which occurred in the growth and nutrition of animals. By looking closely at the phenomena of life, it would be seen that it involved a balance and alternation of protoplasmic and ferment action. In man the starch taken as food was converted into sugar by the saliva and pancreatic juice, absorbed, and carried to the liver, where it was brought under the protoplasmic action of the liver cells, which reduced it to glycogen—a process of dehydration. In the vegetable kingdom transmutation in the direction of dehydration might be observed in the production of cellulose and starch, which were end-products. In the animal kingdom an instance might be given of the production of a carbo-hydrate as an end-product, since it was present in the texture basis of the test or outer investment of the tunicata. But in the animal kingdom this was an exception; as a rule, carbo-hydrates were not end-products, but were utilised in the production of fat or proteid. That carbo-hydrates were thus utilised for the production of proteids was proved by the fact that the yeast plant could grow in Pasteur's liquid.

PASTEUR'S LIQUID.			
Ammonium tartrate $(\text{NH}_4)_2\text{C}_4\text{H}_4\text{O}_6$	...	...	1 part
Cane sugar $\text{C}_{12}\text{H}_{22}\text{O}_{11}$	...	...	10 parts
Ash of yeast	...	...	1 part
Water	...	...	100 parts

THE ORIGIN OF PROTEIDS BY INCORPORATION OF CARBO-HYDRATES.

In the multiplication of the yeast plant there was a production of fresh protoplasm, and therefore of fresh proteid which, under the special conditions of the growth of the yeast plant in Pasteur's liquid, must be derived from the incorporation of the sugar with the nitrogen of the tartrate. If it were objected that the carbon of the newly-formed proteid might be derived from the tartrate, the answer could be made that the tartrate in the fluid might be replaced by ammonium nitrate (NH_4NO_3) , and the yeast would still grow. This afforded a demonstration that the carbo-hydrate (sugar) was used in the construction of the proteid.

The current opinion among vegetable physiologists was that in the deposition of cellulose and starch there was, first, an incorporation of the sugar with a proteid, and that then from this newly-formed body starch and cellulose were cleaved off. The proteid body with which this incorporation took place was probably asparagin $(\text{C}_4\text{H}_7\text{N}_2\text{O}_3)$ —a crystallisable and therefore diffusible principle, which existed largely in the vegetable kingdom. This body, under the influence of protoplasmic activity, was caused to become incorporated with the sugar as a new complex proteid body, from which the starch was cleaved off.

CARBO-HYDRATE DERIVED FROM ANIMAL PROTEIDS.

Dr. Pavy then proceeded to sketch the steps by which he had been led to the conclusion that a carbo-hydrate could be obtained from the cleavage of a proteid body by chemical means available in the laboratory. Finally, he arrived at a method of treating egg albumen which yielded a carbo-hydrate body readily soluble in water, yielding no coloration with iodine, and possessing no cupric-oxide reducing power.

This was obtained by whipping white of egg, and pouring it by degrees into a large capsule of boiling water acidulated with acetic acid. The coagulum thus obtained was washed, squeezed, and placed in a flask with a solution of potash. (For the whites of 12 eggs 20 grammes.) It was then kept on the water bath for two or three hours, or set aside till the following day, with, in either case, an occasional shaking. To the liquid thus obtained potash was added to bring up the proportion of potash to 10 per cent. The flask, fitted to an inverted receiver, was then boiled for half an hour. The liquid was rendered faintly acid with acetic acid, filtered, concentrated by evaporation, and poured slowly into alcohol (85 to 90 per cent.). The material separated out in a finely divided form, and on the following day was found to have settled into a gummy mass. (*The Physiology of the Carbo-hydrates*. London: 1894. Pp. 33-34.)

This body, which in its physical and certain of its chemical properties resembled the "animal gum" described by Landwehr, was by the action of mineral acids converted into a material which reduced cupric oxide. Emil Fischer, during the past decade, had prosecuted valuable researches founded upon the observation that with phenyl-hydrazine cupric-oxide reducing sugars formed osazones—compounds possessing definite crystalline characters. Dr. Pavy exhibited several of these osazones, and also a series of photo-micrographs shown with the lantern. He pointed out some of the differences observed in the characters of the crystals, and observed that the nearer the sugar from which the osazone

was derived was to glucose the more acicular were the crystals. Finally, he exhibited the osazone obtained from the cleavage product of egg albumen, and demonstrated its crystalline character in a lantern slide.

A FURTHER NOTE ON THE RETURN OF THE KNEE JERK IN A TABETIC PATIENT AFTER AN ATTACK OF HEMIPLEGIA.

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THROUGH the kindness of Dr. D. M. Taylor, at present in charge of the infirmary attached to Stepney Union, one of us (J. T.) had recently (February 6th, 1894) the opportunity of making a new examination of the patient B., whose case we described in the *BRITISH MEDICAL JOURNAL* of July 11th, 1891. This was a case of return of both knee jerks in a tabetic patient after an attack of right hemiplegia. The following is the note made at this recent examination.

B., aged 55. The patient is almost constantly in bed. He occasionally gets up, and with the help of another patient is able to walk somewhat feebly, but without any one-sided weakness, and without ataxy. He is almost totally blind. Psychically he is dazed, and cannot be got to answer questions intelligently. He keeps repeating that he is paralysed. There are no signs pointing to "general paralysis."

As he lies in bed it is seen that he has external strabismus of the left eye. The ocular movements, however, seem to be unimpaired. The pupils are unequal, the left being the larger, and no response either to light or on convergence is made out in either. With the ophthalmoscope distinct waxy atrophy of the right optic disc and patches of choroidal atrophy in the right fundus are visible. In the left eye there is a similar condition of atrophy of the optic nerve, but the choroidal changes are much more extensive and distinct. There seems to be no perception of light by the left eye, and but little by the right. In the limbs there is no paralysis, but all movements are weak. He is able to stand, even with his eyes closed, and also to walk, but his gait is feeble although not markedly unsteady. The right knee-jerk is still present, but it is very slight, and is only obtained with great difficulty by Jendrassik's method. No knee-jerk can be obtained on the left side after repeated and persistent trials to elicit it. The testing of sensibility is unsatisfactory and inconclusive on account of the dazed psychical condition of the patient and the untrustworthy character of his answers.

In our former paper (July, 1891) we wrote as follows:

"Before the hemiplegia or before the lateral sclerosis was well established, it may be that from the sclerosis of the posterior columns, there were too few fibres left intact in those columns for strong enough or sufficiently numerous impulses to act on the anterior horns concerned, so as to produce the jerk. Upon the ensuing of lateral sclerosis, according to current doctrine, the anterior horns become more excitable. Thus it may be that after this change in the horns the few fibres left intact in the posterior columns were sufficient for action on the horns, so that the jerk could be elicited. If this be so, and if the posterior sclerosis increases, the presumption is that the jerks will be once more lost. It is not likely that the lateral sclerosis will increase, at any rate so far as the lesion causing the hemiplegia is concerned with it."

As will be seen on reference to the report of the present (1894) state of this patient, what we predicted has to a certain extent taken place. On one side (the left), on which the knee-jerk could at one time be elicited, none now can be obtained, whilst on the right side—the hemiplegic one—where it was formerly obtained with facility, it can now only be obtained with difficulty, and is very slight. It thus seems that

the posterior sclerosis has progressed whilst the lateral sclerosis consecutive to the cerebral lesion which caused the hemiplegia had already, when the case was first published, reached its maximum. Presumably the posterior sclerosis will continue slowly to increase, and it seems more than likely that the knee-jerk on the hemiplegic side also will in course of time again disappear.

Since the publication of our paper already referred to dealing with this case, several observations of interest with reference to the state of the knee-jerks in hemiplegia, more especially when that paralysis is complicated with tabes or other conditions in which the phenomenon is usually absent, have come under our notice. A short time before our paper appeared Goldham gave an account¹ of two cases illustrating the same state of things as we subsequently described. The first case was that of a man, aged 34, who had suffered from tabes for three years, and in whom both knee-jerks were absent. The patient then had an attack of right hemiplegia, and the knee-jerk returned on the paralysed side; it is not stated how soon. It remained present during the three or four months that elapsed before his death. The patient committed suicide; there was no *post-mortem* examination. The second case was that of a man, aged 57, who had suffered from tabes with loss of knee-jerks for some years. He then had an attack of loss of power affecting the left side of the body and the right arm, with unconsciousness. There was then—it is not stated how soon after the attack—great excess of all reflexes, and both knee-jerks were noted as very active.

Dr. John Ferguson, of Toronto, Canada,² has recorded two most interesting and important cases illustrating peculiarities of the knee-jerk. The first case was the converse of ours and is very valuable because of the *post-mortem* examination which was made and the changes of the cord which were found. It was that of a man who at the age of 36 had an attack of right hemiplegia. In the course of the next five years he had two more attacks, the paralysis affecting the same side. It was when suffering from his third attack that he was first seen by Dr. Ferguson. At this time his right knee-jerk was much exaggerated while the left one was only slightly more active than usual. On the right side there was also foot clonus, but this was not present on the left side. For six years after this he remained under Dr. Ferguson's observation. Soon after the third attack of hemiplegia he began to complain of paræsthesia in the feet and legs, while the knee-jerk, especially that on the left leg, gradually became weaker. In course of time there were lightning pains, ptosis and Argyll Robertson pupils, and the patient died in a laryngeal crisis. Before death the knee-jerk had entirely disappeared from the left leg and could only just be elicited in the right. The *post-mortem* examination revealed, besides a condition of local cerebral softening due to thrombosis, distinct degeneration in the right lateral and left anterior tracts of the cord and a few bundles of degeneration in the left lateral tract. In the posterior columns was the sclerosis characteristic of tabes dorsalis. This case, a case of tabes developing in a patient already hemiplegic, is, as we have said, the converse of that of our patient B. In the early condition of Dr. Ferguson's patient the state of the knee-jerks was that which is usual in hemiplegia, namely, exaggeration of both, but more especially of that on the paralysed side. But the subsequent development of sclerosis in the posterior columns presumably rendered functionless fibres in these columns which transmit impulses from the patella tendons to those anterior horn cells subserving the extensor muscles of the thighs and so abolished the knee-jerk. The condition of the cord was actually in Dr. Ferguson's patient that which we surmised to be probably present in our patient, namely, one of combined posterior and lateral sclerosis, although in his patient the lateral sclerosis was no doubt present before the posterior, whilst in our patient the posterior sclerosis was no doubt the first to occur in point of time.

The other case mentioned by Dr. Ferguson is somewhat analogous to that of our patient, although the original disease was not tabes but diabetes, in which also, as was first pointed out by Dr. Buzzard in this country,³ the knee-jerk is sometimes lost. The patient was a man who began to suffer from

diabetes in his forty-first year. During the next eighteen months the knee-jerk could not be detected with the most careful search. At the end of this time he had an attack of left hemiplegia, which is said to have developed slowly. By the end of a week a knee-jerk could be obtained on the left side, and it remained present up to the time of his death, but no jerk could ever be obtained on the right side. In this case it is to be assumed, according to current theories, that the lateral sclerosis produced by the lesion of the right side of the brain cut off the inhibitory influences passing down to cells of the left half of the lumbar enlargement which subserve the knee-jerk, thus permitting those cells to be acted on by fewer impulses from the patella tendon.

Of course knee-jerks lost in cases of diabetes may return when there is no hemiplegia, but in the case of Dr. Ferguson's patient the knee-jerk returned on the paralysed side alone. Whether in diabetes an abnormal condition exists in the nerves or whether it exists in the posterior columns of the cord is doubtful. However, Dr. Williamson⁴ has lately found sclerosis in the posterior columns of the cord in several cases of diabetes, a most important observation.

In connection with the subject of the knee-jerk in hemiplegia, we may also refer to a case recently published by Dr. Ormerod⁵ in which hemiplegia due to arterial occlusion was present. At first no knee-jerk could be obtained on either side. Some days after the onset of the hemiplegia, however, the jerk could be elicited on the paralysed side but it was never elicited on the sound side. The patient died of ulcerative endocarditis. This case is to some extent inconclusive, as it cannot be asserted that the knee-jerk was present before the hemiplegia, and the cord does not seem to have been examined. In such a case it is, of course, possible that the blood state indicated by the ulcerative endocarditis may have been such as to lead to abolition of the jerks. A careful examination of the cells of the lumbar region of the cord might have thrown some light on this point.

A CASE OF VIRCHOW'S PSAMMOMA OF THE PITUITARY BODY, WITH REMARKS AS TO THE FUNCTION OF THAT STRUCTURE.

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IN the BRITISH MEDICAL JOURNAL of January 13th, 1894, a paper appeared on the Evolution of Function of the Pituitary Body and its Relation to the Central Nervous System, by Dr. W. Lloyd Andriezen, who, after an exhaustive series of experiments showing the important trophic influence of the pituitary gland on the central nervous system, drew certain conclusions as to what symptoms should be produced by ablation or destruction (by disease or new growth) of the pituitary gland.

Since the publication of this paper, a case has come under my observation, of which I append brief notes, which bears out in such a striking way many of the conclusions at which Dr. Andriezen arrives, that I venture to publish it.

M. C., female, aged 11, was a bright and particularly intelligent child till within five months of her death she was far above others of her age at school, and very fond of reading at night.

The family history was unimportant; some members of the family were strumous, but not markedly so. Five months before death she first complained of vertical headache and dimness of vision. In three weeks she had to leave school, and was taken to the eye infirmary, when the following note was made: "Nearly blind; pupils unequal; left larger than right; both act to light, but very sluggishly; no reaction to accommodation; right eye failed first, and now has only perception of light; ophthalmoscopically nothing abnormal save a deep pigment layer outside the disc."

One week later she was brought to me in a state of great depression and apathy; her expression was vacant, and unless spoken to and bothered to answer, she would sit or lie about, taking no interest in anything. She could walk without any difficulty or unsteadiness, but was very quickly tired, and did not care to move at all unless compelled to do so. She was very thin, weighing 3 st., 12 lbs.; this loss of flesh I was told was recent. The lungs and heart were normal. The temperature was subnormal. There was complete loss of appetite. No areas of anæ-

¹ Berlin, klin. Wochenschr., 1891, No. 8.

² Alienist and Neurologist, January, 1892.

³ Lancet, vol. ii, 1835.

⁴ BRITISH MEDICAL JOURNAL, February 24th, 1894.

⁵ St. Bartholomew's Hospital Reports, vol. xxvii.