REMARKS UPON CLINICAL PHASES

OF

POISONING BY ALCOHOL.

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"CHINESE historians affirm that wine in which the feathers of the Tchin are macerated becomes a deadly poison."* Modern science confirms this clinical observation, but ascribes the fact to a more constant and less fabulous cause. Alcoholic liquors are poisonous because they contain alcohol. Since the various intoxicating beverages in use consist essentially of dilute ethylic alcohol, their toxic effects may be considered in common under the comprehensive title of alcohol-poisoning.

From the universality of the use of these beverages by all races† of men and in all ages, it would at first sight appear that alcohol might be a valuable food. So it was considered by Liebig; whose views were, however, entirely controverted by Perrin, Lallemand, and Duroy, who showed that it was excreted by the lungs, the kidneys, and the skin without undergoing oxidation in the body. Later observers, notably Subbotin, have shown that the ingestion of alcohol does not increase the excretion of carbonic acid, but, on the contrary, reduces it; does not raise the daily amount of urea, but lowers it; does not increase the temperature of the body, but positively reduces it; and does not increase hæmatosis, but, on the contrary, interferes materially with the blood-making function. Such effects are certainly not those of a food, but of an agent which directly impairs nutrition. Anstie and Dupré declared that a certain amount of alcohol disappeared in the passage through the system, and believed that this comparatively small amount was assimilated as food. Parkes and Wollowicz subsequently determined the maximum amount that could be thus assimilated by a healthy man to be from one and a half to two ounces of absolute alcohol per diem.‡ The latter observers found that when an amount of liquor corresponding with only one ounce of absolute alcohol was administered, no alcohol could be detected in the urine. Upon repeating these experiments, however, I found, in an adult of temperate habits, that so small a quantity as one-fourth of the above gave decided evidence of its presence in the urine; and I believe Anstie also detected alcohol in the urine after a single glassful of sherry. Possibly, this may be explained by idiosyncrasy, but it seems more probable that the person experimented upon by Parkes and Wollowicz had by habit acquired the power of consuming a greater quantity than could be assimilated by an organism unaccustomed to its use.

I am, therefore, forced to the conclusion that alcohol is not a true food in the sense that it favors nutrition in a state of health. Of its value as an accessory food under certain emergencies I shall not speak at present, as it is not germane to our subject; nor shall I enter upon a consideration of the dietetic use or abuse of alcohol. I have broached the subject of its use in health merely in order to establish, if possible, the commencement of its toxic effect. From personal experiment I have concluded that the standard adopted by recent experi-

* I. Klaproth, Lettre à M. Humboldt sur l'Invention de la Boussole, p. 89. Quoted by Thompson in "The Philosophy of Magic," vol. i. p. 41, London, 1846.

† The Mohammedans and Hindoos have been cited as exceptions to this rule because such use is forbidden by their religion; but no one will claim that therefore drunkenness is entirely unknown among them.

‡ The equivalent of alcoholic beverages for a healthy adult, as stated by Parkes, is as follows: two fluidounces of brandy, or five ounces of the strong wines (sherries, etc.), or double the quantity of the weak wines (clarets and hocks), or twenty ounces of beer. "If these quantities be increased one-half, one and a half ounces of absolute alcohol will be taken, and the limit of moderation for strong men is reached." Parkes's Principles of Hygiene, 5th ed., Philadelphia, 1878, p. 298.

menters of one ounce and a half to two ounces of absolute alcohol is rather above the average amount which can be assimilated by the healthy organism, unless given in frequent and very small doses. Alcohol is eliminated slowly from the system, except when excessive amounts have been taken; it then rapidly appears in the urine, in which traces of it usually continue for from sixteen to twenty-four hours. In one case, within ten minutes after drinking about four ounces of whisky, a quantity of limpid urine was passed, whose specific gravity was only 1001½; it evidently contained considerable alcohol.

Of the fact that alcohol, in certain amounts, is capable of causing death, there can be no question whatever: on this point all writers upon toxicology are agreed, and numerous cases might be cited in proof. Tardieu, for instance, records a medico-legal case where a man was induced by a wager to drink within a short time a bottle of brandy. He accomplished the task, fell to the floor in a state of coma, and died in sixteen hours.*

In its concentrated forms, when taken into the stomach, alcohol may cause death from local action upon the mucous membrane, producing softening and abrasion and violent gastro-enteritis. Two tablespoonfuls of sixty per cent. strength have proved sufficient to cause the death of a child.† As in other remedies, different effects may be expected when exhibited in the single toxic dose, in the interrupted dose, and in the continued dose. For convenience of discussion, although not exactly synonymous, let us consider the clinical accidents arising from the first two under the head of acute alcoholic poisoning, and those of the latter under chronic alcoholism. In so doing we shall follow the dictum of Magnus Huss, who first called the attention of the profession to the symptoms of chronic alcoholic poisoning under the title of alcoholism.‡

Let us first consider the effects of acute alcohol-poisoning, where coma, with complete muscular resolution, exists. Since this condition often terminates fatally, it is important to distinguish it from insensibility from other and widely different causes, such as cerebral apoplexy, hysteria, uræmic coma, opium-poisoning, concussion of the brain, and similar conditions. Dr. H. C. Wood says that "when the patient is simply seen in the advanced stage of deep coma an absolute diagnosis cannot be made out."§ I think, however, that by attention to the objective conditions we may make a tolerably certain diagnosis by exclusion. Without taking up each of these different diseases with which alcoholism may be confounded, let me say, in brief, that, while in this condition coma and complete muscular prostration may exist, there is no difference between the two sides of the body as regards muscular tonus or surface-temperature; the latter is below rather than above the normal. More especially is it to be noted that there is no hemiplegia. The pupils are not constant; generally they are moderately contracted; before death they may dilate. There is no conjugate deviation of the eyes, as often occurs in apoplexy and injuries of the brain. Hemorrhage into the pons Varolii, however, is said to be accompanied by contracted pupils, coma, and general muscular resolution. The pupils in cerebral hemorrhage are usually irregularly dilated. MacEwen, of Glasgow, recommends the alteration which occurs in the pupils when the patient is disturbed as a reliable sign of alcohol-poisoning. The pupils in such a case will dilate temporarily if the patient be shaken or his beard pulled. This merely proves that the condition is rather one of deep stupor than of complete coma. I cannot believe that a case of true coma would have a mobile pupil, or, if it should dilate under such circumstances, that it could afterwards again contract.

I have not mentioned as distinctive the test commonly relied upon, *i.e.*, that furnished by the alcoholic odor of the breath. Where it exists it furnishes confirmative rather than conclusive evidence, since the individual may have taken a stimulant just before the attack of illness, or it may have been administered afterwards. It thus becomes a nice question to decide in some cases whether it is alcohol or disease, or both. A much more reliable test is furnished by the urine. In all cases of narcotic poisoning the bladder, as a matter of routine treatment, should be evacuated by a catheter. The urine may be tested for alcohol by adding to it a small quantity of chromic acid test (potassium bichromate one part, acid sulphuric three hun-

* Clinique sur l'Empoisonnement, p. 848. Paris, 1867.
† Deutsch's case, Schmidt's Jahrbücher, Bd. xxv.; from Preuss. Vereinzeitung.
‡ Die Chronische Alcoholkrankheit, Stockholm, 1852.
§ Therapeutics and Materia Medica, 3d ed., p. 125.

dred parts), as proposed by Anstie. But I would recommend the adoption of a slight modification, which has yielded very satisfactory results in the few cases in which I have tried it, as follows. In a medium-sized test-tube about a drachm of clear, colorless sulphuric acid is placed, and about three times the quantity of the urine to be tested is then to be poured down the side of the test-tube, so as to prevent immediate mixing of the fluids; a small crystal (split-pea-sized) of potassium bichromate is then dropped into the tube, which is then given a gentle rotary motion, so as to dissolve a little of the bichromate and diffuse it through the sulphuric acid. The tube is then set aside in the rack. The limpid lower stratum of liquid will in the course of from a few minutes to an hour assume a decided emerald-green coloration, the depth of the color being proportionate to the amount of alcohol. This test may be confirmed by fractional distillation, provided that enough alcoholized urine can be secured to render it practicable.

In attempting to differentiate these cases from those of apoplexy, it should not be forgotten, however, that alcohol stands in a direct causative relationship to cerebral hemorrhage, as autopsies have proved. In seven cases of death during acute alcoholism, Tardieu* found two with hemorrhages into the lungs, meningeal apoplexy in six, and in four there were also ventricular effusions of blood. This authority concludes that "in death coming on rapidly during the state of drunkenness, pulmonary apoplexy, and especially meningeal apoplexy, are lesions which, if not constant, are at least extremely frequent and almost characteristic." Hughlings Jackson reported a case in which alcohol impregnated not only the breath but the urine, and at the post-mortem examination a large clot was found covering nearly the whole of one hemisphere. The question of treatment of these cases I reserve for discussion farther on.

In other cases of acute poisoning by alcohol, convulsive symptoms supervene, and occasionally epileptiform seizures are very marked, probably owing to the action upon the medulla oblongata of blood deficient in oxygen. I have seen but one case of this kind, which occurred in a mulatto some years ago. After taking from the arm about ten ounces of extremely dark blood, the convulsions ceased and did not return. The next day the man was apparently as well as usual and ready for another spree. In this connection I may also be permitted to mention a case of catalepsy in a slender young man, about nineteen years of age, who came under my care some eight years ago, while resident physician in the Pennsylvania Hospital. He was brought to the institution perfectly unconscious and perfectly rigid. I remember there was some difficulty in getting him out of the carriage, as his body was fully extended, and he was carried in supported by his heels and his head. I found that on forcibly placing his limbs in any position they would remain thus extended in the air as if they were frozen. Upon lifting the closed eyelids the eyes were seen to be rolled upwards and constantly in motion (nystagmus); the pupils were dilated moderately. It was said that this condition came on after drinking a single glass—his first glass—of whisky, and that he was not subject to such attacks. The application of faradic electricity quickly restored the patient to consciousness, and he would gladly have gone home at once if he had been permitted; but he was kept in bed until morning, and then discharged perfectly well, no further treatment being required.

I now come to speak of a form of acute poisoning from the abuse of alcohol, with which all are familiar, the so-called mania a potu, or delirium ebriosum. I will not, therefore, weary you with any reference to cases. Let me say, however, in a word, that the distinguishing trait of this condition is that it occurs as a result of an overindulgence in alcohol in an organism unaccustomed to its use: it is the form that appears in men who go on occasional sprees, with periods of temperance or total abstinence between. Such cases have active delirium, characterized by delusions and homicidal tendencies. The condition is characterized by great nervous and vascular excitement, the face is flushed, the eyes bright, the ego elevated. The diagnosis and prognosis are based solely upon the history of alcoholism, for to all intents and purposes the patient is suffering from acute mania. When this condition is compared with chronic alcoholic poisoning and, in

* Observations médico-légales sur l'Etat d'Ivresse considéré comme Complication des Blessures et comme Cause de Mort prompte ou subite. Ann. d'Hyg. Publique et de Méd. Légale, tome xl., 1848, et Dict. de Méd. Prat., *loc. cit.*

its characteristic form, delirium tremens, a marked contrast is seen to exist. This condition, as originally described by Sutton, is essentially one of nervous and vascular depression. The face is pale, the eye dull, there are illusions in place of delusions, a suicidal tendency takes the place of a homicidal, and melancholy replaces mania. Sleeplessness exists in both, but in one it is due to cerebral congestion, in the other to cerebral exhaustion. Tremor of the muscles is a marked symptom of the latter, and gives it its name.

I shall not here attempt a detailed consideration of the various diseased states that are associated with chronic alcoholism, since many of them are only indirectly due to it. In the words of Boehm,* "The poison of alcohol, either alone or combined with other pathological causes, produces bodily or mental diseases which in themselves afford nothing characteristic of the effects of alcohol." That it is a most fruitful source of disease, both physical and mental, all authorities are agreed, the tendency of alcohol being to cause fatty degeneration and sclerotic changes in all the soft tissues of the body. Nor shall I essay the enumeration of the various diseases attributed to alcohol; in the words of Bartholow, they may be summed up as "sclerosis and steatosis."

The point, however, that I would now insist upon, and which I consider has a most direct bearing upon the treatment of alcoholism, is this: the long-continued existence of alcohol in the blood produces important changes in nutrition, to which the system in a measure accommodates itself, so that the patient requires less food to support life than without the alcohol (as in a case quoted by Anstie,† where a tailor drank a bottle of gin daily for years, and who took in addition a small piece of bread each day as his only sustenance). In such cases it cannot be doubted that alcohol plays the rôle of an accessory food, and changes take place, converting the organism into an alcohol-burning apparatus, and correspondingly unfitting it for the ordinary carbo-hydrates and hydro-carbonaceous food. This will, I think, serve in a measure to explain why depriving a drunkard of his drink may cause a sudden failure of nutrition with the rapid appearance of an outbreak of delirium, denotive of cerebral exhaustion, and characterized by failure of mental power, hallucinations, prostration, and muscular tremor. Let me repeat the fact that in health, if at all, only very small amounts of alcohol are consumed by the system, but in chronic alcoholism the tissues have undergone such changes as to confer upon them the power to derive force from alcohol, which unfits them to a corresponding extent for normal nutrition.

The term delirium tremens was adopted by Sutton in a work published in 1811, in which he took especial pains to show that the symptoms were not due to phrenitis or meningitis. He showed also that bleeding and blistering were generally fatal, and that these cases need a supporting treatment, and especially opium.

Strangely enough, in "Ziemssen's Cyclopædia" (*loc. cit.*), Prof. Boehm, in referring to Sutton's work,‡ gives this author due credit for originality in the use of the term delirium tremens; but, on the other hand, he states that it was reserved for Rayer in 1819 to indicate alcohol as the prime factor in the etiology of the disease. It is quite evident that he had not read Sutton's tract on Delirium Tremens, or he could not have said that this author had overlooked its connection with alcoholic excess. Here is what Sutton, after reporting several cases, says:§ "It has been remarked in several of the above instances that the parties attacked by delirium tremens have been given to drinking; and I feel firmly persuaded that all cases of this disease are connected with indulgences of that nature." Could language be more plain and unequivocal than this? Again, he says,‖ "But that fermented liquors, and more especially spirits, are the general cause of the disease, is rendered certain by the frequency of it in situations where the indulgence of them can be had at a reasonable rate. On the coast of East Kent, where I was first led to distinguish this affection, and at the time alluded to, spirits brought in by smugglers might be had in great abundance at a cheap rate; and such as labored under delirium tremens in that quarter were mostly those who confessedly indulged in the use of spirits to excess."

Several varieties of delirium tremens

* Ziemssen's Cyclopædia, Am. ed., vol. xvii., New York, 1878, p. 400.

† Stimulants and Narcotics, London, 1864, p. 451.

‡ Tracts on Delirium Tremens, on Peritonitis, and on some other General Inflammatory Affections, and on the Gout. By Thomas Sutton, M.D. London, 1813, p. 2.

§ Loc. cit., p. 47.

‖ Loc. cit., p. 50.

have subsequently been indicated by industrious investigators which we cannot now consider, such as febrile delirium tremens, which, according to Magnan, generally runs a fatal course in a few days, and is probably connected with some local inflammation, such as meningitis. Nor need I discuss the uræmic form of Surmay.* Epileptic insanity may at any time occur as a complication. Dipsomania is undoubtedly a psychosis, often inherited, sometimes due to traumatism or severe mental shock, not necessarily dependent upon previous alcoholic excess. The clinical forms of confirmed alcoholism known to the alienist—such as pachymeningitis hæmorrhagica, general paralysis, melancholia, and hopeless insanity (hopeless because dependent upon sclerotic and fatty changes with atrophy of the brain)—are conditions belonging directly to our subject, but which need not at present engage our attention.

Let us, however, in conclusion, consider very briefly the therapy of the three forms most commonly encountered by the general practitioner,—*i.e.*, alcoholic coma, mania a potu, and delirium tremens,—a subject which has given rise to a good deal of controversy. In the first place I will consider coma. In rapidly-fatal cases of alcoholic poisoning, failure of respiration commonly occurs previous to cessation of the heart's action. Therefore, besides the ordinary treatment of narcotic poisoning by the use of the stomach-pump and purgative enemata, and the application of warmth to the extremities, it will be proper to fortify the action of the respiratory centres by the hypodermic administration of (gr. $\frac{1}{120}$) atropia with (gr. $\frac{1}{12}$) morphia, repeated at proper intervals. The urine should be drawn off, both for examination and to encourage the action of the kidneys. It may also be necessary to employ electricity and artificial respiration to assist the lungs in excretion of the surplus of carbonic acid, which now tends to accumulate in the blood. Indeed, Sampson, an English physician, in the treatment of such a case found himself obliged, as a last extremity, to resort to tracheotomy, which proved successful.†

Reginald Southey,‡ in a recent lecture on this subject, deprecates active measures, because if the case should happen to be one of hemorrhage into the pons Varolii, instead of one of alcohol-poisoning, such treatment would be improper; but since the cases of hemorrhage into the pons must be quite rare compared with those of alcohol-poisoning, and since they are invariably fatal under any method of treatment, I regard them as entirely out of the question, and should treat such a case as one of alcoholic coma. As soon as the patient has his stomach emptied by the stomach-pump, a pint of hot coffee may be thrown into its cavity and allowed to remain. Inhalations of ammonia will greatly assist in reviving the patient. An individual supposed to be insensible from alcohol should never be allowed to remain in a state of coma to sleep off a fit of drunkenness. Too often it proves his last sleep, either from carbonic acid poisoning or from secondary cerebral hemorrhage.

Acute mania induced by alcohol is commonly subdued by ether- or chloroform-inhalations and hypodermic injections of morphia. In such cases I have known a grain of morphia to be administered with only good results. The standard prescription for the minor forms of acute alcoholism is one containing bromide of potassium and chloral in decided doses, given every two hours until sleep is obtained. Where there is much arterial tension, the tincture of hyoscyamus or hyoscyamia has been highly recommended. Care should also be taken to give nourishment frequently, in a form easy of assimilation on account of the possible gastric inflammation. The patient will be found more manageable when confined to his bed, and it often becomes necessary to strap him down. During convalescence stomachic tonics to improve the digestion may be given. For this purpose occasional laxatives and tincture of capsicum, tincture of nux vomica, and compound tincture of cinchona may be used with advantage. To diminish the appetite for strong drink, a freshly-prepared fluid extract of cimicifuga with tincture of capsicum may be steadily employed, as recommended by Bartholow.

I will not dwell upon the treatment of delirium tremens further than to say that it requires pre-eminently a sympto-

* De quelques Formes peu connues de la Cachexie alcoölique, etc., L'Union Médicale, pp. 19–21, 1868.

† Article Alcoölisme, Nouveau Dict. de Méd. et de Chir. Pratiques, Paris, 1864.

‡ London Lancet, December 18, 1880.

matic and supporting treatment. Such cases generally suffer from numerous other evils, among which we notice prominently chronic gastric catarrh, owing to local action of the alcohol upon the mucous coat of the stomach causing atrophy of the peptic glands and increase of submucous connective tissue. Foods, therefore, which are digested in the small intestine, and peptones, are particularly required.

If I have correctly stated the conditions existing in acute and chronic alcoholism, I think it will be seen that good reasons exist why the administration of alcohol as a part of the routine treatment is as necessary in true delirium tremens and in chronic alcoholism as it would be improper in acute alcoholic poisoning. I am fully aware that the routine administration of stimulants is not uncommon in these cases, —which is partly due, I think, to the fact that these states are often confounded clinically under the common title of alcoholism, although their different pathology is insisted upon by almost all the text-books. Then, if we separate clinically the effects of acute alcoholic excess from the condition of chronic alcohol-poisoning, with or without delirium, I believe that we are in a position to institute a rational treatment for mania a potu and delirium tremens.

To summarize:

1. Acute alcoholic poisoning, manifesting itself in the forms of coma, convulsions, and mania a potu, is characteristic of the physiological action of alcohol upon a system unaccustomed to its use. Its treatment, in cases of coma and convulsions, is like that of the other narcotic poisons producing paralysis of the respiration, but in mania powerful cerebral sedatives are required. During the after-treatment alcohol is not necessary, but, on the contrary, every encouragement, both by precept and by prescription, should be given the patient to adopt total abstinence as his only chance of safety.

2. Chronic alcoholic poisoning, exhibiting itself in the form of the horrors, vigilance, delirium tremens, or melancholia, on the contrary, bespeaks the existence of a depressed condition of the vital powers due to saturation of the system with alcohol, and consequent degenerative changes. Such unfortunate cases, suffering from what might be called an alcoholic diathesis, require careful nursing, a supporting treatment, and the continuance of stimulants, which to them have become both food and drink.

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