abscess. There is a certain proportion of cases in the list of fatalities where there had been a localized abscess, but before the time of operation the abscess had ruptured, general peritoneal involvement following. There is your danger in waiting. It was my rule at first in these cases where I felt there was a perforated appendix to operate almost immediately, but I have departed from that rule of late. I have found that many of my cases come in in a very much depressed condition. Then to the fatigue of moving as well as the ride are important, and it has been my rule to simply wait until the patients have had a moderate period of rest in bed in the hospital. Where vomiting exists this is a cause for waiting. We always resort to lavage in cases of vomiting. When the patient has come to the hospital with a localized mass in his right iliac fossa, whether his history points to perforation of the appendix or not and you think he could safely take ether I always operate and never wait. By this means I believe we avoid danger and do not increase it. Under no circumstances has death resulted from the operation.

DR. LAPLACE, in closing—Dr. Ochsner's position presupposes that he can tell what is going on in the abdominal cavity. He presupposes that this abscess has not burst nor is about to burst. The last case I operated on was only taken sick seventy-two hours before. I insisted on immediate operation. As I opened the abdomen I found a large mass. To have waited twenty-four hours would have been the death of that boy. I agree that in a great many cases surgical interference and the swabbing of the peritoneum causes peritonitis, but that simply means we do not know how to treat peritonitis. We have more severe cases of peritonitis to treat than slight ones. When we succeed in curing these bad ones we surely should be able to cure the mild ones. Our efforts must be toward doing the operation as soon as possible and apply those principles in the treatment of peritonitis that we would anywhere else. Why should we delay an hour in the treatment of or operation for peritonitis? I agree with Dr. Ochsner that his treatment is ideal when it is impossible to operate.

ALCOHOLIC EPILEPSY.*

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The term "alcoholic epilepsy" is comparatively new in medicine and describes a class of inebriates who have convulsive symptoms resembling epilepsy. Some writers call it a form of delirium tremens and acute alcoholism; others consider it a distinct epilepsy due to the toxic action of alcohol on the brain centers. In some instances the craving for alcohol precedes the paroxysm or epileptic fit like an aura or premonition. Sometimes the convulsion is intensified and increased by alcohol, or in other instances it may be neutralized and the direction of the symptom changed. The sudden maniacal impulse to become narcotized by alcohol, as seen in the dipsomaniac and periodic inebriates, closely resembles epilepsy. A number of cases have been noted where a distinct convulsion preceded the use of spirits and was followed by a continued impulse to drink. When this craving was neutralized by treatment, the convulsion returned. When spirits were resumed the epileptoid symptoms disappeared. Convulsive symptoms and paroxysms of brain disturbances are often due to toxins, and yet, when they occur with great uniformity, other causes are both active and predisposing.

Convulsions of a uniform type following the long-continued use of alcohol are called epileptoid. It is not clear whether this uniformity of type in appearance is due altogether to the action of alcohol, for the reason that the use of alcohol is in many cases a symp-

tom and not a direct cause. As in other psychoses and neuroses, the dividing lines between the use of alcohol as a symptom or an active cause, can not be easily traced.

In all these cases hereditary tendencies and predispositions, together with the present conditions of vigor or weakness, are the main factors in altering the type and form of degeneration present. So many factors make up the so-called diseases of alcoholism and epilepsy that it is impossible at the present time to make a clear discrimination. My purpose in this paper is to group some of the general symptoms which appear most frequently in a clinical study of alcoholic epilepsy. The following classification is sustained by the study of a large number of cases, and will give some idea of the diversity and complexity of the causes: 1, The convulsive and maniacal types; 2, the demented and confusional form; 3, the trance automatic and psychic classes. These divisions are clearly described by the history of some illustrative cases.

THE CONVULSIVE AND MANIACAL TYPE.

In the first type the following is a descriptive case:

CASE 1.—G., an iron merchant, aged 44, had used spirits from infancy, given at first by the physician as a tonic, then later as a stimulant. He was first intoxicated during his college life, and up to 40 years of age, while in business, he drank daily small quantities of spirits. During a severe nervous strain, he had an attack of profound drunkenness, marked by excessive muscular and mental agitation. During this period he both walked and talked incessantly and was very irritable, then he became stupid and slept twenty-four hours.

This was followed by another attack of drunkenness with an increasing period of sleep and prostration. Then came recovery with a free interval of several months during which he was a total abstainer. Similar attacks continued with increasing frequency until at present they occur every two weeks. The attacks are convulsive, marked by mental excitement and trembling, which continues for hours. In some instances he bites the tongue and the stupor which follows is profound and long continued. His mind is affected and delusions of persecution with great irritability follows.

CASE 2.—In the second illustrative case, the use of alcohol began at 30, following exhaustion and business strains in a previously healthy man, who was an inventor. Spirits were used irregularly at night for the purpose of relieving the state of exhaustion. Five years after he came under my care for some strange symptoms occurring when asleep. He would go to bed partially intoxicated and in the night have a convulsive attack; then beat the bed, turn over the chairs of the room, bite his tongue, fall into a deep sleep and awake next morning without any recollection of what had happened. During the day he was maniacal in his anger and hypersensitive to every exciting condition. He had delusions and hallucinations and feared injury from every one he came in contact with. The removal of alcohol lessened many of these symptoms, but the fits at night continued.

These two cases are examples of a class of inebriates in which intoxication is of a sudden maniacal type followed by muscular explosions of extraordinary character and ungovernable conduct. Such cases are not infrequently seen on the street in the fighting delirious drunkard, who at home beats his wife and family, resists arrest and may commit all kinds of violence on the slightest provocation. These attacks are usually followed by profound stupor, and in many of them injury of the tongue and convulsive twitchings of the voluntary muscles are noticeable.

DEMENTIA AND CONFUSIONAL STATES.

The second form is that of dementia and confusional states, in which the epileptic symptoms are prominent and may be illustrated in the following cases:

* Read at the Fifty-third Annual Meeting of the American Medical Association, in the Section on Nervous and Mental Diseases, and approved for publication by the Executive Committee: Drs. Frederick Peterson, Richard Dewey and H. A. Tomlinson.

CASE 3.—A physician, aged 45, with a very laborious practice drank whisky three or more times every day with much regularity. He was never seen intoxicated, but was considered generally stupid and dull and inclined to fall asleep on every occasion. This was attributed to his use of spirits and over-work. He was attacked with severe chills at night, associated with spasmodic twitchings of the muscles, which after a short time were followed by profuse perspiration and profound sleep. Quinin and whisky were given in large quantities, supposing the attack to be malarial. The chills and convulsive trembling increased and the coma following became more prolonged. His mind failed and he was taken to an asylum where the epileptoid characters of these symptoms were discovered.

CASE 4.—The second example was that of a brewer, aged 44, who drank beer daily and spirits at irregular intervals. He was bloated and very dull and stupid in his mind. He complained of sudden attacks of trembling and inability to control himself. These attacks occurred at irregular intervals and were followed by periods of great dulness and apathy. He became melancholic and talked of some great disaster and peril to his life and property. His family physician ordered whisky alone. This gave temporary relief, but was followed by a return of these muscular tremblings with increasing stupor and dementia. By my advice, all spirits were removed and the convulsive character of the symptoms disappeared, but the dementia continued and terminated in death three years later.

CASE 5.—The third example was that of a civil engineer who had drank for years regularly many times a day. He was called a moderate drinker, having never been intoxicated. For three years past he has complained of great nervous exhaustion in the morning, which at times was so intense as to oblige him to return to bed. There were no preliminary symptoms of these extreme depressions the night before. His sleep was sound and without dreams. It was found that during the night he had attacks of muscular trembling, during which he would moan and roll up and down the bed, sometimes striking out with his arms wildly as if in great distress. The family physician diagnosed nervous and brain exhaustion and ordered strychnin, cinchona and spirits.

The symptoms of exhaustion increased and attacks of shivering and so-called chills occurred during the day, sometimes lasting for ten minutes, and were followed by prolonged sleep. Often the sphincters would relax and he would be unconscious of the consequences. The treatment was changed to bromids and bromidism followed, resulting in death.

These examples are not uncommon, but the epileptoid conditions are rarely recognized. The dementia and confusional states are treated as primary. Whatever the real condition may be the convulsive tremblings and unconsciousness point to a form of epilepsy.

AUTOMATIC TRANCE AND PSYCHIC CASES.

In the third class are included the automatic trance and psychic cases. These are more numerous and obscure than the others and have only recently attracted attention. In literature they are described as double consciousness, epileptoid trance states and automatons. I have studied a number of histories of persons of this class under the title of alcoholic or inebriate trances, the principal symptoms of which were sudden blanks of memory, followed by automatic acts which the person never remembered. Many of these cases gave no evidence of disease, but seemed to be conscious and clear up to a certain point, then a sudden partial narcotism followed in which some parts of the brain were intact, but memory and consciousness were suspended. In other instances short periods of alcoholic stupor or excitement preceded this condition. The narcotism from alcohol was always prominent. In some instances strong hints of epileptoid symptoms were present. Following is an example:

CASE 6.—H. B., a physician, aged 38, who drank spirits steadily for ten years, then had sudden blanks of memory, followed by strange, unusual acts. He drank at meals and before retiring and smoked excessively. He was never apparently intoxicated, but for some time had been growing dull and irritable. He would have attacks of anger or depression at real or imaginary causes, go to sleep in his chair or any place he might be, then awaken and go about his usual work, but be unable to recall any event that had happened.

During this blank or trance period, he would visit patients, administer remedies and act with apparent self-control. The only unusual symptom was gravity of manner with prolonged staring, and indisposition to talk on any subject outside of routine work and this only in monosyllables. These trances increased in frequency and were preceded by a chill with sharp muscular shivering and emotional disturbance. Under my care we were able to trace much mental disturbance before the attack, while during the attack he was clearly in a demented condition. The withdrawal of the spirits resulted in restoration.

CASE 7.—In a second example, a farmer who had drank steadily for years, would suddenly stop all work and without reason or motive go to bed and remain on his back perfectly still with his eyes open. When spoken to, he answered rationally, but would give no explanation of his conduct. In an hour or more he would arise and go back to work as if nothing had happened. It was particularly noticed when he arose from the bed he did not recognize the night from the day, but would go on with the work in the night just the same as the day. When told it was night he seemed surprised, but continued to be active with some work by lamp-light. After a sleep he recovered and had no recollection of what had happened.

He continued the use of spirits regularly as before and a distinct connection was traced between the spirits and these trance states. These attacks increased and were preceded by more pronounced unreasonable conduct, such as rushing out of church, leaving his horse and wagon in the street or in the field, and seeking his home and bed at once, despite all obstacles. The trance state following increased in irregularity and duration. He finally died suddenly.

CASE 8.—A third example was an inebriate, a traveling man, who drank with great regularity for months at a time and never seemed intoxicated. Suddenly he would stop all business and manifest religious proclivities, attend churches and prayer meetings, take part in the Salvation Army, then after a day or more go to his room, sleep a few hours and start out on his accustomed work.

After an interval of from a few hours to several days, he would recover and be unable to recall any events which had happened in the interim from leaving his business, showing religious fervor, and returning to business again. He depended on his notebook for evidence of where he had been and what he had done. To his customers, during this trance state, he was excessively polite, bland and clear. He refused to drink, but smoked continuously. On recovery from the blank period he complained of great depression of spirits and commenced to drink again. It was evident that these sudden delusions and religious fervors followed unusual efforts and business strains. During the attacks there was loss of appetite, great pallor and extreme uneasiness and inability to sit still or converse. This man fell into the hands of a quack and died of some obscure disease.

Other examples less prominent, exhibiting a great variety of complex symptoms, have been noted. In most of them there is much uniformity in the symptoms, particularly in the sudden alterations of personality and conduct and the automatic trances which follow. When these persons come under medical care, explosive impulses are considered signs of insanity and the blanks following are regarded as dementia. Thus a moderate drinker may commit some overt act, then fall asleep and go about in a dazed condition for some time. This is an epileptoid state. Not unfrequently such cases are brought to the asylum and when they awaken have no

conception of why they came, and afterward console themselves with the idea that they must have been drunk at the time. In some instances, evidently, the most prominent early symptom is a sudden maniacal impulse to drink to stupor, and on recovery the trance condition begins.

Gourmands and wine drinkers of this type display most unusual conduct or paroxysms, associated with chills or profuse sweating followed by stupor, then the dazed trance state follows. A number of cases under my care have but indistinct recollections of events preceding their admission for days and sometimes weeks. To their friends their conduct was strange, but the general symptoms were not those of alcoholic intoxication, but rather of general stupor from some brain trouble.

In these trance cases there is not necessarily any sudden explosive symptoms, but more often a sequence of mental degeneration, noted by defects of reason and emotional instability. In these three classes nearly all alcoholic epileptics may be grouped. The paroxysms and explosions of nerve energy, also hallucinations and delusions, are probably always present. Acts of violence, homicidal impulses, sudden chills, followed by motor spasms and sleep, are most significant symptoms.

A number of inebriates suddenly develop delusional states which are commonly seen in nearly all epileptics. Delusions of grandeur or fear of sudden death from injury, passing off in a short time, are intimations of epilepsy. These symptoms following or associated with the excessive use of alcohol are still farther confirmations. There can be no doubt that alcoholic epilepsy is increasing. In a recent examination of two hundred cases over 50 per cent. showed well-marked symptoms of epileptoid states. Inquiries indicate the increasing frequency of convulsive symptoms, resembling epilepsy, and there can be no doubt that these symptoms are coming into greater prominence yearly.

ETIOLOGY.

In some personal studies made ten years ago epilepsy was noticed in 5 per cent., and obscure convulsive symptoms associated with inebriety were recognized in, at least, 10 per cent. more cases. In all not over 15 per cent. of the cases then studied exhibited symptoms of this condition. In the police records the number of convulsive spasmodic intoxications with strange symptoms of mania and sudden nerve explosions of conduct have been known to occur in over 50 per cent. of all the cases. They are termed delirium tremens cases in many instances and the convulsions which occur are all considered part of the alcoholism from which the patient is suffering. Many of these police-court cases with well-marked epileptoid symptoms die before the later stages of epilepsy are reached. It is a fact that has not been recognized that sudden and convulsive intoxications are very often followed by pneumonia and death. Pneumoparesis seems to be the special form of disease which follows. In the first class of epilepsies which I have noted, these sudden pneumonic collapses are very common. In the second class of dementia, cerebral hemorrhages and nephritis are also common sequences. Homicide and suicide are frequent in both these classes. In the third class of trance cases well-marked epileptic convulsions are more pronounced.

In both of the other classes epilepsy may appear in its classical forms and inebriety subside, or the use of spirits may continue in diminishing quantities. In all these cases acute inflammations of the kidneys, of the liver and stomach, may occur suddenly, followed by

death. Well-marked classical epilepsy, seen in an inebriate, is of short duration.

INFLUENCE OF COMPLEX ALCOHOLS.

The increasing prominence of these epileptoid symptoms in the inebriate of to-day is apparently traceable to the changed form of spirits used. While it can not be stated authoritatively that this is the most prominent cause, it is evidently very influential and responsible in a large degree for many of the symptoms which follow. Within a few years highly fortified and flavored alcoholic drinks have become very popular, such as bitters, vermouths, absinthe, cocktails, high-balls, champagnes and other high-sounding names for fortified and strong-flavored alcohols. These drinks are nearly always composed of new and cheap alcohols, and frequently wood-spirits and often whisky from potatoes and beet roots are used, depending largely on the price in the market. These alcohols are concealed by flavoring substances, such as bitters and syrups, the purpose of the dealer being to increase the profit and make the drink attractive.

When these different drinks are given to animals, well-marked convulsive phenomena follow. The intoxications are always sudden and paroxysmal. Champagnes resemble these drinks in their effects only the period of exhilaration is longer and more intense and the coma following slower and more profound. By these mixed drinks the blood is driven to the brain with great violence and vasomotor paralysis comes on early, in some cases resembling a shock from injury. The control centers of the brain are very quickly broken up and the degree of intoxication is marked and prolonged. The bitters, absinthe and cocktail compounds have a peculiar paralyzing action on the sensory and motor centers. This resembles toxic convulsions in the disturbance and explosive character of the symptoms. In club-houses and in high-class bar-rooms where these drinks are popular, the exhilarations and stupor following seem to have a direct connection with the spirits used.

Among the lower classes and in cheap bar-rooms the same convulsive class of intoxications with wild fitful manias and heavy stupors are seen. How far wood alcohol is used in these highly-flavored drinks can not be ascertained, but every year the production of this alcohol is increasing beyond the normal demand, and this excess, selling at half the cost of other alcohols, is used up in some unknown way. The cheap brandies and whiskies, no doubt, contain this form of spirit. The large importations of bitters and absinthes and their manufacture in this country are also evidence of the changes in the character of the drinks used. The cheap champagnes manufactured in this country, while less irritating than foreign drinks of this kind, are more largely used than ever. The physiologic effect is that of intense irritation, congestion and paralysis.

It is the form of alcohol that is dangerous, not the compounds mixed with it. In the experiments of Dumont's, in which various alcohols were given to dogs and goats, it was proven beyond doubt that complex alcohols were convulsive in their action, producing local as well as general paralysis. The more complex the alcohol the more dangerous it was. Beyond the influence of mixed alcohols in the etiology of these cases, there are to be considered the neurosis and psychosis, both from hereditary surroundings and bad living. There is in many cases predispositions for nerve explosions which makes the person more susceptible. This must be taken into account in estimating the symptoms.

DIAGNOSIS.

The diagnosis in these cases is usually not difficult. While the general toxic states are alike differing only in minor particulars, there are sometimes very striking variations which have apparent connection with causes that are known. It is evident that the patient who drinks quantities of mixed alcohols and develops epileptoid symptoms is suffering from some form of toxic concussion, manifested in this way.

In other instances, the accumulation of toxins, formed both within and without the body, concentrate on the nerve centers, producing explosive symptoms. The masked cases are always difficult to determine except from a careful study of the habits and surroundings of the patient. The sudden onset of strange, unusual symptoms suggest epileptoid conditions. The excessive use of spirits preceding the paroxysm or fit are always significant hints of this condition. In a recent case under my observation, a moderate drinker after two glasses of champagne became delirious, maniacal, and while walking to his room had a distinct convulsion. This was the culmination of a long premonitory period in which the toxic symptoms showed the tendency to take on this form.

PROGNOSIS AND TREATMENT.

The prognosis is always grave and unless the alcohol is removed at once the prospect of intercurrent disease or death or pronounced epileptic symptoms is very serious.

In the treatment, total abstinence from all spirit compounds with brisk elimination, restricted diet, nerve and brain rest continued for a long time give the only hope of restoration. Often a radical change of business and surroundings is most essential for successful treatment.

SUMMARY.

Some of the facts I wish to make prominent are:

1. Alcoholic epilepsy is increasing rapidly in this country.
2. It is a psychosis and neurosis which should be recognized, having distinct symptoms, the recognition of which is imperative in the treatment.
3. Where the toxic symptoms are convulsive and explosive and come on suddenly, the future of the case is very ominous and the warnings from these symptoms should be heeded.
4. The connection between mixed drinks and these spasmodic symptoms are traceable and should be considered in the prognosis and treatment.
5. The treatment and curability of these cases can be carried on with great hopefulness, and undoubtedly in the future will become a prominent part of the work.

DISCUSSION.

DR. C. H. HUGHES, St. Louis—I do not agree with all that Dr. Crothers has said on the subject, among other things being his assertion that the sudden onset of alcoholic epilepsy is the gravest form of the disease. The first case of genuine alcoholic epilepsy that ever came under my observation, outside of a lunatic asylum, occurred for the first time in an individual who was on a drunk. It was the first attack of this kind that he had ever had. He was seized just as Caesar was in the market-place at Rome, although he sat at the drinking table and fell under the table, convulsed and unconscious, and with frothing at the mouth, and the usual sequelæ of a well-established case of severe epilepsy. I was interested in that case, of course, because I saw it in its inception, and I then for the first time knew of alcoholic epilepsy. I wondered if the man were going to become a constitutional epileptic. I did not know so much then on the subject as I do now. I had not then

been superintendent of an insane hospital, as subsequently I saw several hundred patients daily. I was then a young army surgeon, not 22 years of age. I took this man, who was a personal acquaintance of mine, to my hospital and took care of him and nursed him through the paroxysm. He went to sleep after the paroxysm had passed off and he slept in the hospital until the next morning, when his recovery seemed complete. I have watched that man ever since and he is living to-day and has filled some important positions in life. To my knowledge, he has never had any similar attacks. He has modified his habits of drinking. I told him exactly the condition he had been in, and I think I frightened him into a better life. This was purely a case of alcoholic epilepsy. Of the existence of this form of epilepsy I have never since entertained a doubt. Alcohol is a most potent causative and developmental factor in the generations that immediately succeed inebriate lives. The tables of Mognan and others have clearly established this.

DR. J. P. MCBRIDE, Los Angeles, Cal.—I recall a case of this condition in a young man about 25 years of age, who was a very light drinker. About once a year he would go on a drunken spree and he always had an epileptic attack during the continuance of this condition, which would usually last from three to four weeks. During the intervals between these sprees he never had any attacks. At the age of 26 he suffered from delirium tremens and died, and during this last spell of drinking he had suffered from very frequent epileptic attacks. It has occurred to me to ask the Doctor what has been his experience in this line and whether, if these cases abandon the drinking habit they get well or if some of them get well, and about what proportion.

DR. JAMES H. LLOYD, Philadelphia—I think there is no subject more obscure, nor one about which we ought to dogmatize less than the subject of the etiology of epilepsy. I have listened to the paper of Dr. Crothers with a good deal of interest. I think we may admire Dr. Crother's skill in psychologic analysis and I believe his large experience in this class of cases entitles him to respect and deference; yet in some ways I hardly feel like following him in all that he has said. In the past I have had relations with hospitals containing large numbers of epileptics; I have seen a great number of them, but I have never quite assured myself that there was any distinct form of epilepsy that could be called alcoholic epilepsy. I grant, of course, and we all know, that there are plenty of epileptics who are alcoholics; that there are plenty of epileptics who drink to excess, who get drunk; but that we have a distinct class of epileptics whose disease bears a relationship to excess in alcohol is a question, about which, as I say, I will not speak dogmatically, but about which I have always felt very doubtful. My feeling has been that we should regard alcohol as an exciting cause of epilepsy, a contributing cause, but not necessarily the essential cause. There are a great many things that will cause a convulsive attack; for instance, uremic poisoning, the poisoning which occurs in the pregnant woman, an injury to the brain, the poisoning of scarlet fever or the effects of some other diseases; all these act as exciting causes of convulsions, but we do not say that we have a uremic epilepsy; that we have a scarlet fever epilepsy, etc. We know that these factors act on the nervous system and produce epileptic attacks. My belief is that that is practically what we have in the cases of alcohol producing these epileptic attacks. In other words, I think if you take a man who is perfectly normal, who has no distinct tendency toward epilepsy, you can not make an epileptic out of him by making him drunk. If this could be done, I think a good many more of the American population would be epileptics. You can not make epileptics out of inebriates, but you can make inebriates out of epileptics; and that is a very different thing. There is a tendency in certain forms of epileptics to become inebriates; it is a part of their degeneration; it is a part of their run-down nervous system. After the onset of the epileptic attacks, they may take to drinking and the epilepsy in that case acts as a predisposing cause of their drinking, as much as alcoholism is a producing cause of epilepsy. It is hard

to say which has occurred; it is hard to decide which began first. I think we must consider this matter carefully and not dogmatically. This whole subject has to be settled with the greatest accuracy and care, and I am not certain that we have yet at hand the method or the knowledge that will permit us to say positively that we know what is the cause of epilepsy. I feel that this is an important question, because, as neurologists assembled here, we do not want to put ourselves on record too absolutely; we want to leave our minds open in reference to this whole subject.

DR. J. M. KENISTON, Middletown, Conn.—In reply to the question asked by Dr. McBride, I might state that in Middletown we have had a number of cases where the epilepsy has arisen in the victim of chronic alcoholism, and in these cases the symptoms did not disappear on the removal of the alcohol. The seizures are apt to be rather infrequent, from 4 to 10 yearly, some of the cases having remained without attacks for a year or more. My experience has been somewhat limited in these cases, and has been confined entirely to male epileptics. I feel from my experience that there is rather more of a tendency to dementia in this class of cases of epilepsy than in any other, and that moral indifference is one of the inevitable sequels of alcoholism. The onset of all my cases occurred in late middle-life.

DR. A. B. RICHARDSON, Washington, D. C.—I agree that the class of cases the Doctor's paper describes are found in the experience of all of us, and the only question I shall raise is in regard to the interpretation of them, and in regard to the erection of this new form, or rather special form of epilepsy called alcoholic epilepsy. I think he will agree with me that the class of cases he describes arise also from other causes as well as from the excessive use of alcohol. Take, for instance, the acute forms of the disease—they are frequently produced by other causes and, in fact, I recall but very few cases where they could be attributed to excessive use of alcohol. Now, I think it is always better to keep our classification of disease just as simple and practical as possible. In any case addicted to the use of alcohol, especially where the use has extended over a long period of time, occasionally you will have convulsive attacks. Should all of those attacks be called epilepsy? I do not suppose that the Doctor would agree that they should be so called. As the last speaker has stated, convulsive attacks, of course, occur in a great many diseases. The excessive use of alcohol acts as an irritating cause in some manner or other to produce these epileptiform attacks. I think we all agree that occasionally the excessive use of alcohol may produce a typical epileptic attack. But we have a great many other causes also, and you may as well call every one of these a typical form of epilepsy. The question is whether, as our knowledge is at present, the condition which he has described is peculiar to this cause of the disease. I think we ought to restrict the term "epilepsy" to that form of the disease which is permanent in its manifestations and frequent in its occurrence, and not apply it to convulsions which come on from irritating causes now and then.

DR. WILLIAM J. HERDMAN, Ann Arbor, Mich.—I think we all recognize the fact that chronic alcoholism may play a part in the production of these epileptic convulsions, just as it acts as an agent in the production of dementia. There is in all these cases due to chronic irritation a mental reduction which undoubtedly is structural in character. Now, as has just been said, that reduced structural condition can be brought about by many other causes than alcohol. We recognize a great many other causes producing it aside from alcoholism. It would have been very interesting to me if the Doctor had added to his paper some information concerning the previous history of these cases. Some of them were evidently cases of acute alcoholism; others were cases of chronic alcoholism, but if in all of them he could have given us evidence of hereditary alcoholism in their ancestors, it would have enabled us to arrive at conclusive decisions along the lines with which we are all familiar. These cases of chronic alcoholism in many cases terminate in epileptic manifestations. I do not recognize in the description he has given of these cases that they were different in any particular from the epileptic attacks due

to other causes and, therefore, that does not justify a separate classification, and as has just been said, if we should put into a separate classification each case of the condition according to the factors producing it, we would have a great many more classes of epilepsy than necessary.

DR. RICHARD DEWEY, Wauwatosa, Wis.—We know that it is a striking fact regarding this class of cases (of which I have happened to see a large number) that in some cases the epilepsy or any convulsive manifestations have not been present, so far as one can learn, at any other time, except in conjunction with alcoholic excess. There seemed to be no convulsive attacks except when the individual was heavily under the influence of alcohol, and the number of attacks was frequently very small, in some cases occurring but once or twice in a lifetime. Then again there are other individuals who have never used alcohol to excess who have had but a single attack in a lifetime, or perhaps may have had two or three attacks in a lifetime; this makes the matter more obscure. One other point: the cause to which Dr. Crothers has referred, of a toxic effect being produced by the use of an inferior class of spirits, especially wood alcohol, would not apply to all cases, for I have seen cases largely where the patient used only a very good quality of spirits.

DR. F. SAVARY PEARCE, Philadelphia—The subject is most interesting and I will simply detain you for a few moments in regard to the treatment of these attacks. I believe that a good many of these attacks or epileptic phenomena are due to congestion of the brain itself and, even when the patient is in an acute alcoholic state, he has, of course, a congestion of the brain. I had under my care only a few days ago a gentleman who has a very good past history in every way. He was suddenly taken during the recent warm spell with a typical attack of epilepsy. I saw him when he was in the third convulsion which he had had during four consecutive hours. The sphincters were intact except in the third convulsion; and it was a typical case of epilepsy as regards all three of the convulsions. I promptly bled the man, withdrawing over a pint of very dark blood, which seemed to be the most rational form of treatment. The man improved at once and in a week he was apparently well. I believe that this form of treatment is the proper method whether alcohol is the cause of the attack or not. In this condition the brain congestion itself at the moment is responsible for the epileptic phenomena.

DR. HUGHES—I want to ask the Doctor if he will, in his reply, kindly explain what he means by the statement that the treatment of one of his cases was changed to the bromids and then bromidism followed, resulting in death. Then, I would like him to explain what he means by the term alcoholic epilepsy being a new one to the literature. I have in mind the fact that Esquirol, before I was born, claimed that many of the cases of epileptic insanity are due to alcoholism, and in discussing the subject of epilepsy he speaks of the excessive use of alcohol as being one of the causes of the development of a form of epilepsy associated with insanity. Then, the conditions of double consciousness mentioned and the automatic states are also referred to this same cause. In dementia, what we call "alcoholism" now, but which was called "inebriety" then, was commonly known as the cause of this condition. I would like to know the number of cases in which the epilepsy developed where there was reason to suppose that there was epilepsy in the family and the manifestations were brought out by the alcoholism, and the other cases where it was produced solely by the alcohol. In the case that I mentioned there was no history, and when the drinking subsided the epilepsy did not recur. I think that Dr. Crothers has had a peculiar kind of experience; he has had an experience with alcoholic epilepsy without the insanity and this is especially valuable from the standpoint of insane asylum physicians.

DR. T. D. CROTHERS, Hartford, Conn.—In my regard, alcoholic epilepsy is simply a name and not a distinct disease. We do not know anything of the pathology of epilepsy and can only speak of it as a form of degeneration possessing symptoms more or less uniform. There is no true division of

these symptoms in the alcoholic from other cases, because there is such a multiple of causes. We can only take the excessive use of alcohol as one of them. It is not a distinctly new term, but it has many symptoms which seem to be peculiar and common to it. Most of these symptoms indicate a toxic condition. Epilepsy is often a toxic and convulsive state. Alcoholic toxins and other causes are likely to produce this state. We know that epilepsy is one of the active causes of insanity. The term alcoholic epilepsy is practically new. Ten years ago the term was seldom used, while to-day it is frequently mentioned. Many of the toxins may produce symptoms which resemble alcoholism. Epilepsy, alcoholism, morphinism, dementia, consumption, sudden collapse and hemorrhage of the brain may all follow from the presence of toxins. The symptoms will change widely. There are some cases which could be attributed solely to the use of alcohol, but even here some long predisposing causes are at work, usually some diseased state of the neurons, some former degeneration existing before the attack. Such persons nearly all die from acute nephritis or cerebral hemorrhage. Then, it is another point of clinical interest, that is that the use of the more toxic alcohols produce symptoms which resemble epilepsy. These are active causes which in modern times are very prominent. Concerning convulsive alcoholism, convulsive epilepsy and convulsive disease of the brain, it is a striking fact that these symptoms are becoming more common, and I think that a great many of them can be directly attributed to the use of the mixed drinks to which I have referred. If the police court records of drunkenness with convulsive symptoms were examined, many of them would be explained by the term alcoholic epilepsy.

THE PATHOLOGY OF CHROMIDROSIS.*

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The name chromidrosis implies an anomalous secretion from the sudoriferous or sweat glands characterized by colored perspiration. Its occurrence is exceedingly infrequent; Foot,¹ in a careful review of the literature dating from 1709 to 1868, has been able to enumerate but 38 cases, to many of which a doubtful character must necessarily be attributed. Fowie's² careful historical review, 1709-1891, does not add many well-defined cases, and surprisingly few have been reported in the literature of recent years. Its marked infrequency can be best appreciated by referring to the Van Harlingen's³ statement, that "it is so rare as to be a curiosity rather than a disease."

Those who have written on the subject, almost without exception, are of one accord as to nature, and attribute to the condition a disorder, functional or otherwise, of the sweat glands. This is especially evident in all the text-books of skin diseases, where the subjects are not alphabetically arranged and follow some system of classification (Hyde,⁴ Hardaway,⁵ Morrow,⁶ Kaposi,⁷ Duh-ring,⁸ Joseph,⁹ Stelwagon,¹⁰ Shoemaker,¹¹ Schamberg,¹² Fox,¹³ Gottheil,¹⁴ etc.); in these the affection is invariably classed with the anomalies of glandular secretion, namely, the sudoriferous glands, and is grouped with hyperidrosis, anidrosis, bromidrosis, uridrosis, hemati-drosis, etc. In those which follow an alphabetic arrangement an anomaly of secretion is, as a rule, directly attributed, or indirectly implied (Jackson,¹⁵ Van Harlingen,¹⁶ etc.), apparently a few (Lesser,¹⁷ Mracek,¹⁸ Bulkely,¹⁹ Neumann,²⁰ etc.), studiously refrain from

committing themselves. It is surprising that some of the earliest observers (Nelligan, Wilson and Gintrac) have attributed the condition to a stearrhea, and Turenne to a metastasis of the eye pigment, facts which seem not so remarkable when we consider that the early investigators were very keen observers and relied almost exclusively on their powers of personal observation.

The color of the secretion varies with different cases, green, blue, purple, black, brown, yellow, red and intermediate shades. Black is the most common occurring in one-half the cases reported by Foot. Cases characterized by red pigmentation are generally associated with a reddish incrustation of the adjacent (usually axillary) hairs, the bacteriologic examination of which reveals the condition to be due to a *Zooglea* (*Bacillus prodigiosus*), which grows in rose-red colonies and often gives a history of direct contagion. These cases can not be properly classed with the so-called chromidrosis, but belong, by nature, with greater propriety to the class of so-called vegetable parasitic affections of the skin.

In my own clinical and private practice I have noted eight cases of this so-called red chromidrosis during the

Fig. 1.—Hyperkeratosis in opening of hair follicle. Accumulation of pigment in rete, P and P'; in corium, P', and stratum corneum, P'', of immediate neighborhood. (Winckel, oc. 2, obj. 5.)

past two years, in most of which there was not only a reddish discoloration of the incrustated hairs, but also distinct reddish discoloration of the superimposed garments. If this class of cases is included, chromidrosis is by no means a rare affection, and it probably falls to the lot of nearly every close observer to note these cases from time to time. At present I have, among others, two patients under observation with this form of affection, an uncle and nephew, the latter of whom is a practicing physician, who has taken up his residence with his uncle during the past four years. The physician states that his infection is of five years' duration, and that his mother, with whom he then resided, is similarly affected, and has had her affection for almost the same length of time. His uncle acquired the affection almost four years ago, and more recently his daughter has also been infected. The axillæ in all the cases have been involved, and all the cases show the red discoloration to a marked extent. From these and other cases in the literature it is clearly evident that these forms of reddish discoloration are readily contagious, and owe their cause to a local infection, entering from without. Their para-

* Read at the Fifty-third Annual Meeting of the American Medical Association, in the Section on Cutaneous Medicine and Surgery, and approved for publication by the Executive Committee: Drs. W. T. Corlett, L. Duncan Bulkley and W. L. Baum.