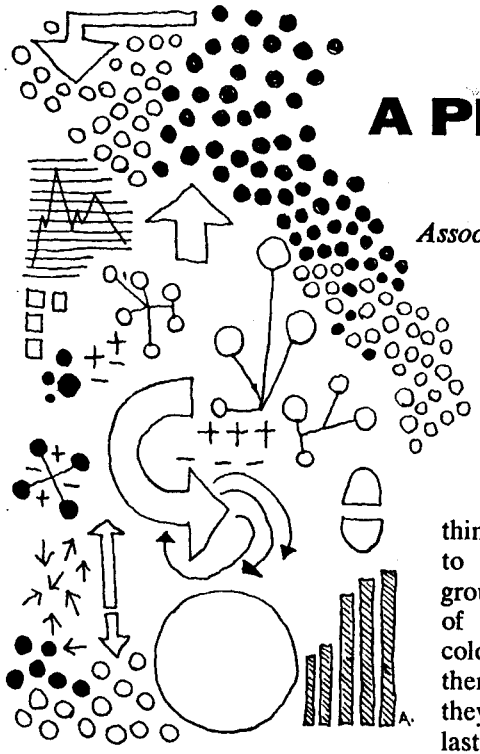

A Pharmacological Approach to Alcoholism

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"The simple act of prescribing sedatives in order to relieve the anxiety symptoms of the alcoholic is the one thing that will guarantee failure almost 100% of the time. There are for the most part no drugs of any great value in treating alcoholism."

THE DRUG I intend to discuss is ethyl alcohol, also commonly known as ethanol, and I want to start by orienting you as a pharmacologist would be oriented. Alcohol is one of a group of aliphatic sedatives or soporifics, which include liquid substances such as paraldehyde, solid drugs such as the barbiturates, and gases like ether and chloroform. These drugs are not narcotics, which take away pain but do not necessarily put one to sleep. The soporifics don't do anything against pain but do put one to sleep. They all belong to one group of compounds, the members of which possess similar pharmacological behavior. Quantitatively, there are differences in how quickly they start to act and how long they last, but they are all sedatives with the same qualitative effects.

The reason ethyl alcohol has deserved special attention and has a disease associated with it called alcoholism is, perhaps, that it was the first of the sedatives to be discovered and it has always been used socially. Alcohol is easily available without a prescription, without a physician, and has become interwoven with the fabric of our society and our social functioning. That, I think, is the reason why alcohol, rather than one of the other sedatives, has become the basis for the title of a disease, but a more correct title would actually be "sedativism." This "sedativism" is addiction to any one of the soporifics, and they are all grouped together in any good pharmacology text. What is it that they have in common? What is it that they do?

We will take one example, ethyl alcohol, and examine its properties. First of all, it's an irritant. When it is ingested, it irritates the stomach lining and increases the secretion of hydrochloric acid. Hence, other lesions of the stomach, such as peptic ulcer, esophagitis, and diaphragmatic hernia, which are made worse by the presence of acid, are all exacerbated by alcohol. It is also an irritant to the lower gastrointestinal tract, increasing motility and producing an outpouring of fluid, which will not infrequently cause enteritis or colitis.

In addition to its properties as an irritant, ethyl alcohol is the only drug known which is absorbed in significant amounts through the stomach lining. Thus, it will reach the bloodstream faster than most of the other sedatives and will work sooner. Ethyl alcohol reaches measurable blood levels within fifteen to twenty minutes after it is imbibed. Obviously, if you want to get a very quick blood level, you can breathe ether and get it in a matter of seconds; indeed, there are people addicted to breathing ether, chloroform or nitrous oxide, but these drugs are not so easily obtained.

Once alcohol reaches the bloodstream, three things can happen to it. It can be excreted through the lungs, and anyone who has taken a few drinks knows that others can smell alcohol on his breath. It can also be excreted through the urine. Both of these pathways take care of minimal amounts compared to the total amount of alcohol that has

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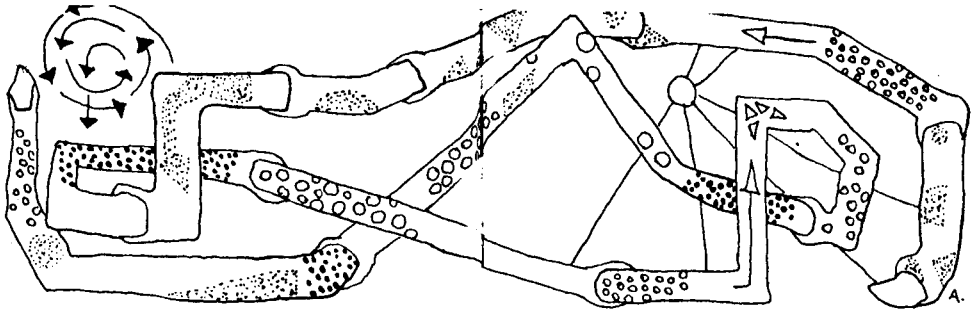
been ingested. Most of it is broken down to two carbon chain compounds which are the basic foodstuffs of the body. Once these have been formed, the particular food from which they came is no longer important; the body doesn't care whether calories come from alcohol or potatoes or carrots. However, alcohol goes through an intermediate acetaldehyde step, which is important only because the body cannot tolerate acetaldehydes. Normally they are broken down very quickly by an enzyme called aldehyde dehydrogenase to produce acetic acid, which is innocuous and can be burned up as a foodstuff. However, there is an enzyme-inhibitor known as disulfiram (Antabuse) which prevents the action of the dehydrogenase. Hence the acetaldehydes can no longer be gotten rid of, and they are very toxic. If an individual takes Antabuse, the enzyme-inhibitor, and then drinks alcohol, he is left with the toxic acetaldehyde and this will make him sick.

What determines the blood level of alcohol? The body has a limit as to how fast it is able to get rid of blood alcohol. The individual can metabolize about an ounce every two hours, and his blood-alcohol level will remain fairly constant. When alcohol is ingested, a measurable blood level is attained within fifteen to twenty minutes and reaches a peak at the end of the first hour or hour and a half. By



the end of the second hour, it starts down. The blood level reaches a normal (meaning unmeasurable) level within twelve hours. If the individual takes a very large dose, the peak will be higher and last a bit longer, but the blood level will nevertheless reach zero within twenty-four hours. In other words, no matter how much the individual is capable of drinking at one sitting, all of the alcohol is gone from his body within twenty-four hours. If he has any left, he has been drinking after that first drink. If that were all there was to it, people would not be drinking alcohol socially, because it wouldn't be serving any purpose. But when the alcohol blood level goes up, something happens in the brain, and that is the only reason alcohol is used.

The brain is essentially a switchboard with a large number of nerve cells (neurones) whose function is to transmit impulses from one point to another. The transmission of impulses depends on a group of biochemical events, and if you interfere with any one of these, you stop the neurone from functioning. You can stop it with a sledgehammer, with a right cross to the chin, or with a "chemical hammer"—it doesn't make any difference which. If you were to use a large dose of cyanide to do this, you could permanently stop the nerve cells from working. On the other hand, ethyl alcohol will stop the cells from working, but in a reversible fashion: It will interfere with function, but the body is able to fight off the drug and repair itself. However, if you take enough, you can kill the nerves. Now I can hit you on the head with a hammer so as to leave you unconscious, or I can hit you on the head with a hammer so you are dead. It's the same hammer, but it's a question of how hard I hit. So, too, with this drug ethyl alcohol: I can give enough of it to kill or a small dose to stop only certain of the neurones from working. Man discovered long ago that stopping some of the neurones from working on a temporary basis may be pleasurable.

All of the sedatives or soporifics —alcohol being only one of them— are irregular depressants of the central nervous system. By depressant is meant a substance which diminishes or stops normal function. The sedatives are irregular, because if they depressed everything simultaneously down the line, they would hit not only the thinking centers, not only the centers concerned with balance, but also the vital centers in the medulla which are necessary to keep the heart beating, the blood vessels contracting, and the lungs breathing. Any sedative which worked on these things evenly would be totally useless, because it would kill as quickly as it would sedate; but all of the soporifics work first on the cerebrum and the cerebellum, second on the spinal cord, and last on the vital centers. Hence a little alcohol works only on the cerebrum (the individual gets a little confused or high); a larger dose knocks the person out and he's unconscious with no reflexes; and a large enough dose will kill him.

When alcohol depresses the brain, it diminishes the psychomotor-activity level. The psychomotor-activity level may be measured by the amount of anxiety or tension noted. Diminished psychomotor-activity level means relief from anxiety, from whatever it is that bothers the individual: insomnia, tension, job and family pressures. When the person's blood alcohol rises, his psychomotor-activity level goes down; he is relieved, relaxed, less anxious, less frightened, less worried than before. But as soon as the blood-alcohol level starts to fall, this sedative effect begins to get lost and even with a big dose, such as a

couple of jiggers, wears off very rapidly after the second hour. If that were all, there is a distinct possibility, in my opinion, that we might not have the disease called alcoholism, because under those circumstances an individual could drink to get relief, his anxiety level would diminish, the drink would wear off, and he would come back to where he was before. But that is not what happens, because there is another effect and this acts in the opposite direction.

The second effect is to increase the psychomotor-activity level. This increase takes place immediately, right after the drink, but is not immediately felt because it is a change of less intensity than the sedative effect. This latter lasts two to three hours, while the opposite or agitating effect usually lasts for about twelve hours after one big drink. Therefore, as the short-term sedative effect wears off, the other effect of alcohol, increased psychomotor activity, becomes apparent. No one in this world can get a sedative effect from any known drug without it being followed by an agitating effect which wears off more slowly. The piper must always be paid.

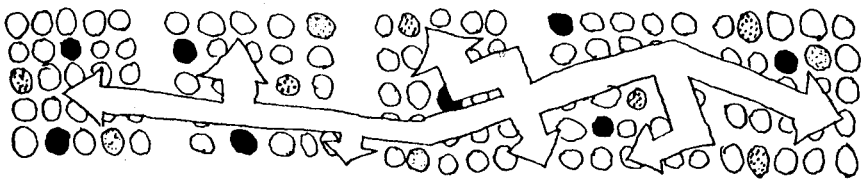
Around the end of the second hour after the original drink, when the sedative effect wears off and the agitating effect starts to gather momentum, the individual is more tense than he was before he drank at all. And so he will have another drink and the anxiety level will diminish again. But in about two hours, the psychomotor-activity level will rise again, higher than ever. This cycle of increased tension, drinking, short-term sedative effect, longer-term agitating effect, higher tension, drinking again, can go on for some time. Eventually, however, a point is reached when decreasing the psychomotor-activity level to a really low state is difficult, no matter how much alcohol is taken. This is because the agitating effects from all of the previous drinks add to one another.

This agitating effect occurs in experimental animals and in humans; it's the pharmacology of the drug. You don't have to be an alcoholic in order to experience it; anyone gets this effect if he drinks enough. For example, after an evening of heavy social drinking, you have a hangover, the common name given to this condition of after-agitation. A drink will relieve this because it provides sedation to counteract the agitation of last night's drinking. Of course, the morning drink is going to be followed by its own agitation for another twelve hours, but the drink may get you through a trying period until you begin to get a decrease of the agitating effects of the heavy drinking of the night before. By afternoon, you are feeling pretty well again. If, however, an individual has been drinking heavily over a long period, it may take many days for his psychomotor-activity level to return to its normal state.

The person who has taken so much alcohol over a protracted period of time that his psychomotor-activity level is extremely elevated may suffer one of several effects. He may start to shiver and shake, pace back and forth, sweat, palpitate, and show great agitation and tremulousness. This has been called a withdrawal state or syndrome, which is something of a misnomer, since the individual may still be drinking all he can hold. However, his agitated condition is breaking through because he has raised it so high that no amount of current sedation can possibly reduce it to bearable levels. If at this point he runs out of money or has an accident or for some other reason stops drinking and is left in his agitated state with no current sedation, he may get an extremely severe withdrawal syndrome. He may get alcoholic hallucinosis, which means basically that his brain is so irritable it sees, hears, feels things that aren't there. It makes up its own sensory input. And if the individual is one step sicker, he will go all the way to a condition known as delirium tremens (D.T.'s), in which not only the sensory input but the motor output has become tremendous, and he is literally unable to stop moving. He paces around, tears at his bed linen, hallucinates actively. A sine qua non to this delirium is not only the tremulousness, but absolute terror. The individual behaves as though someone were attempting to kill him. His psychomotor activity may reach the point of causing spontaneous generalized convulsions.

Basically, this is a condition in which the brain is tremendously overagitated. Thus, the guy who merely had six drinks last night and is edgy this morning and the guy with spontaneous seizures and a full-blown case of delirium tremens are suffering from the same disease, and this is caused by the pharmacologic effects of the sedative drug. It's just a matter of how much you take and how long you take it. One can produce D.T.'s with seizures in any individual at will, and in fact this has already been done with human volunteers who were not alcoholics. Probably the greatest single cause of convulsions in the U.S. today is alcohol, not epilepsy. With a man who develops seizures in middle life, has never had them before, and is a pretty heavy drinker, it's usually alcohol, not a brain tumor.

Up to now, we have assumed that what is happening is a reversible phenomenon, but this is true only up to a point. The central nervous system has the only cells of the human body which never regenerate. Every other cell, including those of the peripheral nerves, can grow back or reduplicate, but brain cells, once destroyed, do not. The individual is born with the maximum number of brain cells he will



ever have, and from then on, he has a decreasing amount throughout life. He never gets any more, and he is losing some, probably, every day of his life. Brain cells are destroyed by traumatic injury, by arteriosclerosis (less blood supply going to the brain as the person gets older), or by toxic substances. Fortunately, the individual has many more brain cells than he needs, and if ten percent of them are destroyed, the probability is that nobody will know the difference. On the other hand, if you lose twenty to thirty percent, even you may become aware that the memory isn't quite there, that you can't think your way through a problem the way you used to. The brain just shrinks and eventually will be half the size of a normal brain. In a chronic alcoholic, after many years of drinking, postmortem examination will show that not much of the brain is left. Now the alcoholic may of course do other things to his nervous system from bad eating habits, but the probability is that he can do this damage just with ethyl alcohol, eating a beautiful diet the whole time, because ethyl alcohol per se can apparently injure the brain biochemically. It's probably a reversible injury most of the time, but not always, so that eventually brain cells will get destroyed. When enough of them are damaged, you get irreversible changes in the behavior and the psychological status of the individual. These may never return to normal, no matter how long he's sober and eats a good diet.

The effects of alcohol on other parts of the body are relatively unimportant, since they involve but a minority of heavy drinkers when compared with the effects upon the brain, reversible or not, which take place in 100 percent of alcoholics. For example, cirrhosis of the liver occurs in about eight percent of alcoholics and one percent of the nonalcoholic population. Then there is pancreatic disease in a very small percentage. There is an increased likelihood of pulmonary infections, perhaps due to the sedation of the patient so that he doesn't clear the secretions out of his chest well enough. There is also alcoholic cardiomyopathy, a disease of the heart muscle due to heavy intake of alcohol over a period of years and especially prevalent in Negro males. All of these complications of heavy ethanol ingestion are comparatively rare.

Now let us consider the nature of addiction. Addiction is a circumstance in which a drug has been utilized and is now gone from the body, totally gone, but has resulted in an organic, measurable, overt disease state, a condition which demands more of the drug in order to get relief. This doesn't necessarily mean the individual is habituated or emotionally dependent, but just that he is measurably sick after the drug is gone, and he requires more of it for relief. Alcoholism was for years mistakenly considered to be a habituation, but alcohol is a true addicting substance, as is every one of the aliphatic sedatives or soporifics. Now what is cross-addiction? This simply means that among this group of sedative drugs, I can replace one with any other at any time, at random. I can replace alcohol with nembutal, nembutal with pentothal, pentothal with paraldehyde, paraldehyde with chloral hydrate, and if I adjust the dosage correctly and give it intravenously, the individual will probably not know which he is getting, because the effect upon the brain is almost identical. Hence there is cross-addiction among the different members of this group of compounds, but there is not cross-addiction between these and narcotics. Both sedatives and narcotics are capable of addicting, but these addictions are separate disease states. Of course, one individual can become addicted to both groups of compounds, but this is very rare. We don't know why, but it is unusual to find an alcoholic who is also a narcotic addict, or vice versa.

The withdrawal state from narcotic drugs is less severe than withdrawal from sedatives. Addicted individuals rarely, if ever, die from narcotic withdrawal, whereas death from alcohol (or other sedative) withdrawal is quite common. Secondly, an individual who is taking a new dose of sedative to control the psychomotor agitation caused by yesterday's intake is not a well-functioning human being. Either he is near to sleep or he is very uncomfortable and functioning poorly. On the other hand, if narcotics are given to the narcotic addict to stop the withdrawal syndrome, he can function and he need not continue up a scale of increasing psychomotor activity. A narcotic addict can be maintained on a stable amount of drug for years and can function all during that period of time. Thus the narcotic addict can maintain a plateau, whereas the alcoholic, due to the pharmacology of his drug, is compelled to reach beyond, losing functional capacity in the process.

Now I want to discuss tolerance. Suppose you take ten drinks and I take ten drinks, and within an hour I am lying asleep on the floor and you are walking around appearing as though nothing had happened.

This means that you are more tolerant of the pharmacologic effects of alcohol than I. Thus, tolerance accounts for variability of effect relative to a single dosage of a drug. There are individual differences in the rapidity with which alcohol is metabolized. In addition, if we look at one individual over his lifetime, we will find that his tolerance diminishes with age. For example, if an alcoholic is abstinent for ten years and then relapses, he will get much sicker, with less alcohol intake, than he used to. The difference is simply that his body and especially his brain are ten years older, and every year his tolerance for alcohol has decreased, even though he wasn't drinking. From this point of view, alcoholism is a self-limiting disease because, with diminishing tolerance, the amount the alcoholic can drink eventually is very little and the amount of suffering he does for it is very great. Sooner or later, either the disease kills, or he decides to stop drinking, and our job is to try to convince him to quit before it kills him.

So far, we have discussed true tolerance, tolerance to both the sedative and the agitating effects of alcohol. There is another phenomenon often described as tolerance, which refers to current sedation being countermanded by agitation from past drinking, and which really isn't tolerance at all. Take, for example, an alcoholic who is knocking off a quart a day. Give him ten or twelve drinks. You or I would be lying on the floor, but he is not even sleeping. His psychomotor-activity level from past drinking is very high, and he can take a heavy load of sedation just to bring him to a relatively normal psychomotor level. He demonstrates the two opposite pharmacological effects of alcohol operating together, and such individuals will not infrequently be found walking around with blood-alcohol levels above one-half of one percent, which is usually considered to be within the lethal range. These people may be driving a car, walking around, talking for hours on the telephone—but with one difference: Tomorrow they will be amnestic for the event. This is called a blackout, and people get these quite soon after they start to drink in an alcoholic fashion; yet there is already a considerable amount of dysfunction, of illness, in a brain which achieves a blackout state. It isn't that the individual forgets what he was doing; even *while* he was doing it, his consciousness was clouded and he did not consciously "know" what he was doing.

I want to distinguish between alcohol addiction and alcoholism, for they are not the same. One is a pharmacological term; the other, a medical term for a disease state (and, as indicated before, it really should be "sedativism" rather than "alcoholism"). I can produce alcohol addiction in any one of you by giving you ten drinks today, twenty tomorrow, twenty the next day, the next, and so on until I stop the alcohol and you start having seizures. You are in a withdrawal state, alcohol-addicted, but that does not make you an alcoholic. The sine qua non of alcoholism is that the individual goes back and does it again and again, even though he suffers loss of job, loss of health, loss of family. The definition of the disease includes not just addiction, but compulsivity. Now why, when he suffers so much, does he do it again and again? Because he is compelled to, but not primarily because of the suffering that goes with it. He would like to find a way of avoiding the suffering and not infrequently will come to the physician asking for medication that will let him have the sedation but prevent the suffering. The reasons for the compulsion to drink may be psychological; I used to think that, but now I am not so sure. These individuals may have a different psychomotor-activity level on a biochemical basis. The norm, for them, may be higher than other people's because of a biochemical defect within the brain, so that they do not feel like other people until their psychomotor level is artificially brought down by sedatives. This may or may not be so, but many people believe that such a biochemical defect does exist.

There is a clue in the frequent statement of alcoholics: "I had my first drink at eighteen, and I finally found out what it was like to feel normal. I had felt abnormal all my life until that first drink, and then I felt like everybody else. If I could just maintain that level . . ." But of course an alcoholic can't. If his initial level of anxiety was already too uncomfortable, too agonizing to bear, how can he tolerate the increased anxiety from the agitating effect of the sedative? The only way he knows to relieve it is to take another drink, and so he's off and running. His decision to drink or not to drink has to be made before he takes the first one.

If you or I have a hangover after a party, it is unlikely that we are going to drink to relieve our temporarily elevated psychomotor-activity level, unlikely that we are going to take a "hair of the dog," except perhaps one Bloody Mary, and the majority of us won't even do that. Most of us will say: "I shouldn't have drunk so much, and I won't do it again for a long time." We can tolerate this degree of psychomotor activity, and after a few hours we begin to feel better. The alcoholic can't tolerate it and must drink again. This is not voli-

tional; he must drink again to relieve the discomfort which he couldn't tolerate even when it was less intense before. It is relatively easy to understand continual drinking under these circumstances; but suppose we see this man after he has been sober six months or six years. He gets anxious, takes a drink, and in the majority of cases begins the cycle again. He can't plead ignorance, and he did it cold sober.

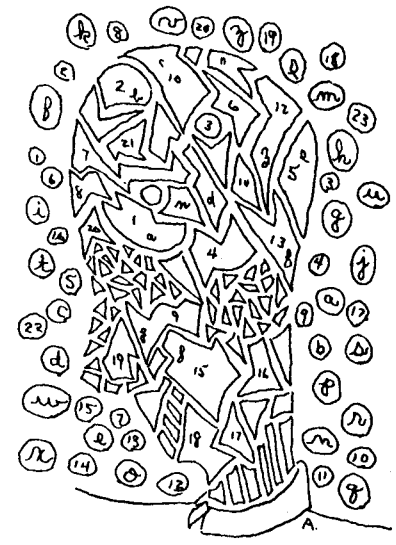
For this, there are only two possible reasons. One is that alcoholism is a compulsive psychiatric syndrome of such a nature that his psyche is going to force him at knifepoint to drink throughout his life. The other possibility is that on a biochemical basis the alcoholic is truly uncomfortable without sedation. How much is psychological and how much is biochemical? Is the individual starting out with a normal anxiety level, or at a level higher than normal? If the psychomotor-activity state can be markedly modified by drugs, as it can, how do we know it was "right" in the first place? We don't. It is quite possible that the individual has a biochemical defect to start with, which creates a fertile soil for the growth of alcoholism.

The treatment of this disease is to stop the use of the causative agent. When the physician first sees the alcoholic, he is a very agitated fellow and needs sedation, but if we sedate him, whatever we use will cause greater agitation later. The doctor is in a circumstance where he's damned if he does and damned if he doesn't. Hence Rule One: If you're going to have to relieve the symptoms of this alcoholic by giving a sedative drug, you must rarely if ever give it outside the hospital. Give it only in a situation where you have *complete* control of the patient and the dosage of medication. In a hospital, the physician can give some sedatives in association with phenothiazines, drugs such as Thorazine or Sparine. These drugs will not usually prove addicting, will increase the effects of the sedatives the physician is giving, and will enable him to relieve the patient without causing a marked increase in agitation. By the fourth or fifth day, the alcoholic will perhaps still be a little tremulous, but will usually feel well enough to go back to his job. But, though the withdrawal state is at its worst for only a few days, its more subtle effects—psychological abnormality, character disorganization—may last for weeks.

Now the patient has been withdrawn from alcohol and is ready for long-term therapy for the recidivism or compulsivity that makes him start the whole cycle again, over and over again. You've got to help him build some sturdy walls before the wolf comes to blow again at his house. And you begin to appreciate the vigor and persistence necessary in therapy.

Obviously, the way to handle this individual in order to keep him from drinking again is *not* through the use of sedative drugs, because as soon as you give sedation of any sort, he's off and running again. In fact, the simple act of prescribing sedatives in order to relieve the anxiety symptoms of the alcoholic is the one thing that will guarantee failure almost 100 percent of the time. There are for the most part no drugs of any great value in treating alcoholism. Librium, Valium, Miltown (Equanil), and Doriden are often used, but share an undesirable propensity for being variably addicting. Remember, we noted at the outset that all of the sedative drugs are qualitatively identical and can replace one another: alcohol, barbiturates, chloral hydrate, paraldehyde. The individual develops a cross-tolerance, so that if, for example, he builds up a huge tolerance for alcohol, it will take a huge amount of ether to knock him out on the operating table, a fact which all anesthesiologists know. If you use other sedatives to treat the alcoholic, you go back and forth between the two and never solve anything—the individual remains addicted. Patients who take Miltown or Librium often get so jittery on these drugs that they go back to alcohol to get sedation.

The case of the phenothiazines is somewhat different. These drugs (Thorazine, etc.) work on a different part of the brain, and alcoholics



usually don't like them, don't get the relief they want. Thorazine does not significantly increase the psychomotor-activity level and hence is practically nonaddicting, but I believe that the individual who takes it will more readily make a transfer back to alcohol than those who use no drugs. Since Thorazine doesn't give the alcoholic what he wants, he is almost bound to take something else in short order. I'm also convinced that if you give any sort of "oral magic" to an alcoholic, you are almost guaranteeing a relapse. If you give a pill (even if you fill it with sugar), you are in essence saying: "Yes, there is something wrong that is correctable by some magic taken by mouth." But the fact of the matter is, there are no sedatives or tranquilizers that do anything for this disease except cause trouble, and one should do

everything possible to assist patients to realize that there is no magic.

Antabuse, on the other hand, has something to offer in the treatment of this disease. Antabuse is not a drug which works on the central nervous system; it does not relieve anxiety; it does not change the psychomotor-activity level of the brain. It has no effect whatever, unless the individual takes alcohol on top of it, in which case alcohol metabolism stops at the acetaldehyde stage. The acetaldehyde level builds up in the bloodstream, and it gives a toxic reaction, which includes nausea, vomiting, flushing, vasomotor collapse, and, in extreme cases, death. Most patients, even without trying it out, will believe this. Antabuse is taken every day and gives four or five days' protection against drinking.

Suggesting Antabuse permits me quickly to gauge the degree of motivation of the patient. If I get a negative attitude, I know the odds of doing anything with this patient are poor. But if the patient is willing to take it, I know I have a more promising situation. Second, Antabuse stops impulsive drinking completely, because if the person on Antabuse drinks, he will be quite ill. If he comes in with a relapse, I have the opportunity to prove to him that he decided in advance to get drunk and so stopped taking the Antabuse several days ahead. I always have told him in advance that if he stops his Antabuse, it will be for one of these reasons: "I ran out and forgot to buy more"; "I forgot to take it"; "I went on a trip and forgot to pack it"; "I wanted to do it on my own and not use a crutch"; "I was so well, I thought I had no further need for it." Now, if he relapses, I have predicted these "reasons" in advance and so can help the patient see that he planned the drunk, and then we can get down to the real precipitating causes, not the phony ones.

The third thing Antabuse does is to assist patients to answer, once a day, the question of drinking or not drinking. They are not preoccupied about drinking all day long, and they feel a tremendous relief, at least for a temporary period. Certainly by itself Antabuse is not the answer, but it is a helpful tool, and it gives you time to work with your patient. Some people take it for years in order to guarantee their sobriety, but it must be taken voluntarily and only because the individual wants to stop drinking.

In treatment, cessation of alcohol intake is the first goal. Education about the nature of the disease is essential, but most important is to bridge the alcoholic's isolation by the use of simple understanding and compassion. Usually the alcoholic has been trying to say right along (and pretty much no one in his environment has understood): "I'm strung up, I'm caught, and nobody seems to realize how violently ill, how terribly trapped I am. All people say is, I just won't quit drinking, or I bring it on myself." When the alcoholic becomes aware that you do understand, you may have a successful patient. With compassion and consistency, a great deal can be accomplished.

The long-term treatment of alcoholism must be directed toward increasing the abstinent alcoholic's capacity to tolerate anxiety without recourse to sedation. Obviously, he has to be given something to replace the "oral magic," and the only thing I know is a helping hand. The helping hand may be Alcoholics Anonymous or psychotherapy; it may be the clergyman, the social worker, or the physician. The alcoholic needs people who understand and are compassionate, who offer the ear, the time, and the hand to help him through all his discomfort.