either case, however, the effects on the obstructed organ or part will not be materially different.

With respect to arterial embolism, not only the large concretions which form in the left auricle and ventricle, but also the smaller ones which are deposited on the valves, as well as the degenerating matters which escape from both forms of concretions, are liable to be swept away by the blood-current to form embolic plugs in the arterial branches. The clots also which form on the coats of diseased arteries, and likewise the fatty or calcareous matters which form there or on the valves, are carried away and block up the arteries of the spleen, the kidneys, the brain, or the limbs; the relative degree of liability to be affected being in this order, and being due to the particular conditions of the circulating system of these organs.

I need not describe the appearances of infarction and softening presented by each of those organs in consequence of embolic obstruction. We are all, however, I believe, in the present day aware of the fact that embolic obstruction of the arteries of the brain is a frequent cause of softening and paralysis, which cannot be removed by any active depleting and counter-irritant treatment, but is more likely to be benefited by rest and gentle stimulation. And I would remark that, just as Paget, so far back as 1844, drew attention to coagulation of the blood in the pulmonary artery, ably illustrating the subject by a series of carefully observed cases, so also Dr. Kirkes was, in 1852, the first to demonstrate clearly by another series of cases that fibrinous deposits in the interior of the left side of the heart are a frequent cause of softening of the brain, which always results when the obstruction is beyond the circle of Willis, owing to the absence of anastomosing branches. In his very original paper, he also illustrated the effects of embolic obstruction in producing infarctions of the spleen and kidneys, and also the violet-coloured petechial spots on the mucous membrane of the stomach and bowels, which are produced by the same cause.

I have condensed from the Infirmary Clinical Medical Prize Reports of this year a case which occurred lately in the Infirmary, which shows well the close connection between valvular disease of the left side of the heart and cerebral softening.

A woman, twenty-one years of age, admitted with disease of the heart consequent on rheumatic fever, became suddenly unconscious, with right hemiplegia, the day before her death. The middle cerebral artery was found plugged by white fibrine; and on the mitral and aortic valves were found numerous fibrinous and calcareous vegetations, which showed the source from which the embolic matter was derived. The cause of the obstruction in this case is sufficiently clear; but in another case, taken from the same reports, and which occurred in the hospital practice of my colleague Dr. Glynn, the cause of the arterial obstruction was more obscure, and was probably from thrombosis, seeing that there was also venous thrombosis.

A girl, nineteen years of age, was carried helpless into the Infirmary on January 4th, having had a sudden seizure, followed by left hemiplegia and anæsthesia, which had been preceded by pain in the right side of the head. She died on March 13th. There was extensive softening of the right middle cerebral lobe. The right internal carotid artery was plugged by what appeared to be an embolus at the bifurcation of the anterior and middle cerebral arteries; the higher part being firm and of yellowish colour; but lower down, of dark brown colour, and having a conical termination in the external carotid. The thyroid body was enlarged; the heart sound. There was also a thrombus in the lower end of the vena cava, and in the left common and external iliac veins adherent to the walls.

Here, again, we have coagulation of blood in both the arterial and the venous system which has been observed in so many other recorded cases.

I might have alluded to some other forms of embolism, to the effect of embolism of the coronary artery in arresting the heart's action, to the effect of plugging of the central artery of the retina in destroying vision, and to the connection between embolism and paraplegia, chorea, and some other nervous affections. I might also have alluded to the view taken by Dr. George Johnson, that the lobular pneumonia which often follows extensive burns arises from the passage of coagulated blood from the injured parts to the pulmonary vessels and the duodenal ulcers from a similar cause. We can readily understand the wide range of effects produced by the blood distributed throughout the whole system; but yet we must be careful in not ascribing to the effect of embolism anything that is not proved by rigid observation. Sufficient has, however, now been adduced to show how great are the consequences that may result from the coagulation, the lessened vitality, or it may be the death of a minute portion of the blood passing through the heart or circulating in a vein or an artery, and to prove the importance of a knowledge of this subject to all who are engaged in the practice of medicine or surgery.

BULBAR PARALYSIS.

BY THOMAS S. DOWSE, M.D., F.R.C.P.E.,

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[Concluded from p. 583 of last number.]

BEFORE entering upon the pathology of this disease, I wish to draw attention to two typical cases, which are at the present time under my care—one of complete cure, and the other a retrogressive case of reflex bulbar paralysis.

CASE III. *Bulbar Paralysis of five years' duration: Cure.**—Daniel Megan, aged 22, of neurotic tendency, and with a history of convulsive seizures in infancy, had fairly good health in childhood, but became subject to a series of epileptic fits when sixteen years of age. These were succeeded by paralysis of the seventh, eighth, and ninth nerves, and by increasing failure of motor power all over the body. He came under my care three years afterwards. When first seen by me, the countenance was calm and intelligent; he was unable to bring the lips together, to spit, or blow out a candle. The lower jaw could not be fully depressed, neither could the lateral movement of mastication be effected. Saliva was constantly dribbling from the mouth, and the tongue lay flaccid, furrowed, and helpless behind the lower row of

Fig. 4.

teeth. He was able only, in a very limited degree, to retract it or to move it in a lateral direction. The want of power in deglutition showed itself chiefly, though not entirely, in the first act, which was performed apparently with great difficulty. The palate muscles were more or less involved, and he would occasionally suffer from partial asphyxia, from particles of fluid entering the larynx. There was some inhibition of the vagus, evidenced by respiratory and cardiac irregularities; and, like many other cases, his head seemed fixed so that he moved it with difficulty. This, doubtless, arose from palsy of the spinal accessory nerve, whose origin, according to Dr. Lockhart Clarke, is intimately associated with that of the vagus and hypoglossal. The vocal chords were found to act feebly. He was quite dumb, and unable to make a sound. There was complete motor paralysis with impaired sensation of the upper limbs, and entire paralysis of the lower to motion, and partially to sensation. The muscles of the latter were greatly atrophied, especially the extensors, so that the heel was drawn up, as in talipes equino-valgus. There were no tremors or muscular fibrillations. There was dulness at the apex of the right lung, with crepitation. Altogether, I thought the case most unfavourable, so I merely fed him well, and gave him steel, with cod-liver oil. He gained flesh, and his general health improved; saliva ceased to flow from the mouth; the act of deglutition was performed fairly well; the palate and vocal muscles also gained power; yet the tongue remained as motionless as ever.

In December of last year, I determined to galvanise the tongue regularly; and, to show how much its sensibility was impaired, I could get no response to an induced current of Stohrer's two-celled battery. I persevered daily for three weeks, and at the end of this time electric sensibility was aroused; and, to his own surprise as well as mine, he protruded the tongue, and went through the alphabet, with the exception of R, O, F, P, W, and S. The following day, he was unable to protrude the tongue until it was galvanised; but from this time he exercised voluntary control over it, and he now has perfect use of lips and tongue, can swallow, and speak with almost perfect accuracy. The tendo Achillis has been divided, and by the daily use of the constant current, and injection of a quarter of a grain of strychnine, he is fast regaining the use of his lower limbs. In this case, which is of unusual interest (and in English medical literature I do not find another recorded), we have good evidence of lesion of the medulla, and possibly

* Patient shown at Clinical Society last session.

of the anterior grey matter of the spinal cord, in association with general muscular atrophy. Professor Hausmann of Friburg connects the two conditions in the following words. "If we compare bulbar paralysis with progressive muscular atrophy, we find that they are both dependent on manifold degenerative processes of the same anatomical character in each; and which agree with one another in their tendency, on the one hand, to spread gradually over the anterior grey columns of the spinal cord, and the nuclei of the medulla oblongata which correspond to them; and, on the other hand, to destroy the ganglion-cells embedded in these parts, either singly or in groups, and in an irregular manner. According as such a process at first attacks a higher or a lower region, the medulla oblongata or the cervical or lumbar enlargements of the cord, the results are different, yet without losing their typical character. The greater danger of bulbar paralysis, and its short duration, are easily explained by the close proximity of the diseased parts to the *centrum vitæ*." I believe that this is now the accepted doctrine, and in the main is true enough; yet, in my experience of bulbar paralysis and associated muscular atrophy, one finds a want of muscular-cutaneous sense and sensibility, which one fails to observe in ordinary spinal muscular atrophy. However much progressive spinal muscular atrophy resembles, or is associated with, paralysis of the nerves originating in the motor nuclei of the medulla, we do not find progressive muscular atrophy accompanying the latter form of disease in the same ratio. Dumenil thought that this arose from a lesion of the sympathetic; but of late a theory has been advanced by Duchenne and others, who consider that the ganglion cells of the anterior horns of the spinal cord and motor nuclei of the medulla are of two kinds, motor and trophic. So, where the progressive muscular atrophy appears with its typical characters, they believe the lesion to be limited to the trophic cells. On the other hand, where, as in cases of progressive paralysis of the tongue, the paralysis runs its course without atrophy of muscle, the lesion is one of motor cells alone.

The following is a brief record of a case of bulbar paralysis, retrogressive in its character, which I consider to be of reflex origin.

CASE IV. *Reflex Bulbar Paralysis*.—E. B., aged 59, a pale unhealthy-looking woman, was attacked with inflammation of the sub-maxillary and parotid glands. She was also suffering from Bright's disease. The attack of inflammation was severe and prolonged, and, upon recovery, there was a decided paralysis of the facial, hypoglossal, and spinal portion of the spinal accessory nerves, as well as the third division of the fifth. The vocal cords acted feebly, and she could

Fig. 6.

only speak in a whisper. My attention was first directed to her condition because she was unable to pronounce the linguals R and S, and she could not protrude the tongue over the lower row of teeth. Food lodged in the cheeks, which the tongue was unable to dislodge; saliva was constantly dribbling from the mouth; and enough air could not be sent through the lips to extinguish a candle. The first stage of the act of deglutition was alone affected, owing to the palate muscles acting fairly well. She has improved very considerably of late, and I think the improvement is due to the use of the constant electric current.

Concerning the microscopic pathology of this disease of the medulla, we have now ample evidence, through the researches of Lockhart Clarke, Kesteven, Duchenne, Charcot, and others. We find Dr. Clarke's researches on this subject in the *Medico-Chirurgical Transactions*, vol. iv, 1873, where the grey matter of the medulla and cord presented a variety of lesions. Hypertrophy of the connective tissue, with proliferation of its corpuscle, and aggregation of these in masses at the angles of junction in the network, are described by the author. Several patches of disintegration were observed—one of large size consisted of remnants of partly disintegrated grey substance, irregularly connected with each other, and forming together a kind of honey-comb

structure. Several large areas of disintegration and hæmorrhage clots existed, involving considerable destruction. In all regions of the cord the nerve-cells had undergone degeneration and disintegration. Some were completely, others only partially, filled with dark-brown pigment-cells, which in many instances enveloped and concealed their nuclei. All the remaining cells were reduced in size. Many seemed to have been lost by gradual atrophy, and numbers had wholly disappeared by complete disintegration, or fallen into granules. These lesions were traced in the nuclei of the facial, hypoglossal, vagus, and spinal accessory nerves, and explained the symptoms of glosso-pharyngeal paralysis. The extensive loss of substance in the anterior and lateral grey substance of the cervical and dorsal regions, more especially of the tractus intermedio-lateralis, explained, in the latter stages of the disease, the feebleness of the respiratory movements.

The question which I am desirous to raise is this. There are certain pathologists who seem anxious to maintain that clinical observers are in error when they apply the term bulbar or glosso-pharyngeal paralysis to lesions, which, although existing in the medulla, extend to and involve other motor nerves, and even the lufcephalic motor tract. No one is more anxious than myself to preserve exactitude in diagnosis; but my practice in the study of nervous disorders, aided by the careful microscopic researches of my friend Dr. Kesteven, to whom I am most deeply indebted, has convinced me that intrinsic nerve-change of an interstitial and subacute inflammatory type, such as we know to give rise to the morbid lesion of the medulla, evidenced by bulbar paralysis, may be associated from the onset with a similar change in the pons Varolii, as well as the cervical spinal cord. It is undoubtedly necessary that care should be taken in making a diagnosis in respect to this disease, otherwise we should soon fall into error. I have several cases at present under my care, where there is apparently a pseudo-bulbar lesion existing, with more or less paralysis of the limbs. Yet, it must be noted that the bulbar nerves are not affected bilaterally, and there is a marked hemiplegia of one side over the other.

I know of nothing in the whole range of nerve literature to compare with the original and exact observations of M. Auguste Voisin, in the *BRITISH MEDICAL JOURNAL* of June 19th, 1875, and can practically verify every statement which he has made. The troubles of language, he says, are of various orders, and may be termed—

1. Stuttering, drawing, hesitation;
2. Jabbering, stammering, and quavering.

The first three are caused by lesions found in the course of the nerve-fibres, passing from the cortex of the anterior convolutions to the medulla, through the corpora striata crura and pons Varolii, and involves disturbances of the intellect and will. The second three do not result from any troubles of intelligence or will; but are consequent upon an absence of harmony in the coordinate acts performed by the muscles animated by the nerves proceeding from the medulla oblongata. I mean the hypoglossal, facial, spinal, and glosso-pharyngeal nerves. He goes on to say that the first order of troubles of speaking is determined by the infiltration of the cortical substance of the frontal convolutions, and of the island of Reil with blastema, and by the softening of it by the production of embryo-plastic nuclei, first in the vessels, next in nervous substance among fibres conducting the will, and by the ulterior organisation of these embryo-plastic nuclei into fibrillary tissue. The second order of symptoms is occasioned by the infiltration of blastema, and by the multiplication of embryo-plastic nuclei in the vascular sheath of the perivascular spaces; next, amidst the nerve fibres of the bulb, and by necrobiotic alteration of the cells of the original nuclei of the nerves of the medulla, and especially of the facial. The third group of the troubles of speech is produced by atrophic lesions at the apparent origin of the bulbous nerves, and by fatty degeneration of the muscles of the tongue.

In concluding the paper, what we have lastly to consider is this. Have we at our command any means, in the whole range of therapeutics, which will obviate the tendency to the progressive course of the disease? I believe we know of few medicines which would directly check and control it. I certainly have seen good done by a seton in the back of the neck, and also by the local application of the continuous galvanic current; but the chief and vitally essential point in practice is nutrition and rest. Directly deglutition becomes at all difficult, it is necessary to feed the patient freely and regularly. This is best done by a piece of India-rubbertubing passed through the nostril. Patients of mine have soon acquired the habit of feeding themselves in this way; and it is a curious fact, that nothing seems to satisfy their craving for food. A man under my care passed into his stomach an enormous quantity of fluid nourishment, and, not satisfied with that, would steal food from other patients, and upon more than one occasion nearly choked himself through his voracity, but he gained flesh and held the disease in check. There are other points, too, which ought to be considered.

The urine should be carefully tested for sugar and albumen—the specific gravity is usually much below normal. I have found injections of one-fortieth grain of atropine to moderate the excessive salivary secretion; and, as the digestive powers are usually good, cod-liver oil, with phosphorus and steel, are undoubtedly useful.

ON THE STRUCTURE OF THE MATRIX OF HUMAN ARTICULAR CARTILAGE.

By H. A. REEVES, F.R.C.S.,

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HAVING for some months past been engaged in the study of joints, I have had occasion to apply various methods, too numerous to mention here; but I wish to bring under the notice of histologists some means by which the demonstration of the structure of so-called hyaline cartilage may readily be accomplished.

The perusal of Dr. Thin's paper on the Structure of Hyaline Cartilage (*Quarterly Microscopical Journal*, January 1876) stimulated the conviction which I have long held and taught as to the uniformity of structure; and I have endeavoured to convince myself of the existence of normal fibrillation in human cartilage, if it were possible.

Some weeks ago, I placed the fresh articular elbow-ends of the humerus, ulna, and radius of a woman aged 65, whose arm had been amputated for the results of senile gangrene, in a 0.5 per cent. silver solution for ten minutes. They were then removed and exposed to the light in the dry state (*i. e.*, without being placed in any liquid) for nearly three days. I had intended to examine them at once, but other work prevented it. On making horizontal thin sections, and examining them first as they were and without a cover-glass, and subsequently other similar sections in glycerine, I was struck with the appearance of straight bands running in various directions and planes, separated by bright but somewhat interrupted lines, which in some parts appeared to be made up of minute round elements, reminding one of the "elastic grains" described and figured by Ranvier. I must mention that the cartilages were washed in ordinary water before immersion in the silver, and that the capitellum was rubbed with a clean towel, in order to get rid of adhering synovia. My object in doing this was to avoid any false appearances caused by the deposition of silver in the synovia and the subsequent shrinking of the latter.

Thinking that this appearance might perhaps be due either to the age of the woman or to the fact of the limb having been so long inflamed, or to the change effected during the three days the cartilages were exposed, I examined the cartilages of younger people very shortly after removal, fresh and without any reagent, and also after staining in gold, silver, and aniline dyes; also in salt solution. I almost invariably recognised the same appearances; but they are not so distinct in fresh as in stained preparations. The fibres are perfectly straight, in this respect and in others differing from those figured by Dr. Thin of the kitten and sheep. In some of the sections, rounded, oval, and irregular figures—probably the transverse sections of similar fibres—were visible; but on this and other interesting appearances I intend to dilate more fully ere long. It is important to note that these appearances are not in any way artificial or due to the methods used, as perhaps may be said of the methods Dr. Thin employed, and of those of Tillmanns and Baber. The fibrillation is a continuous right-lined one, differing much from that figured by Tillmanns and Baber. Once recognised on stained sections, they are readily made out in fresh ones.

The gold method I employed was the following. Fresh articular ends of bones were placed for twenty minutes in a 0.5 per cent. auric chloride solution. They were then allowed to remain all night in a 0.02 per cent. solution, and in the morning were removed and exposed to the light in the dry way all day. In the evening, horizontal sections were made and examined.

I may add that, after taking every possible precaution to avoid fallacy in silver preparations, I observed figures very similar to those represented by Tillmanns, which he deems artificial, of which more anon.

Conjunctival End-Bulbs of the Gold-Fish.—In osmic acid preparations of the conjunctiva of the gold-fish I have observed end-bulbs, similar to those first discovered by Krause, which are of a somewhat complicated structure. They will be figured and described shortly. So far as I have been able to seek, I have not come across the mention of their existence in fishes.

Parasites in the Cray-Fish, Newt, and Earth-Worm.—Some years ago, when working, through Professor Huxley's courtesy, in his labora-

tory, I noticed under the microscope peculiar oval bodies in the muscles of *Astacus fluviatilis*. They only occurred in one specimen. I showed them to two or three gentlemen, and at the time referred to the works of Cobbold, Küchenmeister, Von Siebold, and other helminthologists, but could not find any description to correspond to the bodies observed. I cannot be more precise in the description, as the preparations are now spoiled; but I direct attention to the fact, as the cray-fish is largely eaten, in order that some one interested in the subject may investigate the matter.

On laying open the abdomen of a young newt, I observed what appeared like a hair in lively motion. Under a low power, it looked like a small thread-worm; but its motions were too lively to permit of study. My instruments were quite clean, and I am sure that the creature did not get into the abdomen after the latter was opened. It was found near the posterior part of the abdominal cavity; and, as I was dissecting under the microscope, I should have noticed it at once had it been introduced *ab extra*. Had it been swallowed, and then perforated the intestine?

On placing a portion of the segmented organs of an earth-worm under a quarter inch, I noticed, coiled up, what looked like trichina spiralis; but it was not encysted.

I regret very much that I cannot be more accurate in my statements, not being well versed in helminthology; but, crude as these observations are, they may induce some one with more knowledge to inquire into and discover more of a subject which may turn out to be of importance.

HYDATID OF BRAIN: CHRONIC MENINGITIS: SEROUS EFFUSION: APOPLECTIC SEIZURE: DEATH.

By W. H. MACNAMARA, M.D.,

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PRIVATE J. F., 106th Regiment, aged 37, in service seventeen years, principally Indian, had head-symptoms, attributed to the effects of sun in 1864. He was frequently in hospital with remittent fever, and several times with chronic rheumatism. He had had no trouble with his head since 1864. At 5.30 P.M. on September 19th, he was seen by a comrade; he seemed all right, but had been drinking. About half an hour afterwards (6 o'clock), he was found naked in a bath-room, with his head in water, to all appearances dead. Artificial respiration brought on hissing breathing. Dr. McEwen saw him about 6.30, and found him insensible; pupils dilated; breathing laborious and hissing; pulse quick, but firm. His bowels were moved. His feet and hands were quite cold. Warm bottles were applied to the hands and feet, and cold to the head. When I saw him a quarter of an hour afterwards, he was in the same condition, but with twitching of the muscles of the arms and stiffness of both legs. Epileptiform seizures came on every ten minutes or quarter of an hour. No smell of liquor, enlargement of the heart, or sign of atheroma of the arteries, was detected. About 8.20, the temperature began to rise rapidly; at 8.45, the thermometer registered 106.6. Respiration became quicker and more laborious; the epileptiform seizures more frequent and violent. The pulse remained firm. Urine was passed freely. The patient was packed in wet sheets; ice applied to the head. The temperature was gradually reduced. The spasms became less frequent, but the pulse became weaker towards morning. The wet sheets were discontinued. Brandy and beef-tea were administered by the rectum. The patient gradually sank, and died comatose at 7.25 A.M. on September 21st, about thirty-six hours after the attack.

POST MORTEM EXAMINATION, twenty-eight hours after death.—The membranes and sinuses of the brain were congested. The dura mater was thickened, and the visceral and parietal layers of the arachnoid adherent in the vicinity of the superior longitudinal sinus. A good deal of serum escaped when the medulla oblongata was cut across. There was considerable serous effusion on the surface of the brain under the arachnoid. The brain-substance was soft; its cut surface was glistening, a large quantity of serous fluid oozing from it. There were no blood-clots. The ventricles contained but a little fluid. A cyst, about the size and shape of a pigeon's egg, was found in the substance of the left hemisphere, on the outer side of the corpus striatum; it was attached to the brain-substance by areolar tissue and blood-vessels. The capsule was thick, opaque, and calcareous; its contents were fluid. I have not had them examined microscopically. There were no parasites in the intestines.