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PART I. ORIGINAL COMMUNICATIONS.

ART. IX.—*Diabetic Coma, with some Remarks on the Pathology and Treatment of Diabetes.* By JAMES ALEXANDER LINDSAY, M.A., M.D., M.Ch.; Assistant Physician to the Belfast Royal Hospital.

I HAVE recently had the opportunity of observing three cases of diabetes terminating by coma, and I think them worth putting on record, as the facts may tend in some slight degree to enlarge our knowledge of an interesting and important condition which has lately attracted great attention :—

CASE I.—On Monday, May 21st, I was asked to look at a patient (Samuel W.) in the extern department of this hospital. He complained merely of weakness and languor, and was manifestly much emaciated. A few rapid questions elicited no information of importance, and a careful examination of the thorax was equally barren of result. Acting on an excellent clinical rule, which I owe to Dr. Stephen Mackenzie—viz., “In cases of marked emaciation without obvious cause think of phthisis and diabetes”—I proceeded to examine the urine for sugar, and was not surprised to find it loaded. The patient was admitted to hospital, and put upon an effervescing alkaline mixture, the tendency to languor and drowsiness discouraging treatment by codeia.

The history elicited was as follows :—The patient was thirty-three years of age, and had served for ten or twelve years as a soldier in Gibraltar, Malta, and China. For the last three years he had been a worker in an iron foundry. He had been a free liver, and frequently drank to excess. Three months previous to admission he began to lose

flesh, but it was not until some time subsequently that he noticed any polyuria. During this time his appetite was excessive, and he began to suffer much from thirst.

The chief features in the case were marked emaciation, dry skin, great weakness, and considerable mental torpidity. There were no signs of disease in the lungs. The breath had an ethereal odour.

The patient survived just one week after admission. During this time his general condition underwent no change of importance. The quantity of urine passed varied from 120 to 160 oz. daily.

On Sunday, May 27th, he became worse, and was confined to bed. He seemed heavy and stupid, refused food, but drank eagerly. His friends were warned that his state was extremely grave. Next morning I did not see him until 11 o'clock, when I found that well-marked coma had gradually supervened. He lay back in bed with the eyes half-open, and occasionally swung his arms listlessly about. The breathing was peculiar. The inspiration was normal in character, but the expiration was greatly prolonged, and consisted of a series of jerky panting gasps. The pause was much longer than normal. Respirations 12 per minute. The breath was quite cold and still retained the ethereal odour. The pupils were normal and responded to light. The pulse was very fast and almost imperceptible. The skin felt very cold, and the thermometer in the axilla registered only 96.5°. Some urine was drawn off by a catheter and examined; it was light in colour, had a specific gravity of 1034, contained a large quantity of sugar and a trace of albumen. Ferric chloride gave first a curdy white, and then (on shaking up) a dirty claret-coloured precipitate. Some blood was obtained; it appeared quite normal both to the naked eye and under the microscope. About 4 o'clock the patient revived slightly, opened his eyes, looked round, and swallowed some milk. Temperature still 96.5°. At 7 o'clock he was profoundly collapsed. The respirations were still about 12 per minute, but expiration was now short, and the pause proportionately prolonged. Temperature as before. The patient died at 7 40 p.m.

CASE II.—Samuel A., aged twenty-two, was admitted to hospital on February 24th. Patient was a lamplighter. A few days before Christmas he slipped and fell, coming in contact with a stone, which struck him on the right groin. Five days subsequently a swelling appeared in each lumbar region—that on the right side being the larger and more painful. He drank cream of tartar freely, and the swellings disappeared, the pain also subsiding. Soon after, however, he began to suffer much from thirst, and noticed that he was passing large quantities of urine. At the end of a fortnight his thirst disappeared, but he began to lose flesh rapidly, and his strength began to fail. Patient was much emaciated, and stated that he had lost 30 lbs. during the previous two

months. He complained chiefly of weakness and frequency of micturition. He felt some pain in the back as he lay in bed, and there was some tenderness on pressure in both groins. His expression was dull and heavy. The urine was of a pale straw-colour, very acid; sp. gr. 1033; much sugar was present, but no albumen; quantity, 6 pints. Codeia, 1 grain three times daily, was ordered.

On the 27th February the patient was manifestly worse, being much prostrated. The codeia was stopped, and stimulants ordered. About 1 o'clock, p.m., the patient gradually sank into a state of coma, from which he never rallied until the time of death, which occurred at the end of ten hours. During the coma the pulse continued fair, the breathing was stridulous, the face pale, the skin cold, and the pupils much contracted (probably from the effect of the codeia).

CASE III.—Alfred C., aged twenty-three, was admitted to hospital on March 15th; but, as he left it again almost immediately, there was unfortunately no record taken of his case. As far as I can recollect, the following is a brief outline of the history:—The patient was a distinguished student, and attributed the onset of his attack (which occurred a few weeks previous to his admission to hospital) to severe study in preparation for one of the higher university examinations. The quantity of urine was large, and the specific gravity high, but I have no exact record.

A week or ten days after leaving hospital the patient fell into a state of coma, and died in a few hours. Before admission to hospital, and after his departure therefrom, he was treated on the skimmed-milk principle without drugs; and, according to his own statement, he found this treatment effectual for the relief of thirst, though it exercised no influence on the progress of the disease.

The above cases illustrate several important points with reference to diabetic coma, which may now be regarded as fairly established:—

1. As regards its *frequency*.—It so happens that during the present year *all* the fatal cases of diabetes at the Royal Hospital, Belfast, have terminated in coma. This is, no doubt, an accidental circumstance, but, nevertheless, the fact is striking and significant. At the London Hospital, in the series of 37 fatal cases recorded by Dr. Stephen Mackenzie, 19 terminated in coma; and Dr. Frederick Taylor has recorded 53 fatal cases at Guy's Hospital, of whom 33 died comatose. We shall probably be within the mark if we conclude that at least *one half* of all the fatal cases of diabetes end in coma. In view of this fact it is

remarkable that this termination of the disease was almost entirely overlooked by the earlier writers, and it becomes a question alike of clinical interest and practical importance whether death by coma in diabetes has become more frequent since the treatment by codeia has been popularised. To this important topic I shall presently recur.

2. As regards the class of cases in which death by coma is to be specially apprehended, it is relatively much more frequent in the young. The ages in my three cases were, respectively, thirty-three, twenty-two, and twenty-three—average age, twenty-six. Considering the frequency of diabetes in the middle-aged and the old, and the great chronicity which often marks its course, this preponderance in the case of the young is sufficiently striking.

3. Coma is a much more frequent termination in acute than in chronic cases. The average duration of the disease in the above three cases was under three months, while the average duration of ordinary cases of diabetes terminating in various ways is probably rather more than a year.

4. Coma is said to be more frequent in those cases which present no trace of pulmonary disease—a fact to which Dr. Stephen Mackenzie has drawn special attention. There was no evidence of any lung affection in any of the cases I have recorded, though careful examinations were made with the view of detecting some pulmonary lesion.

The chief features which distinguish diabetic coma are the peculiar form of dyspnœa which has been aptly compared to that seen in a healthy person after violent exercise—coldness and lividity of the skin, usually combined with great rapidity of the pulse. Most authorities agree that the ethereal odour is usually absent from the breath; but it was well marked in one of my cases.

When we come to inquire into the pathology of diabetic coma, we are confronted with a number of conflicting theories, each supported by a mass of more or less plausible *post mortem* evidence. The whole question, indeed, of diabetes has been a sort of *corpus vile* for the exercise of much ingenuity and acumen on the part of pathologists, with the result that we are still far from the attainment of certain knowledge. Let me briefly summarise the various views which have been held regarding the causation of diabetic coma.

It has been attributed to—

(1.) Some form of toxæmia, either

α. Uræmia,

β. Acetonæmia, or

γ. The presence of some other toxic agent in the blood, possibly aceto-acetic acid.

(2.) The presence of fat embola in the minute pulmonary vessels.

(3.) Dehydration of the blood.

(4.) Some lesion of the sympathetic system.

It would be easy to multiply such theories, but I shall rest content with enumerating the above.

The uræmic hypothesis may be dismissed without further consideration, the clinical and pathological evidence being strong against it.

The theory that diabetic coma might depend upon fatty embolisms has been a favourite one since the discovery that in some cases of coma the blood has a milky appearance, and contains some molecular matter bearing a great superficial resemblance to fat. But the theory fails, unfortunately, from two points of view. In the first place, it has been shown that this milky appearance of the blood is rather an exceptional condition—the blood being frequently normal in its naked-eye characters, and sometimes thick and dark-coloured. In the second place, fatty embolisms have been proved to exist in many conditions unattended by coma, or indeed any symptoms at all. Thus they are constantly present in cases of fracture of bones, and even in cases of mere contusions of the soft parts; and, so far from causing coma and death, they are apparently productive of no morbid process whatever.

The theory advanced by Dr. Hilton Fagge—that dehydration of the blood is the cause of diabetic coma—deserves to be treated with some respect, as such a condition of the blood is both *à priori* probable in diabetes, and has been proved to exist in many cases, its appearance being sometimes described as thick and black, or “tarry.” It is not so clear how such a condition of the blood could produce the symptoms of coma, but we can conceive several ways in which this might be effected. Against Dr. Fagge’s view is the admitted fact that the blood in diabetic coma presents no constant features, the “tarry” condition being as exceptional as the milky appearance on which so much stress has been laid by other authorities.

The view that the sympathetic system is at fault rests rather on theory than on evidence, and we are thus thrown back upon, on

the whole, the most probable view—viz., that the coma is due to the presence of some toxic agent in the blood, whether acetone, aceto-acetic acid, or some other product of alcoholic fermentation which has hitherto eluded observation. The evidence is still inconclusive, but this is the line which observers are now mainly following with the best hopes of a successful issue.

As regards the prognosis in diabetic coma, I am not aware of any case of recovery being on record. Death seems to supervene in less than 24 hours, and often after a very much shorter period.

Probably few attempts at treatment have been made, the manifestly moribund condition of patients after the onset of coma discouraging any active interference. Intra-venous saline injections were tried by Hilton Fagge in one case with some slight temporary advantage. The marked fall in surface temperature which has been frequently noted would indicate the propriety of trying the effect of warmth to the skin, warm water enemata, &c.; and the fast weak pulse might lead us to try alcohol, ammonia, or digitalis. So far as I have observed the patients retain the power of swallowing, which renders treatment rather less hopeless than it would otherwise be.

Leaving the question of diabetic coma, I shall now make a few remarks on the pathology and treatment of diabetes in general, not in the hope of advancing anything new, but rather of defining the exact point at which we have now arrived. A recent discussion at the Pathological Society, though highly interesting, served mainly to show how far we still are from any exact knowledge of the *vera causa* of diabetes. Cases were brought forward exhibiting a vast variety of morbid lesions affecting almost every organ, but none of these were shown to be constant or diagnostic. The discussion was mainly useful in so far as it served to clear the ground for future inquiry. The kidney and the liver having been interrogated with negative results, the attention of pathologists is now mainly directed to the brain and to the blood. Dr. Dickenson sees in certain vacuolations of the nerve centres which he has described an adequate explanation of the phenomena of the disease, while Dr. Pavy is not less convinced that the true cause is some chemical defect in the blood. The *post-mortem* evidence being so extremely conflicting, one would be disposed to attach great relative importance to any points in the history of cases of diabetes which might seem likely to throw light on the ætiology of the disease. In one of the cases which I have recorded there is a very clear history of

the onset of the disease occurring immediately after an accident. It is always our duty to sift such histories with exceeding care, and to be continually on our guard against the "*post hoc, propter hoc*" fallacy. In the above case, however, the history is so clear that it is difficult to doubt that the accident in question determined the onset of the disease, and I can support this view by another precisely similar case, where also an attack of diabetes followed immediately upon a slip on the ice. I have records of two cases where diabetes followed alcoholic excess, and in another case it was alleged to have supervened on recovery from a "feverish cold." I mention these facts mainly for the purpose of accumulating information on the question, and without venturing to determine what value (if any) is to be attached to them.

On the subject of the treatment of diabetes I should wish to speak with all reserve. There are few diseases where such conflicting results are alleged to have followed the administration of certain drugs in the hands of different observers, some regarding codeia almost as a specific, while others find that in the great majority of cases it is not only impotent to check the disease, but that its administration serves to aggravate the patient's symptoms and materially augment his sufferings. My own experience of the drug is disappointing. In only one instance have I seen apparent cure follow its administration, and this was a mild case, which might possibly have yielded to the restricted dietary which was also enforced. In several instances I have seen codeia productive of nothing but mischief, the disease continuing unchecked, while the most obstinate constipation and very distressing headache attended its use. I have already alluded to the very important question whether death by coma has become more frequent since the treatment of diabetes by codeia was introduced—the evidence seems to be, so far, against such an assumption. In two of my three cases of coma, codeia was not administered at all, and in the third case it was continued for only two or three days. On the other hand, numerous cases are on record where codeia was given in large doses for long periods without the advent of coma. Strangely enough, codeia seems to tend as often towards insomnia as narcotism.

The treatment by large quantities of skimmed milk, without medicine, has now been extensively tried, and with varying results. It soon becomes grateful to the patient, relieving his thirst and increasing his comfort. I have seen one case of average severity where this treatment was signally successful, the patient improving

steadily, with a speedy recovery of flesh, and being finally discharged cured. In some cases I have seen much relief and some slight benefit follow the free administration of effervescing alkaline mixtures.

Finally, I have seen the alleged specific, "Bethesda Water," tried in a single case without any benefit whatever.

From the above facts no conclusions of importance regarding the treatment of diabetes can be safely drawn. Codeia has been so highly praised and seems to succeed so admirably in suitable cases, that it will probably retain for some time its present position as the favourite therapeutic agent. The skimmed milk treatment is praised by some and denounced by others. Probably some of the milder cases of diabetes will yield to dietetic treatment alone, others benefit by codeia, and others again by skimmed milk. It seems impossible at present to determine beforehand the therapy most likely to succeed in any given case. The more severe and acute cases of diabetes do not seem to be amenable to any of the present methods of treatment.

ART. X.—*Case of Hemiplegic Epilepsy following Fracture of the Skull.* By CHARLES B. BALL, M.D., F.R.C.S.I.; Surgeon to Sir P. Dun's Hospital, &c.

FOR the notes of this case of hemiplegic epilepsy following fracture of the skull, I am indebted to my dresser, Mr. W. Kiddle:—

CASE.—M. Y., aged thirty-six, a well-built, powerful man, who had been a policeman, but was dismissed the force for intemperance, and was now by occupation a car-driver, was admitted into Sir P. Dun's Hospital on Friday, April 27, 1883. I had seen him that morning at his lodging, where I elicited the following statement of his case:—On the previous Monday (23rd inst.) he was driving a car at Donnybrook, when the horse stumbled and he fell off on his head. He was stunned for a few minutes, but then got up and drove his fare back to Jury's Hotel, where, after imbibing some of the national restorative, he said he felt quite well. On Tuesday, Wednesday, and Thursday, he was about at his work as usual, but on Friday morning he was found insensible in his bed by some neighbours who went to see why he had not come to his work. He lived in a room by himself. Shortly after he was found it was stated he had a fit.

Upon admission his head was shaved, and during the process he had a convulsion which affected the left side of the face and arm only. A