.

The stomach produces hydrochloric acid, which plays an essential role in digestion. The stomach has a natural barrier that prevents the acid from digesting the stomach itself. Heavy consumption of alcohol strips away this protection. This can result in *gastritis* (inflammation of the stomach lining). Gastritis causes pain, nausea, and other gastric distress. It is a common complaint among chronic alcoholics. Chronic alcoholism contributes to the formation of peptic ulcers and is associated with a greater risk of stomach cancer.

Chyme, the product of gastric (stomach) digestion, passes into the small intestine. The small intestine is the entry site of ducts carrying digestive juices from the pancreas and liver. The damage that chronic alcohol abuse causes the small intestine is usually functional rather than structural. However, derangement of enzymatic activity and intestinal function can cause severe problems. Prolonged heavy drinking can result in malabsorption of minerals, folic acid, vitamin B_{12}, fat, and other substances. It is believed that alcohol does this by interfering with the active transport of these substances across the *intestinal mucosa*, or mucous membranes. This malabsorption may be the cause of the nutritional deficiencies sometimes found even in alcoholics who continue to eat reasonably well. Alcohol abuse also increases the motility of the intestine, causing severe diarrhea. The enzymes necessary

for the digestion of complex sugars are deficient in advanced alcoholics. Inability to digest these sugars also contributes to chronic diarrhea.

Alcohol does not significantly interfere with the production of *bile*, a fluid that the liver contributes to the digestive process to aid in the utilization of fats. Alcoholism can, however, damage the pancreas. The mechanism is obscure, but spasm of the duct that conducts pancreatic juice to the small intestine as well as direct toxicity are believed to be involved in this pathogenic process. Whatever the nature of the process, alcohol abuse may result in *pancreatitis*, or inflammation of the pancreas, which is a common complication of alcoholism. The pancreas has two functions. First, it products pancreatic juice, which contains enzymes necessary for digestion. Pancreatic juice is transported to the small intestine via the pancreatic duct. Second, the pancreas produces the hormone *insulin*, which regulates the utilization of sugar. Insulin is released directly into the bloodstream. Pancreatitis can compromise both of these functions. In acute pancreatitis the patient is nauseous, vomiting, and in great pain. He or she seldom continues drinking. *Acute pancreatitis* sometimes responds to supportive medical measures, although mortality is high. If the patient survives the acute attack, there is still the ominous possibility of underlying damage to the pancreas, that could lead to *chronic pancreatitis*. In this condition functional tissue is replaced with fiber, and the pancreas has difficulty performing either of its tasks. Exacerbation of acute symptoms and chronic pain may complicate this state

of affairs. Chronic pancreatitis can be insidious and occur in the absence of acute attacks. If the patient abstains from alcohol, chronic pancreatitis can usually be managed medically. However, if the patient continues to drink, the prognosis is poor.

THE BLOOD

Blood consists of a fluid (plasma) and three main types of blood cells, or *corpuscles'*, red blood cells (*erythrocytes*), white blood cells (*leukocytes*), and platelets (*thrombocytes*). Erythrocytes carry oxygen to the cells, leukocytes defend the body against foreign invaders, and thrombocytes are necessary for normal blood clotting. Dissolved in the plasma are a wide variety of biologically necessary substances, including nutrients, hormones, inorganic ions, and antibodies—proteins that are an important part of the body's immunological system. Alcohol abuse can derange the process by which erythrocytes, leukocytes, and thrombocytes mature and enter the bloodstream, resulting in *anemia*, lowered resistance to disease, and increased risk of hemorrhage, respectively. The most common, and hence the most important clinically, of these alcohol-related abnormalities of the blood is anemia. Let us examine how alcohol interferes with the normal production of red blood cells.

All types of blood corpuscles develop from a common ancestor, the

hemocytoblast, which is a cell in the bone marrow. Through a complex series of cell divisions and modifications, the various types of blood cells are formed. Since any rapidly dividing cell is particularly sensitive to toxins, it is not surprising that alcohol can interfere with this process of blood cell formation. Erythrocytes, for example, have a characteristic life cycle: They develop in and are released by the bone marrow into the bloodstream, where they flourish and live productive lives carrying oxygen to the tissues for approximately 100 days. Then they age and are ultimately destroyed by the spleen, which returns iron and vital amino acids to the body for storage and reuse. *Hemoglobin* is the protein in the red blood cells that combines with oxygen in the lungs and transports it throughout the body. Iron is a vital constituent of hemoglobin. In order for this cyclical process of red cell proliferation, maturation, activity, aging, and destruction to operate normally, thereby maintaining an optimal level of red cells, the bone marrow must be normal, the hormone erythropoietin must be present, and both iron and folic acid must be available in sufficient quantities.

Erythropoietin is produced by the liver and activated by the kidneys. Given the effects of alcohol on the basic chemistry of the liver, it is not surprising that normal production, storage, release, and regulation of erythropoietin can be radically altered by alcohol abuse. Inflammatory processes associated with alcohol-induced diseases of the liver and the pancreas suppress the production of erythropoietin. The result is a failure of

normal proliferation of red cells in the marrow.

Folic acid is required for the replication of DNA during cell division. Since the production of red (and other) blood cells involves several stages of cell division, this process is particularly sensitive to deficiencies of folic acid. Alcohol abuse may affect the level of available folic acid in several ways: (1) drinking without eating may result in inadequate folic acid intake, (2) alcohol interferes with the absorption of folic acid from the small intestine, and (3) alcohol blocks the metabolic pathways involved in the storage (again by the liver) and release of folic acid to meet the body's requirements. The result is ineffective red cell development, which if severe leads to the formation of giant abnormal red cells called *macrocytes*. Macrocytic anemia is associated with prolonged alcohol abuse and poor nutrition.

Finally, alcohol asserts a toxic effect on the internal metabolism of red cells, leading to iron encrustation of the mitochondria in the erythrocytes. These iron rings can be seen under the microscope, and the resulting condition is called *ring sideroblastic anemia*. It is associated with severe and prolonged alcohol abuse.

In addition to these problems in red cell proliferation and maturation, alcohol-related illnesses may accelerate the destruction of red cells, exacerbating the anemia. The treatment for all of these anemias is total

abstinence from alcohol and nutritional repair, particularly supplementation of folic acid. These anemias are stubborn, and recovery is usually quite slow.

The body's immunological system consists primarily of the white blood cells that fight bacterial infection and special serum proteins (antibodies), that defend against viral infection. The leukocytes develop from the common bone marrow ancestor, the hemocytoblast, through a process similar to that of red blood cell maturation. They, too, require normal levels of folic acid, which may not be present in alcoholics. The result is *leukopenia*— poverty of white blood cells, which makes the alcoholic suffering from this condition particularly vulnerable to infection. Given the effects of alcohol on protein synthesis, it is also likely that the antibody system is affected by alcohol abuse. In any case, the chronic alcoholic suffers heightened vulnerability to infections of all sorts and depleted resources to fight infection if one occurs. In an era of AIDS, any depression of the immune system is frightening and the alcoholic with heightened susceptibility to infection may escalate his or her alcohol consumption to dampen the fear of having AIDS when the real problem is the drinking itself.

Similarly, deficiency of available folic acid and possibly direct toxicity may result in *thrombocytopenia*, a deficiency in the number of blood platelets. This shortage slows clotting time and increases the risk of hemorrhage. Again, the treatment is abstinence from alcohol and folic acid supplementation.

THE HEART

The heart is basically a muscle that pumps blood through the body. Alcohol asserts a direct toxic effect on the heart. *Myocardial* (heart muscle) cells leak potassium, phosphate, and enzymes after alcohol ingestion, evidence that alcohol adversely affects the internal metabolism of the heart muscle cell. The result of repeated episodes of such transient toxicity may be *alcoholic cardiomyopathy*. In effect, the heart muscle is replaced with fat and fiber; it enlarges and becomes flabby. The result is a characteristic type of *congestive heart failure*. Alcoholic cardiomyopathy is a slow and insidious disease. By the time it is diagnosed there may be considerable damage. The patient may also develop irregularities in heartbeat *(arrhythmias)*. The prognosis depends on the extent of damage. Treatment consists of complete and permanent abstinence from alcohol and standard medical measures for the control of congestive heart failure. It is believed that heavy beer drinkers are most subject to alcoholic cardiomyopathy, possibly because preservatives added to beer can also damage heart muscle.

Alcohol abuse can also adversely affect the heart in various indirect ways. Nutritional deficiencies may damage it. Thiamine deficiency in particular can result in an inflammatory, degenerative condition known as *beriberi*, which affects the nerves and digestive system as well as the heart. Abnormally low levels of serum potassium, a condition known as

hypokalemia, commonly found in heavy drinkers, adds to the stress on the heart by making contraction of the myocardial cells more difficult. The associated low level of serum magnesium, *hypomagnesemia*, also adversely affects heart muscle cells. The *hyperlipidema* (abnormally high levels of fat in the blood) that is secondary to the altered chemistry of the hepatocytes that is associated with prolonged heavy drinking increases the risk of atherosclerotic cardiovascular disease, commonly known as hardening of the arteries. This damage to the circulatory system significantly increases the risk of heart disease in chronic alcoholics. Additionally, alcohol in sufficient quantities raises blood pressure. Therefore, alcohol abuse contributes to high blood pressure, which in turn increases the risk of heart attack and stroke. It is my experience that alcoholics who suffer from moderately high blood pressure often experience a dramatic return to normal blood pressure when they achieve stable sobriety. However, the heavy cigarette smoking which is so frequently associated with problem drinking, makes matters worse, since smoking also adversely affects heart function and blood pressure. Heavy smoking is a confounding variable in the association of alcoholism and heart disease.

The most extensive longitudinal study of problem drinking to date (Vaillant, 1983) found heart disease to be the most common medical complication of alcoholism. This was unexpected and may be a result of not following the drinkers for a long enough time for liver disease to develop.

There is no question, though, that prolonged heavy consumption of alcohol considerably increases the risk of cardiac disease and stroke.

THE SKELETAL MUSCLES

The skeletal muscles can be damaged by chronic alcohol abuse in much the same way as cardiac muscle. Although the exact mechanism by which alcohol damages the muscles is not known, electron microscopic examination of alcohol-damaged muscle cells reveals swelling of the mitochondria and fragmentation of the *myofilaments* (the organelle of the muscle cell that actually contracts), which are also findings in alcoholic cardiomyopathy.

Acute alcoholic myopathy is an acute syndrome of muscle pain, tenderness, and swelling following binge drinking. The muscles of the pelvic and shoulder girdles, as well as the adjacent arm and leg muscles, are most likely to be affected. The chest muscles may also be involved. Abstinence from alcohol usually leads to recovery within a month. However, if the patient resumes drinking, the symptoms are likely to recur.

Chronic alcoholic myopathy is a slow wasting away of these muscles, without pain or tenderness. It is characterized by progressive muscle weakness, and is a prolonged and insidious process. If the patient abstains from alcohol, slow recovery is the rule. Depending on the severity of the damage, recovery may or may not be complete. Both acute and chronic

myopathy are often associated with nutritional deficiencies, and these also must be remediated. The primary damage, however, is from the direct toxic effects of alcohol on the muscles, not from malnutrition. Alcoholic myopathy is often complicated by alcoholic polyneuropathy. The two tend to occur together.

ALCOHOL-INDUCED ANXIETY AND DEPRESSION

People often drink to alleviate depression and anxiety. This is true for the occasional relief drinker as well as the problem drinker and the alcoholic. As noted, alcohol is actually a pharmacological depressant that worsens the original depression. The early disinhibitory effects of alcohol are deceptive, since the initial euphoria is followed by a "down." Of course, pharmacological depression of the nervous system asserts an anesthetic effect, which is experienced as a cessation of pain, especially emotional pain. Drinkers do not therefore experience either their preimbibing depression or the pharmacological depression. Additionally, drinking may be an aggressive act that serves to externalize anger that had been turned against the self, thereby at least temporarily alleviating the depression. Unfortunately, this aggression is usually followed by guilt, which deepens the depression. Thus alcohol does not cure depression; it merely masks it. Inevitably, with a hangover the depression, worsened by the depressant effects of the alcohol itself and sometimes by guilt, returns in spades. Ultimately the drinker can no longer

find cessation of pain, let alone euphoria, in the glass. Often enough the futile search for the old effect continues indefinitely. If the drinker occasionally reexperiences relief from depression, learning theory teaches that this intermittent reinforcement will be an extremely powerful maintainer of the dysfunctional drinking. In short, alcohol is a terrible antidepressant. It causes what it purports to cure.

Alcohol also enjoys a reputation for tranquilizing, anti-anxiety effects. What is the scientific status of this popular conception? In small quantities (up to three drinks), alcohol's depression of synaptic transmission does assert a tranquilizing effect on the drinker. Subjectively, it dampens anxiety. The initial effect of alcohol in low dosage—inhibition of inhibitory neural circuits—often results in the drinker feeling carefree, exuberant, and free of mental and emotional distress. Alcohol is especially effective in assuaging anxiety caused by the superego—the anxiety caused by guilt over forbidden impulses and desires. In experimental animals, sedative-hypnotic drugs increase the frequency of behaviors previously extinguished by punishment.

Further, the anesthetic effect of alcohol contributes to a general reduction in anxiety. However, the subjective reports of the anxiety-reducing effects of alcohol indicate a much greater reduction than do experimental measures of the objective correlatives of anxiety (for instance, measures of galvanic skin resistance or of heart rate). In fact, some data indicate an

increase in the objective signs of anxiety. To make matters worse, the habitual drinker soon becomes acclimated to the anxiety-reducing effects of alcohol and requires progressively greater amounts of alcohol to achieve the same effect. Further, the anesthetizing effects of alcohol soon wear off, leaving the drinker with the same conflicts and, perhaps, with a physical and emotional hangover, with its concomitant guilt.

Alcohol in doses greater than two or three drinks actually causes anxiety. There is nothing subjective or psychological in this process itself; it is a purely biochemical phenomenon. Alcohol causes the release of adrenalin-like substances, the catecholamines, which mediate sympathetic nervous system arousal—the fight or flight reaction. Subjectively, sympathetic arousal (rapid heartbeat, rapid breathing, elevated blood pressure, and general body tension) is experienced as anxiety. Heavy and even moderate drinking causes a massive release of catecholamines (dopamine, norepinephrine, and epinephrine, or adrenalin), which are all neurotransmitters as well as in the case of the latter two, hormones released by the adrenal medulla, which cause sympathetic arousal. Catecholamines can be measured in urine following heavy drinking. Thus, the habitual drinker, who drinks to decrease anxiety, is actually drinking to reduce the anxiety caused by the alcohol already drunk to reduce anxiety. This cycle may be repeated ad infinitum. In this respect heavy drinkers drink because they drink. This is truly a case of a dog chasing its tail, a quintessential exercise in futility. The rebound effect of the alcohol-

depressed nervous system makes the next day's anxiety even worse.

Feelings of guilt for damage to self and others, fear of retaliation for aggressions committed during a binge, psychological conflict about drinking itself, and fear of the consequences to one's health, interpersonal relationships, and job performance all exacerbate the pharmacologically induced anxiety.

Alcoholism counseling has traditionally maintained that anxiety in alcoholics is a consequence and not a cause of their alcoholism. Since so many alcoholics maintain that they drink to relax, to reduce their anxiety, and since this so clearly makes no sense pharmacologically, their belief has been seen as self-deception and a rationalization for their drinking. More recent research (National Institute on Alcohol Abuse and Alcoholism, 1983) has shown that such conditions as purely synchronized alpha waves, which improves with alcohol consumption, characterize nonalcoholic male children of alcoholics. Subjectively, poor-quality alpha rhythms in EEGs of brain waves would be experienced as tension and anxiety, and their alcohol-induced synchronization would be experienced as reduction of tension and anxiety. It has been suggested that this would make drinking highly attractive to the male children of alcoholics and that this could lead to alcoholism. These EEG findings, which suggest that the popular belief that heavy drinkers drink to reduce tension and anxiety may have some truth to it, are important in our

attempts to understand why some people become alcoholics and some do not. Cumulatively, the evidence suggests that at least one group of alcoholics, those who have a certain kind of genetic susceptibility to alcoholism, may have higher than average levels of anxiety that is tranquilized by alcohol with more than average efficiency. None of this is certain, but it is increasingly seen as probable. However, as suggestive for etiology as these findings may be, they do not change the fact that heavy, let alone alcoholic, drinking causes anxiety. (These and related findings will be discussed in more detail in chapter 5).

In summary, if alcohol is a poor antidepressant, it is a not much better tranquilizer, except in small doses used infrequently. Drunk frequently and heavily, it is the antithesis of a tranquilizer—it is an anxiety- inducing drug.

THE REPRODUCTIVE ORGANS

According to Masters and Johnson (1970), the most common cause of sexual dysfunction in the United States is excessive drinking. Alcohol does help some people overcome sexual repression, and in low doses alcohol undoubtedly increases sexual pleasure for many people. However, objective measures clearly demonstrate that alcohol in more than minimal quantities decreases sexual performance. Measures of penile tumescence show an inverse, nearly linear, relationship between the quantity of alcohol ingested

and the firmness of erection. High and hard are antithetical conditions. An experiment in which a measuring device was inserted into the vaginas of volunteer female college students who viewed erotic films while drinking indicated a similar inverse relationship between quantity of alcohol ingested and vaginal pulse pressure (a measure of sexual arousal) (Wilson & Lawson, 1978). Although one cannot help but wonder how representative of the general population this sample was, there is considerable evidence that, although drinking may be sexually disinhibiting for many women as well as men, decreased arousal, lowered sensitivity, difficulty achieving orgasm, and less satisfying orgasm are linearly related to the quantity of alcohol ingested. Subjective reports do not necessarily agree with these findings, which are based on objective measures of female sexual performance. Thus, the evidence is that for both men and women the more alcohol consumed, the more difficult consummation of the sexual act becomes.

Impotence is an extremely common complaint of alcoholics. Alcohol asserts a direct toxic effect on the gonads (ovaries and testes). Liver disease can also contribute to sexual dysfunction. *Testicular atrophy* is not uncommon in chronic alcoholics. The testicles become smaller and softer than normal. Although not common, alcoholic peripheral neuropathy may affect the nerves serving the sexual organs. The result is impotence based on neurological impairment secondary to alcoholism. Additionally, feminization may result from abnormally high levels of blood estrogen found in some male alcoholics.

The feminized male alcoholic has enlarged breasts *(gynecomastia)*, loss of body hair, and thinned, softened skin. The liver metabolizes estrogen, and alcohol-related liver disease may compromise the ability of the liver to do this work. The result is a rise in the level of blood estrogen. Additionally, testosterone, perhaps already abnormally low as a result of testicular atrophy, may be metabolized too rapidly by the alcohol-induced enzymes in the MEOS. A clinical picture of cirrhosis of the liver, testicular atrophy, and enlargement of the breast is called *Silvestrini-Corda* syndrome. In women, prolonged excessive drinking may damage the ovaries, resulting in infertility.

Alcoholic impotence often remits spontaneously with abstinence; however, this is not always the case. The physical damage may be too great or the emotional inhibition too deep. Sometimes sobriety results in impotence in men who were potent while drinking. Alcohol-induced sexual dysfunction in both sexes is treated by Masters and Johnson-type behavioral techniques and/or insight-oriented psychotherapy to resolve intrapsychic conflict.

THE ENDOCRINES

The *endocrines* are glands that discharge their secretions, called hormones, directly into the bloodstream, where they are distributed throughout the body. Some hormones affect every cell and tissue, while others act primarily on specific receptors. We are already familiar with

ethanol's effect on the catecholamines—adrenaline and its relatives, some of which serve as hormones as well as neurotransmitters, on the sex hormones, particularly testosterone, and on the pancreas, which as an endocrine gland produces insulin. Alcohol's effect on some of the other endocrine glands is not as clearly established, but it is known that alcohol can damage the body's mechanism for dealing with stress, the *hypothalamus-pituitary-adrenal* (HPA) axis. The hypothalamus is a part of the brain involved in emotionality and the regulation of such behaviors as eating and drinking. It tells the pituitary, the master gland of the body, what and how much hormone to secrete. The hypothalamus does this by sending hormones to the pituitary. The pituitary, in turn, releases hormones that regulate the other endocrine glands, including the adrenal glands, which have several functions and secrete several hormones. Alcohol disrupts this delicate feedback system, resulting in the release of catecholamines from the adrenal medulla and the steroid *cortisol* from the adrenal cortex. Thus, alcohol produces abnormalities in the two major adrenal functions. By impairing the stress response system, alcohol can cause the very stress that it is consumed to reduce. Although these effects are usually functional rather than structural, abnormalities in the HPA axis can persist long into sobriety, and permanent damage is possible. It is also probable that alcohol affects other endocrine functions and feedback systems as well.

THE KIDNEYS

The damage alcohol does to the kidneys is believed to be secondary to (that is, to follow from) liver disease. However, alcoholics with or without liver disease suffer far more kidney damage than nonalcoholics if they contract urinary tract infections, and alcoholics are at increased risk for infections of all kinds.

DAMAGE TO THE FETUS

Women who drink alcohol during pregnancy risk damaging their unborn children. Ethanol readily crosses the placenta and affects the fetus. Children have been born with alcohol on their breath. Since rapidly dividing cells are especially vulnerable to the toxic effects of alcohol, it is not surprising that heavy alcohol consumption by a pregnant woman can damage her fetus. The damage appears to be due to an inhibition of growth, particularly of neural tissues. The result is *fetal alcohol syndrome*. Babies born with fetal alcohol syndrome are small, often have facial abnormalities, and may have varying degrees of brain damage. Sometimes the heart also displays abnormalities. These children have a variety of emotional and learning problems. Even with the best remediation and social rehabilitation, they remain gravely damaged individuals. According to one early study (Jones, Smith, Ulleland, & Streissguth, 1973), approximately one third of the offspring of chronic alcoholic women have this syndrome, and approximately one half will show some degree of retardation. Later estimates of prevalence vary

widely. It is not known why some children of alcoholic mothers are born normal and others are not, but it is known that the risk is dose related. The greater the maternal alcohol consumption, the greater the risk of fetal alcohol syndrome. Experiments with animals support these clinical findings; in both rodents and chickens, alcohol consumption adversely affects the fetus, and both risk and severity of damage are dose related (Sandor, 1968).

There are group differences in fetal reactivity to alcohol. Black women are far more likely to produce fetal alcohol syndrome babies than White women who drink the same amount during their pregnancies (National Institute of Alcohol Abuse and Alcoholism, 1994). Social, nutritional, and constitutional factors have all been implicated in this differential, but it is not really understood.

It is also known that beer drinking does far more damage to the fetus than wine or distilled spirits drinking. Again, it is not known why.

Additional evidence of the toxicity of alcohol on the fetus is found in the abnormally high rate of spontaneous abortion among alcoholic women. This is also true for experimental animals. Alcohol-drinking pregnant rats have higher rates of spontaneous abortion.

There is also evidence that in humans and experimental animals that moderate (two drinks per day) alcohol ingestion decreases the average size of

the neonate, although no other ill effects of moderate drinking are known. Although there is no reason to believe that an occasional glass of wine will damage a fetus, it is wisest to abstain from alcohol during pregnancy. Certainly heavy drinking exposes the fetus to great risk. Evidence for humans and experimental animals indicates that children born to recovering alcoholics are *not* at increased risk of abnormality.

CONCLUSION

Thus far we have concentrated on the pharmacology of alcohol and to a lesser extent of other drugs, as well as on the profound damage that these drugs can do to the body and to the mind. The alcoholism or substance abuse counselor must be knowledgeable about these matters; that knowledge will play a vital role in his or her function as an educator, as well as inform his or her understanding of what is going on for the client. When the client has difficulty understanding the counselor, the counselor will know that this may be the effect of the prolonged recovery syndrome or even of organic brain damage; the client's statement "I drink because I am so depressed" will be met by the counselor's "You are so depressed because you drink." Counselors do not need to retain detailed knowledge of the mechanism of alcohol's damage to the various organs—they have this text as a reference; but they do need fingertip awareness of the nature of that damage and how it manifests itself behaviorally. Human beings are, however, more than the sum total of

their biochemical transactions and pathologies; they are also social beings, and their social surroundings profoundly influence what and how they drink or use drugs. The next chapter will focus on how men and women have used alcohol across time and across cultures and how they have felt about that use.

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