AN ADDRESS ON THE MORBID ANATOMY AND PATHOLOGY OF CHRONIC ALCOHOLISM.

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By JOSEPH FRANK PAYNE, M.D.,
Vice-President of the Society; Physician to St. Thomas's Hospital.

MR. PRESIDENT AND GENTLEMEN,—When the Council of this Society did me the honour to ask me to open the discussion on this occasion, I accepted the task perhaps with a somewhat inadequate appreciation of the vast extent of the field which now lies opened out before me. It is only since I have been endeavouring, to the best of my ability, to prepare for the duty devolving upon me, that I have thoroughly realised the multiplicity and variety of the facts which have to be dealt with, and the extreme difficulty of compressing them into a moderate compass.

I must, therefore, ask your indulgence while I appear before you, not in the character of an explorer bringing home the results of travel in new and untrodden ways, but rather in that of a geographer trying to sketch a rough outline map of a country hardly as yet completely explored or surveyed.

The extent of our country, however, though wide, is not unlimited. We are not going to discuss the action of drinks on the human body generally, nor all the diseases to which habits of excess can give rise, nor, still less, the moral or economical consequences of such habits, but solely the material changes which the use of alcohol in excess has been actually shown to produce in various tissues and parts of the body.

HISTORICAL INTRODUCTION.

First Period: Ancient Medicine.—Before entering on the main topic, I will, however, ask your attention for a few minutes to some rough notes on the history of the subject. This begins in comparatively modern times, for ancient medicine knew almost nothing of morbid anatomy. Doubtless, so long as wine has been known, so long has there also been known excess in wine; and among the Greeks even, more especially among the Romans, such excesses were neither rare nor harmless. Hence we find scattered notices in the ancient medical writers showing that the symptoms of inebriety were observed, and some graphic descriptions and some good clinical rules, founded on these observations, might be quoted; but, so far as I know, there is nothing in any ancient writer bearing on the morbid anatomy of the disease.

Second Period: Rise of Morbid Anatomy.—This is, of course, equally true of mediæval medicine, on which, therefore, we need not linger. It was not till the sixteenth century that morbid anatomy began a separate existence as a science, first under the protection of her elder sister, normal anatomy; gradually, in the seventeenth century, assuming a more independent position. But among the many excellent scattered observations on the structure of diseased organs, with which the medical literature of this century abounds, there are very few bearing on the action of wine or strong drinks.

As an instance of the most advanced knowledge of the age on such subjects, we might well quote our own Harvey, not only as a man of genius, but as a skilled physician well versed in all the learning of his time. In Harvey's MS lectures, lately published in *facsimile*, I have found nothing directly referring to inebriety, but there are certain observations which would naturally have led to this being spoken of had its importance been recognised. For instance, in speaking of the anatomy of the liver, he describes twelve different cases or examples of diseased liver which he had himself seen; one was "russet, hard, contracted, absque sanguine," which seems like a small cirrhotic liver. Another, according to the rough notes, was "russetish, ingentem et durum, plane scirrhus tumour, absque fere sanguine, aspera superficie;" a large, hard liver, evidently like a scirrhus tumour, almost bloodless,

and with a rough surface, which could hardly have been anything else than cirrhosis. He also says that such livers are found in cases of dropsy (fol. 39). In another place he discusses dropsy generally, as being said by Fernelius to depend upon the liver, but himself inclined to the opinion that the dropsy is the cause of the morbid change in the liver rather than the contrary. Dropsy may arise, Harvey says, by drinking more than the kidneys can get rid of, so at length even the fleshy parts are turned into water; but so long as any of the liver remains, the patient will live and not die.

This seems to bear upon our subject, but, really, I think it is founded upon the common belief at that time that dropsy could be caused by drinking too much water; a fault which consequently the bold spirits of the time were much on their guard against. Altogether these notes make one regret the loss of the observations on morbid anatomy which Harvey is said to have collected.

Harvey's MS was written in 1616. Not long after this some notices of alcoholic diseases begin to appear, but the only lesion referred to this cause by writers of the seventeenth century was cirrhosis of the liver, and its consequent ascites. The earliest case of this kind which I can find is of the date 1626, though published many years later in the great storehouse of such observations, Bonet's *Sepulchretum* (Geneva, 1679), where it is quoted from Gregorius Horstius. A German nobleman, of intemperate habits, suffered for three years from jaundice, and was found to have the liver hardened, as was evident to the touch. He improved under treatment, but having "indulged his genius" for several weeks, he was attacked with epistaxis, followed by dropsy of the belly and scrotum. The latter was punctured, and two measures (? quarts) of fluid escaped, but the abdominal swelling did not diminish, and after some months he died suddenly. On opening the body ten measures of fluid were found in the abdomen. The liver is described as "scirrhusum et induratum, neonon exsiccum instar ligni putredine corrupti," that is, scirrhus, hardened, and juiceless, like rotten wood. The mesenteric veins were filled with gelatinous material surrounded by clotted blood, which could be pulled out to the length of an ell and a half.¹

Surely this, as a clinical and pathological picture, could hardly be improved. A hard liver, jaundice, ascites, epistaxis, and sudden death with thrombosis of the portal system—scarcely any important feature is wanting.²

Several cases might be quoted from the same work, but I will only give one more.

A French soldier, returning from the wars in a thirsty mood, came upon a pool of stagnant water, where he first bathed, and of which he then drank a mighty draught (*haustus Germanicus*). This he took the first opportunity of correcting with abundant libations of strong wine, and the remedy was so often repeated that he fell into a severe fever, followed by dropsy of the abdomen and legs. The belly was tapped repeatedly, and in all 168 ounces of fluid were drawn off, of which the curious observation was made that, first and last, it always smelt like the stagnant pond-water of which the patient had so incautiously drunk. After death there was still some water in the peritoneum, and the liver was found as hard as wood.

In these cases and others strong wine was clearly recognised as the source of evil, but other causes were not excluded, as Bonet records the case of a boy who contracted fatal scirrhus of the liver and ascites by eating too many figs. Then there is another curious history of a certain glutton who ate as well as drank to excess, and whose liver was found after death of an enormous size—clearly an instance of the other type of alcoholic liver, the fatty, such as we might find now in a robust and plethoric brewer's drayman.

English medical literature of this period yields few valuable observations. One by Walter Harris, the correspondent and friend of Sydenham, author of a book on the diseases of children, and of *Pharmacologia Antiempirica* (London, 1683), may be worth mentioning. In the work just mentioned he relates the case of a gentleman, aged 36, who had brought himself to an insensible necessity of drinking sack several times a day until he fell into an irrecoverable consumption. On opening the body after death the liver was found "so thoroughly boiled with constant heat that a sound liver could not well be more boiled over the fire than his was by the use of sack. It was in colour and brittleness the very

¹ *Sepulchretum*, p. 1052.

² In the same case there is a very good description of xanthelasma in the form of tubercles on the knees and elbows, etc.

same as a long-boiled liver can be." Although dropsy is not named, probably it is meant that there was ascites also. Harris observes that sack does much more harm than French wines; and, though the reason he gives for this is a strange one, it shows that the causation of cirrhosis was beginning to be understood.

There is still one observation of dropsy connected with liver disease which I will venture to quote, although its dependence upon alcoholic drinks quite escaped the surgeon who has recorded it, one John Browne, surgeon to St. Thomas's Hospital. The account, which appears in the *Philosophical Transactions*, vol. xv, 1685, is entitled: "A Remarkable Account of a Liver, appearing Glandulous to the Eye," and is accompanied by a figure, "accurately taken down by Mr. Faithorn," an eminent artist and engraver of the day, which I have thought worth copying to show to the Society.

"The person was about 25 years of age, a soldier in one of His Majesty's regiments here in town, who contracted his distemper by drinking much water, when he could not stirr from his duty, and catching cold at nights in being upon the guard. He was under the care of our physicians for some time, by whose directions his swellings did by times abate; but afterwards it was observed, that the method which had been beneficial to others, had not here the like success, his swellings returning upon him as before, so that there was nothing more now to be thought of, but a paracentesis, which operation, however, is judged very hazardous, by reason of the time of the year, and for that the patient was very much emaciated; yet he being so much swelled, that it was uneasy to him to lie in his bed, he importuned us very often, and with great earnestness, that the operation might be performed. Whereupon, a paracentesis by the physicians consent and directions, was made by me, whereby we drew from the patient about three pints of brinish liquor, and within four days after as much more; the next day, morning, he dyes, and his death, as was found upon dissection, was partly occasioned by a mortification upon his scrotum and penis.

"This operation was performed to the satisfaction of the physicians and chirurgeons that saw it, and by it the patient had some ease for the present. Upon opening the body I believe I took out about twenty-four quarts of water; he had a large inflammation upon the peritoneum, all his other inward parts not much disaffected, except the liver. Its magnitude was not extraordinary, but seemed rather less than usual, but that which was very remarkable (and I think the like was scarce ever observed by any author) and seems much to confirm the opinion of the learned Malpighius, is this: It consisted in its concave, convex and inward parts of glands which (with the vessels) made up one whole substance thereof; these glands contained a yellowish ichor, like so many pustule, and was I suppose part of the bilious humor lodged in the same, though otherwise the liver between the glands was of its usual reddish colour. In the bladder of gall we found a soft friable stone, but otherwise nothing considerable further in that part."

A private in the Guards is hardly likely to have acquired dropsy by drinking water, and there can, I think, be little doubt that the so-called "glandular structures" were the ordinary soft bile-stained masses, separated by fibrous tissue, which we find in cirrhosis of the liver. And the figure, allowing that it is drawn in a formal and conventional manner, appears to me to represent the same. However, it is left to the judgment of the Society; but as being, if I am right, the first published figure of cirrhosis of the liver, it has some historical interest.

Third Period: Use of Distilled Spirits.—We have now seen what the seventeenth century contributed to the morbid anatomy of alcoholism. A new period was now about to begin, that in which distilled spirits came into use as a beverage. With this, new and more severe kinds of alcoholic disease began to appear,

macies et nova febrim
Terris incubuit cohors.

and, but for this disastrous invention, I am convinced that our programme for discussion to-night would be very much shorter than it is.

It was at the beginning of the eighteenth century that distilled spirits began to be generally drunk. The art of distillation, of course, was very much older, and was practised in London at least in the sixteenth century, if not earlier; but, up to the time now spoken of, distilled spirits were chiefly used in medicine, and were for a long time very costly. But towards the end of the seventeenth century they became much cheaper, so

that in 1678 a pint of Nantes brandy cost about sixpence. When the use of such drinks became common I do not know, but in 1724 we find the College of Physicians making a public representation as to the evils of spirit drinking. At this time gin was so cheap in London that a person could intoxicate himself for one penny. A duty was imposed in 1736, and other laws made to check the practice of spirit drinking. The name of the Rev. Stephen Hales, the physiologist, ought to be mentioned in a medical discussion for his efforts in this cause. It is also clear that alcoholic diseases increased, and were more observed by the medical profession, but the morbid anatomy of the subject was little advanced, at least in England, till the end of the eighteenth century. Erasmus Darwin made some good observations on the symptoms of drunkenness, but has only contributed to morbid anatomy in the statement that pigs fed on grains from distilleries get diseased livers.

Baillie described cirrhosis under the name of tubercles of the liver, and declared it to be a disease *sui generis*, different from scirrhus.

J. C. Lettsom first noticed some of the symptoms of alcoholic paralysis, and James Jackson, of Boston, America, gave, in 1822, a very good account of a disease resulting from the use of ardent spirits, which he calls arthrodynia, which is evidently the same.

But all the knowledge of this period is summed up in the classical work of Magnus Huss on *Alcoholismus Chronicus*, translated from Swedish into German in 1852. Huss describes very carefully the morbid changes of all parts of the body met with in drunkards; but, with regard to the nervous system, it is noteworthy that he regarded the disturbances of these parts as being unaccompanied by any change in structure, and hence as being symptoms of a certain kind of poisoning. It is to these that he applied the name, then used for the first time, of chronic alcoholism, and some of his descriptions have left little to be added to.

Fourth Period: Era of Pathological Histology.—The most conspicuous advance on the subject since the work of Huss has been the demonstration of minute changes in various parts of the nervous system affected by alcoholic disease. At the same time the histology of other organs has been studied, the identity of the minute changes found in different parts has been demonstrated, and gradually the uniformity of the action of alcohol throughout the whole body has become clearly manifest. We are now able to look upon alcoholism as a form of poisoning, and to compare it with the action of other poisons, especially with metallic and other so-called irritants. The history of alcoholism must come to an end here, for it would be difficult to separate it from the exposition of different branches of the subject.

ALCOHOL AS A POISON.

I have now to consider the question, In what sense is alcoholism a poisoning; or, in other words, in what sense is alcohol a poison?

Since we must here evidently look upon poisons in a somewhat wider sense than a purely medico-legal one, perhaps I may be permitted to quote a pathological definition of poisons which I have given elsewhere:—"A poison is a substance capable of injuring the body, either by causing damage to the tissues or by producing functional disturbance." On this basis I divide them into two classes, namely, tissue poisons and functional poisons. I will take the latter first. A functional poison disturbs the mode of action of the tissue elements without permanently altering their composition. Hence their action is transitory, and ceases when they are eliminated from the body. Most functional poisons are called narcotic or neurotic, because their most conspicuous action is on the nervous system. But some such poisons—for instance, opium—affect animals without a nervous system, and possibly even plants, so that their action is general. Substances like quinine, which affect protoplasm, though without showing any special predilection for the nervous system, and hence sometimes called protoplasmic poisons, are also included.

But it may be worth while to say that I do not use the term poison as a term of unqualified condemnation. It only means something capable of producing injury, not necessarily doing so. All metallic salts, nearly all drugs, and many substances used as food are in this sense poisons; but we do not on that account deny their usefulness when properly employed. It would be as absurd to condemn alcohol as to condemn common table salt because a large dose of either of them may be fatal. But to pursue this

subject further might be dangerous; I return, then, to the old track.

It is quite clear that alcohol is a functional poison of the narcotic class. Its action on the brain shows the gradations of stimulation, overaction, inhibited action, and actual narcosis. These effects, unless positively fatal, are transitory. But it is also clear that this is not the whole of its injurious effect; since, if the functional disturbance be often repeated, the brain itself will come in the end to be damaged. But it may be supposed this damage is caused by the excess or repetition of the functional disturbance. Such an explanation will not, however, apply. Some organs, such as peripheral nerves, are damaged, in which no functional disturbance from the immediate action of alcohol can be traced. Hence we conclude that alcohol is also a tissue poison damaging the structure of the tissue elements. This effect is not seen after a single dose, even a fatal dose, at least so far as is known, but only after repeated action of the poison. When its action is perceptible it is quite comparable to that of the so-called irritant, especially metallic, poisons, such as lead, arsenic, antimony, etc., with phosphorus, and even mineral acids. It is now recognised that these substances, if absorbed, act on all or most tissues of the body which they reach in proportion to the degree of concentration in which they may be present, and to the susceptibility of the different parts. This is also true of alcohol. It is carried by the blood to all parts (having been detected in the brain and various organs), and acts most powerfully in the first instance on the parts which it reaches with the least amount of dilution, that is, the stomach and liver. In the second place, it acts on the nervous tissues as being more vulnerable than the rest. Again, the influence of concentration in relation to the tissue damage is seen in the fact that alcohol in a dilute form injures the tissues much less than the same amount in a concentrated form, though the narcotic effect may be the same.

Another law of tissue poisons is that they all have, within certain limits, the same action, or at least there are certain modes of action common to all. These common modes of action belonging to all tissue poisons I will endeavour to state, and then see whether alcohol acts in the same way.

The first effect of such poisons is seen on the more vulnerable or parenchymatous elements, namely, nerve, epithelium, muscle fibre. On these parts their action is essentially necrotic, producing, if in a low degree, parenchymatous degeneration; in a higher degree, actual necrosis, though if the injury be not too severe, repair is possible. This is true of arsenic, antimony, sulphuric acid, and, with some modification, of phosphorus. I would submit that this is also true of alcohol, which produces degeneration, or ultimately necrosis, of mucous membrane of stomach, liver-cells, nerve-fibres, nerve-cells, and muscular fibres.

Another effect of all tissue poisons, if sufficiently concentrated, is to injure the blood-vessels, causing exudation and cell migration—that is, inflammation as generally understood. It is obvious that this is true of all irritant poisons. If chronic, this inflammation sometimes results in hyperplasia of connective tissue. I submit that concentrated alcohol acts on the stomach, for instance, to which it is directly applied, in the same way: setting up acute inflammation. The action of alcohol, however, is never quite so intense as that of some metallic poisons, since it never produces suppuration, nor has it an actually corrosive action. Its continuous or chronic action is to produce connective tissue hyperplasia, fibroid changes, or cirrhosis.

Besides these two modes of action, alcohol has one almost peculiar to itself: that of causing accumulation or infiltration of fat in various parts of the body, especially where such accumulation naturally takes place, as in liver, omentum, subcutaneous tissue. Phosphorus is like alcohol in this respect, and so is, to some extent, arsenic. This change may be called "steatosis." It is explained, apparently with reason, as due to deficient oxidation or impeded cell-respiration, the alcohol or phosphorus being oxidised in place of the fat which should be burnt up in the cell. I would ask, are there any objections to this explanation?

I would suggest the relations of fatty infiltration to true fatty degeneration as an interesting though difficult subject of inquiry and discussion. The difference in well-marked conditions is obvious. A liver cell or connective-tissue cell loaded with fat may be healthy, while a fattily degenerated cell is one of which the protoplasm is already altered in structure. But is it not possible that accumulation of fat may destroy the cell in the end, and thus pass into degeneration? If the respiration of a cell is sufficiently impeded, will the cell die as an individual does under

the same circumstances? Since alcohol appears to cause true fatty degeneration of some parts, as well as mere adipose accumulation in others, the question is particularly interesting here. This action of alcohol can hardly be called poisonous since it may aid in nutrition.

To sum up. The action of alcohol on tissues or tissue elements is threefold: (1) as a functional poison; (2) as a tissue poison or destructive; (3) as a checker of oxidation; and in these respects it may be paralleled by other substances called poisons, and by others which are generally considered innocuous. These views of the action of alcohol are put forward with the object of inviting criticism or correction. It is very probable that they may be in some respects one-sided or faulty.

I had intended to give some account of the morbid changes produced by administering alcohol to animals, but find that time will not permit.

(To be continued.)

ON THE OCCURRENCE OF HÆMATOMA OF THE EAR IN FOOTBALL PLAYERS.

By F. A. SOUTHAM, M.B.Oxon., F.R.C.S.

Assistant Surgeon to the Manchester Royal Infirmary.

THE traumatic variety of hæmatoma auris (an effusion of blood into the auricle, occurring in healthy persons as the result of the application of direct violence to the ear) is not infrequently met with in those who take part in the game of Rugby football.

Hinton¹ states that Dr. Farquharson (formerly medical officer to Rugby School) has observed this condition in boys after football, and Burnett² also makes a brief allusion to the same fact. Erichsen,³ in alluding to Hinton's observation on its occurrence in football players, attributes it to "hard hits on the ear." The injury is not usually the result of a blow on the ear with the football, but is produced in a "tight scrimmage" by the pressure and friction exerted by the arms and bodies of the other forward players on the ears and side of the head, when the latter is bent downwards and forwards, as is so often the case with many forward players. Probably a factor which may assist in its production is the congested condition of the vessels of the head and neck, the result of the severe straining efforts which accompany the attempt of an energetic player to take the ball through a "scrimmage." So common is this affection among those who take part in Rugby football, that I am informed it has now become the custom of many forward players to wear a tightly-fitting cap with lateral flaps in order to protect the ears.

In most cases the effusion is slight, and becomes absorbed in the course of a few days without any special treatment. It is, however, not at all uncommon for the extravasated blood, probably mingled with a certain amount of inflammatory exudation, to become organised and develop into fibrous tissue. The latter result is most likely to occur if, as often happens, the individual continues to play, and pays no attention to the swelling of the ear. The consequence is that the upper portion of the auricle, the part usually affected, remains in a thickened and indurated condition, eventually assuming an irregular, shrivelled up appearance, owing to the process of contraction which, after a time, gradually takes place in the newly formed cicatricial tissue. In other cases, suppuration is excited at the seat of injury, a similar deformity being produced after the pus has been evacuated and healing has taken place. This is well shown in the figure from a case recently under my care. The patient, a strong healthy man, aged 20, informed me that, about six months previously, he played forward in a closely contested game where there were some very "tight scrimmages." The same evening he noticed that the upper part of his left ear was much swollen in front, the swelling being of a dark blue colour. He showed it to a medical man, who opened it, and let out a small quantity of fluid blood. He played again several times during the following month, and on each occasion the ear became considerably swollen. Eventually it inflamed and was again opened, some pus being evacuated on the second occasion. After this it gradually shrank, and contracted until it assumed the appearance shown in the figure. As he was anxious to have the deformity removed, I separated the skin and

¹ *Aural Surgery*, 1874, p. 70.

² *A Treatise on the Ear*, p. 256.

³ *Science and Art of Surgery*, ninth edition, vol. 3, p. 1024.