a substance whose composition is $C_{72}H_{118}N_{18}O_{22}S$, differing from Lieberkühn's formula $C_{72}H_{112}N_{18}O_{22}S$ only by six atoms of hydrogen.

I have thus endeavoured to show that albumen is a compound of cyan-alcohols united to a benzene nucleus, these being derived from the various aldehydes, glycols, and ketones, or that they may be formed in the living body by the dehydration of the amido acids; that, from a body so constituted, all the different substances may be obtained which have been extracted from albumenoid tissues; that lactic acid is obtained in two ways, either from

with the simultaneous development of CO_2 , a result which is brought about when a muscle contracts or when it dies; and that urea may be obtained from one series of cyan-alcohols with the production of a cyan-alcohol higher in the series.

Such a compound of cyan-alcohols, therefore, presenting so much resemblance in its properties to albumen cannot, I think, differ very widely (though, perhaps, not absolutely correct) from the molecular constitution of albumen.

Taking this view, then, of the constitution of albumen, the following may be given as a summary of the nutritive changes.

The amido-acids, glycocine, leucine, tyrosine, etc., in passing from the alimentary canal to the liver, are dehydrated, forming a series of cyanhydrins, or cyan-alcohols, attached to a benzene nucleus, and then pass into the circulation. In the tissues, these cyan-alcohols, partly by condensation, partly by hydration and oxidation, give rise to the various effete products which are eliminated from the system chiefly in the form of carbonic acid and urea.

There is still one other point that I do not wish to leave

unnoticed. According to Tiemann, the cyanamides $R-C \begin{cases} NH_2 \\ CN \end{cases}$ are very unstable bodies, and, with the elimination of NH_3 , very easily condense into imido-nitriles.³⁶

If, then, the force holding the cyan-alcohols composing living proteid together were suddenly withdrawn, changes would quickly take place in these unstable bodies, there would be the formation of some acid and the different cyanamides, which latter would undergo the condensation above described; the liberated NH_3 combining with other cyan-alcohols to form other cyanamides, and further condensation taking place. Does this not offer some clue to the phenomena of rigor mortis and the coagulation of the blood?

Again, by combining two molecules of $CH_2 \begin{cases} NH_2 \\ CN \end{cases}$ we should have by Tiemann's formula

which, when hydrated with weak acids, would give

a body having the same composition as aspartic acid, but differing in that it is an imido instead of an amido body. Similarly

which hydrated would give $C_3H_6(NH) \begin{cases} COOH \\ COOH \end{cases}$ a body having the same

composition as glutamic acid, differing only in structure. But by strong HCl and high temperature, these "imido-nitriles" seem always to give aldehyde, HCN and an amido acid. It is not improbable therefore that, under certain conditions, we may have

In discussing the pathology of rheumatism, gout, and diabetes, we shall have to deal chiefly with the lowest cyan-alcohol of the series, namely, $CH_2 \begin{cases} OH \\ CN \end{cases}$, and the substances derived from it. What part these play in regard to these disorders I will endeavour to show in the next lectures.

THE GULSTONIAN LECTURES, ON SPASM IN CHRONIC NERVE-DISEASE.

*Delivered at the Royal College of Physicians of London,
March, 1886.*

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LECTURE III.

II. SPASM IN CONNECTION WITH SPINAL MOTOR MECHANISMS.

IN my second lecture, I finished the remarks which I had to make on the connection between the cerebral system of fibres and the clinical phenomena of spasm, and I shall now proceed to a short consideration of the spinal system, and the relation between diseases which affect it and spasmodic contraction of muscles.

II. By the spinal system, I mean all those nerve-centres which occur between the central ganglia of the brain and the termination of the spinal cord. Each of these centres consists of afferent and efferent nerves and ganglionic cells; and as my object is to treat the subject from a general point of view, we may divide it into: 1. Spasm produced by diseases of efferent spinal nerves; 2. Spasm produced in a reflex manner by diseases of afferent nerves; and, 3. Spasm produced by diseases of the ganglionic cells.

1. *Spasm produced by Diseases of Efferent Spinal Nerves.*—A very large proportion of the cases in which spasm of muscles produces distortion are not to be explained by supposing that irritation of the diseased nerve gives rise to excessive muscular contraction. On the contrary, the healthy muscles are those actively concerned, while the diseased muscles are for the most part passive. Progressive muscular atrophy is a well known instance of this, where a peculiar claw-like distortion is produced by atrophy of the interossei and the continued activity of other muscles (Fig. 42). Here the neuro-muscular disease

FIG. 42.—"Main en griffe" in a case of progressive muscular atrophy.

appears to owe its origin to an affection of the large cells in the anterior cornua of the spinal cord. But even where no gross disease of nerve or muscle exists, it seems to be a rule that, when the origin and insertion of a muscle remain approximated for a certain time, the muscle contracts and gradually shortens. This frequently occurs in

ERRATUM.—On page 531, four lines from the bottom, for "lenticulo-striate and lenticulo-optic arteries," read "lenticulo-striate artery."

potassic cyanide, can furnish para-derivatives of benzene, namely, terephthalic acid. See Miller's *Org. Chem.*, 1880, part iii, p. 526.

³⁶ Berichte, xiv, s. 1953.

³⁶ We have an example of such a change of an imido into an amido body in the action of boiling hydrochloric acid on hydrazo-benzoic acid. See Fownes, p. 526.

joint-disease, and after prolonged application of splints. In the case of progressive muscular atrophy, the "claw" condition results mainly from atrophy of the interossei. These muscles extend the two distal phalanges, and flex the proximal; and when their influence is removed, the opposite condition prevails, owing to the action of opponents, namely, extension or superextension of the proximal phalanges, and flexion of the peripheral.

The case from which the drawing was taken was kindly sent me by Mr. Sidney Harvey. The patient was a woman aged 27, who was healthy in other respects, and the mother of a family. The disease had commenced in the left hand at the age of 17 or 18.

Another instance of a similar kind is found in the disease called "pachymeningitis cervicalis," where the contraction varies according to the part of the cervical region which is affected. If that part from which the median and ulnar nerves emanate be diseased, the muscles supplied by the musculo-spiral nerve, remaining intact, produce the form of distortion in which the wrist is extended; while, if the upper half of the cervical region be the seat of the disease, the muscles innervated by the median and ulnar nerves produce quite a different form of distortion, in which the wrist is flexed.

Great interest has been taken of late in the effects of alcohol upon peripheral nerves, and some of our own countrymen have played a prominent part in the investigation of them—Wilks, Buzzard, Dreschfeld, Hadden, and others. There is very little doubt that a multiple peripheral neuritis results from the circulation of this poison, and produces such symptoms as neuralgic pains, anæsthesia, paresthesia, various degrees of paralysis and atrophy, mainly occurring in the extensor muscles, ataxia, abolition of tendon-reflexes, vaso-motor disorders, and occasionally rigidity. Although our knowledge of this subject is not very advanced, it is probable that this rigidity is of the kind we are now considering, namely, rigidity of the more healthy muscles which overpower their diseased opponents; and, the latter being usually the extensors of the legs, rigid flexion results. Similarly, in cases of disease of the peripheral nerves and muscles on the extensor surface of the forearms produced by lead, rigidity of the opposing flexors is sometimes observed. As I have had an opportunity of making a *post mortem* examination in a case of atrophy of the nerves and muscles of the forearm from lead-poisoning, I have brought drawings which illustrate peripheral neuro-muscular disease due to this cause.

The drawings were made from microscopic preparations of the muscles of a patient, aged 48, a plumber, who was admitted into St. Thomas's Hospital under the care of Dr. Bristowe, on March 18th, 1882, and died three days after admission, from coma following an epileptiform convulsion. The patient had had several attacks of intestinal colic and of gout, and had also had "dropped hands" five or six years previously, from which he said he made a complete recovery. For the two months preceding his admission, he had been labouring under general dropsy and wrist-drop. His arteries were found to be rigid, his heart hypertrophied, and his urine albuminous, with specific gravity 1011. He had paralysis, with marked wasting of the muscles of the forearm, mainly the extensors, and also of the thenar and hypothenar muscles, and of the interossei.

At the *post mortem* examination, the heart was found to weigh twenty-two ounces, the valves being competent. The kidneys were contracted, hard, and granular on the surface, and weighed eight ounces. The common extensors of both forearms were pale and atrophied, and quite unlike the healthy muscles, the supinator longus being among the latter. The posterior interosseous nerve was soft, and greyish, while the median and others were firm and white. The microscopic sections of the extensor muscles from which the drawings were made showed the following peculiarities (Figs. 37, 38, 39, 40, 41).

Longitudinal Section.—(1) Here and there were seen well formed, large, striated muscular fibres; (2) atrophied, but striated, fibres, many extremely thin; (3) waxy-looking, non-striated fibres, some of fair size, some very small; (4) strands of wavy tissue where no muscular fibres could be distinguished, but where long rows of nuclei were seen, like necklaces, often running parallel to each other, and quite close together; (5) very thick-walled vessels.

Transverse Section showed the muscular bundles very small, each one having a homogeneous appearance, and presenting nuclei irregularly distributed upon it. The nerves were so altered as to be incapable of recognition. The sections of the spinal cord in the cervical region showed no evidences of disease. The supinator longus, as is well seen in the drawings, was practically healthy.

In all these and similar cases, the diagnosis depends upon the electric reactions of the nerves and muscles in the affected part. For, where the rigidity is directly produced by contraction of the affected

muscles, the electric conditions in them closely resemble those of health; whereas, when the distortion is due to atrophy of one set of muscles, owing to disease of the peripheral nerves supplying them, what is called the "reaction of degeneration" is found in them. Setting aside such cases as these, in which the distortion is produced by rigidity, not of the diseased muscles, but of their healthy opponents, there exists still another class, in which contracture results from the shortening of diseased muscles, it is true, but nevertheless does not depend upon true spasm. We may take as an example the following case.

FIG. 38.—Transverse section of the common extensor of the forearm in a case of lead-palsy. The darkly shaded masses in the centre (a) are the largest muscular bundles left, most having been reduced to very small dimensions (b).

Case of Distortion of Arm due to Shortening of Degenerated Muscles.—On August 11th, 1885, I saw, with Mr. Clutton, a boy aged 5, who had fallen down in April and fractured the lower end of the right humerus. He was treated with anterior and lateral rectangular splints for one month, and was then found to have loss of sensation and of voluntary power in the hand and forearm. The friends took him to Mr. Golding-Bird, who applied electricity daily for one month, without producing any improvement. When I saw the boy, his right forearm was rigidly pronated, and the wrist partially flexed. The fingers were flexed at the peripheral joints, and slightly overextended at the metacarpo-phalangeal joints. On attempting to overcome this condi-

FIG. 40.—Transverse section of the supinator longus muscle from a case of lead-palsy, showing the unaltered muscular bundles (a).

tion and extend the wrist, the deep structures along the flexor surface of the ulna became tense and hard, and, on bending up the elbow-joint, they were again relaxed. The tips of the fingers were anæsthetic as far as the proximal phalangeal joints. The thumb had not lost sensation. The question which presented itself to us was, whether this rigidity was due to muscular spasm, or to shortening of muscles, etc., from disease. Dr. Kilner tested the muscles electrically with me, and found that, while all the extensor muscles of the forearm reacted normally, the flexors could not be brought into action even by the strongest currents. The muscles were therefore clearly in an advanced condition of degeneration. To confirm this conclusion, the child was put well under chloroform, but no change whatever occurred in the limb; all was as rigid as before.

Here, no doubt, we had to do with inflammation and subsequent

degeneration of nerve and muscle, and the cicatricial contraction which accompanies it. Electricity and chloroform are our principal aids in the diagnosis of such cases.

Dr. Alliot, of Sevenoaks, kindly sent me a man, aged 65, who had dislocated his left shoulder-joint five months previously. On examination of the arm, almost complete paralysis of all the muscles was found, with but little alteration of sensation. The elbow, wrist, and fingers were all somewhat flexed, and incapable of extension; but still there was no active contraction of muscles. Even the strongest faradic current produced no effect whatever in them.

In certain cases of long standing facial paralysis, the muscles on the affected side are evidently shortened, for the angle of the mouth is drawn to that side, and the orbicularis oris partially closes the corresponding eye. And yet the effect produced by speaking or by laughing is most curious, the affected side remaining motionless. In these cases, the electric reactions of the muscles supplied by the diseased facial nerve show almost complete degeneration, which has been accompanied with permanent shortening.

If so many cases of distortion are due to other causes than active contraction of the muscles supplied by the affected nerves, the question suggests itself: Does chronic muscular spasm occur at all from direct irritation of motor nerves? Let us take some of the most frequent and familiar examples of peripheral nerve-disease. Musculo-spiral paralysis is often seen in quite the early stages; but we do not see, nor do we hear the patient describe, muscular spasm as one of its symptoms. The same may be said of most cases of facial paralysis due to peripheral causes. In chronic poisoning by lead and alcohol, various nerve-symptoms are met with, including paralysis, and even muscular rigidity occasionally; but, as has already been pointed out, this is due to the contraction of antagonists, and not of the diseased muscles themselves. In sciatica, from various causes, we meet with twitchings and sudden muscular contractions; but they are very transitory. Again, if we look at those cases where motor or mixed nerves are pressed upon and irritated by new growths, pain is a very prominent symptom, and spasm seems scarcely to occur, though twitchings and transitory contractions are observed. A case which I have already related affords a good example of a tumour pressing upon a purely motor root. The new growth was a myxoma, which grew in connection with one of the left anterior spinal roots, and gave rise to symptoms in the left arm. These were weakness and attacks from time to time of pain and cramp; but no prolonged spasm was ever produced.

Dr. Bristowe has written a paper on Painful Paraplegia in *St. Thomas's Hospital Reports*, vol. xii, in which he relates several cases of paralysis accompanied by pain, which owed their origin to malignant tumours involving peripheral nerve-roots. Some of these cases I saw myself, and also examined *post mortem*. Evidence of spasm occurs in only one case, where the limbs are said to have become stiff occasionally.

Clonic spasm is not unfrequently seen in late stages of facial paralysis, when there is shortening of the muscle from atrophy. Electric examination shows that the latter condition is present, and also that a certain amount of muscular tissue, capable of active contraction, remains. The clonic spasms result from irritation of the nerve and muscle which has survived the process of destruction. In cases of meningitis at the base of the brain, spasm of the muscles supplied by the motor nerves involved is not observed. In spinal meningitis, on the contrary, it is a very marked symptom. Here, however, it is not improbably of a reflex origin, and not due to direct irritation of motor nerves.

Tetany (Fig. 43) is a disease in which the presence of some, although generally trivial, alteration of sensation, increased electric irritability of nerve and muscle, and prolonged muscular spasm, suggest a peripheral cause. The pathology of this affection cannot be said to be known, and opportunities of examining cases *post mortem* rarely fall to the lot of pathologists. Dr. Hadden kindly gave me the opportunity of examining microscopic sections of the nerves and muscles of the affected parts, which were taken from a child who died from diarrhoea, while suffering from tetany. At first I thought there were changes to be made out; but, on procuring sections from the same parts, in a healthy child of the same age, 16 months, who had died of an acute disease, and, comparing them with those from the subject of tetany, I came to the conclusion that there was very little difference between them.

Looking at my own experience, I should say that, while occasional spasm occurs not unusually in peripheral nerve-disease, from direct irritation of the motor filaments, and may even be permanent in disease of the facial nerve, it is quite the exception to find muscular contractions among the marked phenomena of such cases.

Weir Mitchell, who has had a very large experience of injuries and diseases of peripheral nerves, writes thus on the subject (*Injuries of Nerves and their Consequences*, 1872). "Increase of bulk, proli-

FIG. 43.—The hand in a case of tetany.

feration of connective tissue, and wasting of nerve-tubes, are consequences of chronic neuritis." "The nerve-tubes in large part perish or waste, and the symptoms affect at first rather the sensory sphere than that of motility. We have pain and anaesthesia, or hyperaesthesia, but not, as a rule, local convulsions." "In certain cases, the nerve-wound, in place of causing primary loss of motility, occasions either sudden muscular contraction, followed by instant loss of power, or, in very rare instances, long continued spasm. Tonic contractions of muscles are occasionally met with at a later stage of these injuries, but are, perhaps, among the rarest of the secondary symptoms."

Looking, then, at the experience of Weir Mitchell and others, we may safely conclude that muscular spasm is rarely caused directly by chronic disease of peripheral motor nerves.

[To be continued.]

ABSTRACTS OF SIX LECTURES

ON THE

INTESTINAL CANAL AND PERITONEUM IN THE MAMMALIA.

Delivered at the Royal College of Surgeons.

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LECTURES IV, V, AND VI.

The Insectivora.—The stomach in the insectivora is, in its general characters, simple; it recalls the stomach of the monotremes. The large protrusion to the left of the gullet, that is so marked in the rodents, is absent. In the shrew, the two diameters of the viscus are about equal. In the hedgehog, banxring, and tanrec, the transverse diameter prevails. In the mole, the stomach is of great size, and possessed of a large cardiac sac; the duodenum is large and simple, and loosely supported. The "bend" in the intestinal loop is present, but is not very pronounced, owing to the looseness of the duodenum. It is an error to state that the intestine in the shrew forms a single loop with a median mesentery, as in the amphibia. In this animal, the loop has been bent upon itself, so that the right side of the mesentery has become the left, as in the higher mammals. In all the insectivora, the intestine is short; in the shrew, it is three times the length of the body; in the hedgehog, six times; and in the mole, seven times. There is no distinction between colon and small intestine; there is no cæcum; the colon is never sacculated. The superior mesenteric artery is distributed upon the plan that obtains in the monotremes, and is totally unlike the corresponding vessel in the rodents. The superior mesenteric artery is present. The great