mass of the digested (i.e. emulsified) fat passes into the lacteals. Since, however, the portal blood contains more fat than the general venous blood, some of the fats must pass through into the portal capillaries. The portal blood, moreover, during digestion contains a small but appreciable quantity of soaps. Neither in the portal blood, nor in the chyle, nor in the general blood during digestion, is there any appreciable quantity of peptones. It must therefore be left uncertain what becomes of the peptones formed during digestion, and which way they travel. We know, however, that artificially-formed peptones are available for nutrition; thus Plósz and Gyergyai found that dogs fed on peptones and non-nitrogenous food actually put on flesh and gained weight. The chyle contains no large amount of sugar; and though, contrary to statements so frequently made, sugar may undoubtedly be found after meals in the portal blood, we are not at present in a position to say whether sugar is absorbed chiefly by the portal system or chiefly by the lacteals." Systemic lymphatic absorption needs no other proofs, in the present day, than the enlargement of the glands in cases of local poisoning from syphilis or dissection wounds, and is daily brought into requisition in the hypodermic administration of medicines.

The dispute between Dr. Wm. Hunter and Dr. Monro as to the discovery of the functions of the lymphatic system, gave a great impetus to its study, and to this we are indebted for many important researches, not only by these anatomists, but by the illustrious John Hunter, Hewson, and Cruikshank. Their investigations were carried into comparative anatomy as well as into every part of the human body. Hewson showed for the first time that the lymphatics were not limited to mammalia, but existed throughout the vertebrate sub-kingdom, whilst Cruikshank minutely and most accurately described and figured them in man. About the same time the elder Meckel also described them very fully. The nerve-centres and the special organs of sense were the only parts in which the lymphatics had not been traced, but this hiatus was due to the extreme difficulty of the investigations rather than to any doubt of their existence in these organs. The descriptions of the lymphatics in the cranium by Collins, Marchetti, and Ridley, are very imperfect, and Pacchioni's account is obviously incorrect. Monro seems to have once, in the skate, succeeded in injecting some of them with coloured fluid, which passed into and stained the tunics of the eye, the ear, and the arachnoid. In 1787 Mascagni published his classic work, in which he not only revised the labours of his predecessors in this field, but recorded the results of the dissections of a lifetime. To his magnificent illustrations subsequent anatomists are mainly indebted for their descriptions of this system.

The mesenteric glands were noticed by the Alexandrian anatomists, by Celsus, Marinus, and Galen. Eustachius very fairly described their naked-eye appearances, and Rufus pointed out that similar structures existed in the neck, axilla, and groin. Aselli followed some of his predecessors into mistaking a number of glands fused together in the mesentery for a single gland, which he called the pancreas, notwithstanding that the true pancreas was well known long before his time. He traced the lacteals to this gland, but Pecquet incidentally mentioned that the mesenteric glands were isolated and not congregated together as Aselli had described them. The arrangement of these glands is very different in the various species of animals: in the cat the fusion of them, forming the pancreas Aselli, is well seen. In 1660, Sylvius de la Boë named these glands *conglobate* glands, and so separated them from the secreting glands with which they had been confounded. The anatomists who attempted to unravel their intimate structure were divided into two camps; some followed Malpighi, in the view that they contained distinct spaces or acini, whilst others supported the opinions of Ruysch and Albinus, who held that they were composed of a network of vessels only. The facility with which injections pass through a lymphatic gland from the afferent to the efferent vessels evidently led to the latter idea; whilst the former was strenuously maintained by those microscopists who examined uninjected specimens.

Since the discovery of the lymphatic system, and the demonstration that most, if not indeed all, lymphatic and lacteal vessels pass through one or more of these glands, much discussion and interest have naturally arisen with regard to their function. Bartholin thought that the mesenteric glands were of use in preparing the chyle for absorption.

Wharton and Glisson had a confused idea that their office was to separate a nutritive juice from the nerves for the nutrition of the tendinous or white structures of the body, and that this juice was carried everywhere by the nerves, whilst the efferent lacteals carried the surplus to the common receptacle. The observations that the glands are large in young animals in proportion to the size of the body, that they are smaller in the adult, and still less in size, firmer in texture, and that much less juice exudes on section in old age, led to the opinion that they both delayed the passage of the lymph and chyle, and added something thereto, and that this, in the case of the chyle at least, served to dilute that fluid before it mixed with the blood-stream. To Hewson is due the great merit of first showing that this juice contained corpuscles, resembling in size, shape, and reaction the nuclei of the blood-corpuscles (Hewson is speaking generally of the blood of birds and frogs, and not of the mammalia), and that the lymphatic glands were therefore one of the sources of origin of the blood-discs. He attributed similar uses to the thymus and spleen. On his observations rests all the modern pathology of the relation between diseases of the lymphatic glands and the changes in the blood and system generally.

CASES OF CEREBRAL TUMOUR ILLUSTRATING DIAGNOSIS AND LOCALISATION.

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(Concluded from p. 329.)

CASE 2. *Epileptoid attacks, with visual aura, ceasing under treatment; death, with subacute cerebral symptoms, after a fall; large tumour of right occipital and parietal lobes.*—Alfred S—, aged thirty, married. One brother had suffered from fits. Venereal disease denied; no history of constitutional syphilis, personal or marital. The patient stated that he was well until two months before he was first seen (April, 1878), when one morning something seemed very brilliant before him, "as if he had a polished plate on his breast." He felt giddy, but did not fall; he sat down, bathed his head, and was better, but afterwards felt extreme pain in his eyes, "as if they were bursting." Subsequently he had slight attacks, almost daily, of the following character. "A pain commences in the neck, goes across the head, comes down between the eyes, and is felt on each side of the bridge of the nose. If walking, the road or path seems to get narrower and narrower, so that he hardly knows where he is going, and simultaneously his sight fails; he feels with a stick to see if he is not getting off the path. The pain at the top of the head and in the eyes is something awful, and the eyes seem to throb. The loss of sight is not complete; he can only see just before him; nothing on either side; but he can generally see better to the right than the left." During the three days before he was first seen, at the commencement of the attacks, as the sight was going, he had a flickering of light, "like a gold serpent," in the eye, moving in all directions very fast; seen with both eyes he thought, but more before the left eye than the right. The pupils were equal. Acuity of vision normal. I thought that there was a very slight defect in the left field on the temporal side, but I could not, even on very careful examination, be sure of it.

He was treated with bromide and belladonna, and the attacks ceased, except that once or twice he had a slight flickering before the eyes. He had, however, several attacks of pain on the top of his head, coming on suddenly, and he often had pain at the back of the neck. He was seen repeatedly during the next three months, and continued free from attacks.

Five months after he was first seen (as I subsequently learned) he had a fall, and afterwards most severe pain in the right side of the head, just above the right eyebrow, darting through the head. His manner became altered, and his speech confluent, words being run together. He also

had delusions, fancying that persons were in the room who were not. He then became comatose for a day or two, and died.

A post-mortem examination was very kindly made for me by Dr. Henderson, of University College Hospital (the patient having died in the country). Dr. Henderson's report was as follows:—"Dura mater normal; surface of brain pale; convolutions flattened. Base of brain, and arteries and nerves, normal. Membranes opaque over locus perforatus, but no lymph. Left hemisphere normal on surface and section. In the right hemisphere, the occipital and posterior part of the parietal lobes are the seat of a growth.

FIG. 1. Diagram of outer surface of right hemisphere.
FIG. 2.—Diagram of inner aspect of right hemisphere.
In each the shaded portion indicates the position of the growth.
(From a diagram by Dr. Henderson.)

On the outer surface this involves the first and second occipital convolutions and the superior and inferior parietal lobules, and on the inner surface the cuneus and half of the precuneus. It projects into the lateral ventricle at the junction of the posterior and descending cornua, and, overlapping the thalamus, has indented it, causing a depression two centimetres by three, without adhesion. On section, the corpora striata and optic thalami appear normal. The growth has no sharp limitation; it is adherent to the pia mater. Its section is pinkish-grey and somewhat gelatinous, dotted with vascular points, and containing in the centre a spot of hæmorrhagic infiltration two centimetres by three and a half. There was no caseation or margin of induration. The rest of the brain was normal, except that the right posterior quadrigeminal tubercle was a little softer than the left. The growth had the structure of a sarcoma."

Remarks.—This case presented, when first seen, symptoms much more like those of idiopathic epilepsy than of organic brain disease. There was a family history of fits. The attacks began with a visual aura, not uncommon in epilepsy, and ceased under bromide and belladonna. Headache was—such as sometimes occurs in epileptic seizures—paroxysmal. There was no permanent affection of sight. I was so unable to convince myself that there was an actual limitation of the field of vision that I dismissed the idea. There was no optic neuritis. The significance of the symptoms, taken together, was strongly in favour of epilepsy. The graver symptoms of organic disease which came on after the fall were suggestive rather of chronic meningitis than of tumour. Possibly optic neuritis may have come on after I ceased to see the patient, but it was absent as long as he was under observation.

The large tumour which was the cause of the symptoms was situated in a position in which the ordinary symptoms of tumour are often latent. The case is, with one exception, of little value as regards the positive symptoms of tumour in this region, since the patient's later symptoms could not be ascertained. The absence of hemiplegia, however, is in accordance with the position of the tumour—behind the motor region. We do not know in what spot the growth commenced, and therefore can infer little from the absence of any loss of sensation during the early stage of the case. The tumour was in the "sensory region" of the brain, and the paroxysmal symptoms were acute pain and a transient

disturbance of vision. The latter is of great interest, because the tumour had invaded part of the angular gyrus, in which Ferrier's experiments on monkeys have led him to localise the sense of sight, and the visual aura may certainly be associated with the disease in this region.¹

The character of the aura is also of interest, especially if we can rely upon the accuracy of the patient's very intelligent description. At first there was apparently an over-action of the centre—a bright light; afterwards paroxysmal defective action, due to temporary exhaustion or inhibition, shown in the narrowing of the field of vision. The apparently greater narrowing on the side opposite to the diseased hemisphere may have been really as described, or may have been a transient hemiopic defect. The former is the more probable, on account of the clear and circumstantial description, and also because it was associated with a transient change in the acuity of vision.

The object before him (the path) appeared to become further off, narrower, and indistinct. We may correlate the early discharge with the subsequent arrest of function; but the early discharge was wide-spread, as a large brilliant area. If the subsequent arrest of action was equally wide-spread, we are led to the supposition that the discharge which affects central vision much may lead to an inhibition which affects central vision little, and hence that it is the degree rather than the position of interference with the action of the centre which determines the position of the affection of function. We have of course no ground to assume that there is any superficial representation of the field of vision in the centre in correspondence with the field itself.

The apparent recession of the object during lowered function is in harmony with the apparent approach of an object during discharge, which is often described by epileptics. A bright light will appear to get nearer and nearer, until consciousness is lost, just as a pain or a noise will get more and more intense. This means probably that the more extensive discharge (not necessarily superficial extension) produces the same effect on the centre as does an increase in the area of the retina on which an image falls. In arrest of function, the converse process takes place. The stimulation of the less sensitive centre, by the image of the path, became so much slighter as to produce the effect of a diminution of the area of the retina stimulated, and hence the path appeared to recede.

The affection of both eyes, greater on the side opposite to the diseased hemisphere, is noteworthy. The association of the two visual centres is no doubt exceedingly intimate. Of not less interest is the acute pain felt in the eyeballs after the paroxysm, as evidence of a very intimate central association between common and special sensation on the organs of special sense, the physiological utility of which will be obvious enough on a little consideration.

CASE 3.—*Subacute cerebral symptoms; paralysis of both auditory, right sixth, and partial of right facial nerves; general weakness of limbs; optic neuritis; hyperpyrexia; tumour in front of and involving corpora quadrigemina.*—Robert P—, aged twenty-four, unmarried, a police-constable, admitted into University College Hospital on July 30th, 1878, with subacute cerebral symptoms. No family history of nervous disease, of phthisis, or of tumour, and no personal history of syphilis could be obtained. The head symptoms with which he was admitted had come on after several days' exposure to hot sun, to which they were ascribed. It was, however, afterwards ascertained that he had complained of headache to his brother a month previously. Severe headache, and mental dulness, with paralysis of one sixth nerve, were the symptoms on admission. The pain in the head was apparently general, and not sufficient to prevent him from dozing most of the time. His attention was aroused with difficulty, and he sometimes answered questions, sometimes did not appear to understand them. There was no delirium, convulsion, or paralysis of limbs, no affection of special senses. The right external rectus was paralysed, but not completely. Other movements of the eyes were free, and the pupils equal. It was impossible to ascertain the degree of vision; he tried to read any type that was given to him, but closed one eye, as if

¹ According to Ferrier's diagrams of the centres of the human convolutions, the tumour would appear to have destroyed a large part of the visual region. Up to the last, however, no permanent affection of sight attracted the notice of the patient or his friends. I think that it is open to question whether the course of the sulci of the human brain has not induced Ferrier to carry the visual centres farther back into the occipital lobe than accords with their general position in the monkey.

there were diplopia. There was no fever; bowels constipated.

This state continued with little alteration. He was at first under the care of my colleague, Dr. Poore, and came under my care on August 12th. It was then impossible to obtain any answers to questions from him, although he did what he was told. During the next three or four days, however, he gradually became deaf, first partially, but, in the course of a week, absolutely. An ophthalmoscopic examination showed the existence of double optic neuritis, moderate in degree, equal in the two eyes. The outline of the disc was obscured, except on the outer side, where it could be faintly traced; elsewhere it was lost under a swelling, of a reddish-grey colour, striated, and extending a little beyond the limits of the disc. The veins were moderately distended, curving down the sides of the swelling, and obscured for a short distance at its edge. The arteries were considerably narrower than normal, and could be traced on the surface of the disc with difficulty. There was a good deal of white tissue about the centre of the swelling at the point of emergence of the arteries and accompanying them; one artery, passing upwards and outwards, lay in the middle of a broad white tract, which extended nearly to the edge of the swelling. Below the centre of the disc a white area lay in front of and partly concealed a vein, while an artery which passed in front of the vein was not obscured; on the inner and lower edge of the disc was another small white spot. The other eye was quite similar in general characters.

These symptoms continued with very little change. About the 20th of August, first retention, and then incontinence, of urine came on and continued. The pulse, previously normal, became infrequent (50 to 60). On August 24th his temperature, previously normal, rose to 100°, and continued to the end of the month varying from 99·5° to 101°. The swelling of the optic discs became more considerable, the veins forming a greater curve over the sides of the swelling, but there were no hæmorrhages. Two or three white spots, irregular in shape, were visible in the retina, just beyond the edge of the swelling, on the outer side, above and below. A trace of albumen appeared in the urine; no sugar. From this date the patient was in a state of almost constant semi-coma, and the elevation of temperature was greater, varying between 102° to 104°. He was observed also to move his limbs very little, but there was no loss of sensation to pain; when either arm was pinched it was moved, and the features expressed pain. The right side of the face was, however, moved much less than the left, and was evidently paralysed, chiefly in the lower parts, just as in the facial paralysis of hemiplegia. Observations on the pupils were unfortunately interfered with by the previous application of atropine. His pulse became frequent (90 to 100) and irregular. The paralysis of the sixth nerve was the same; no other oculo-motor paralysis could be discovered. On Sept. 7th the temperature was 104·6°, but on the 8th it rose to 106°; râles were heard throughout the chest; and he died, apparently from exhaustion.

Necropsy.—A sarcomatous tumour was found lying between the posterior portions of the optic thalami, above the crura cerebri, and in front of the corpora quadrigemina, which were invaded and otherwise damaged. The tumour was lobulated and irregular in shape, and measured an inch and a half transversely, and three-quarters of an inch from before backwards. From its middle a long pedunculated process of growth extended backwards above the tentorium cerebelli. It measured an inch and a half in length, and about half an inch in its greatest width, the base being about a quarter of an inch wide. No trace of the pineal gland could be found, and this pedunculated growth appeared to have formed in it. The tumour had invaded the corpora quadrigemina, destroying the whole of the left anterior tubercle and the inner half of the right, extending a little below the level of the iter. (Fig. 4.) Backwards from this point it extended a short distance only along the iter. The right posterior tubercle was normal; the left flattened by pressure. It had damaged a little the most superficial fibres of each crus cerebri, the left much more than the right. (Fig. 3.) It had not damaged, although contiguous and adherent to the inner surfaces of, the optic thalami. Slight traces of meningitis were found beneath the orbital lobules, but not elsewhere. No damage to the nerves at the base could be discovered. The ventricles were distended.

Remarks.—The onset of this patient's symptoms after exposure to a hot sun, and their subacute character, led to a frequent bedside discussion of the question whether they

were due to chronic meningitis or to tumour. Always, against the theory of meningitis, were the facts that there was no history of acute onset, and that the symptoms pointed to the base of the brain as the seat of the disease rather than to the region—the convexity—in which the meningitis of insolation commonly occurs. The ophthalmoscope did not help us, because, although neuritis was present, it was moderate in degree, the swelling being slight. It was such neuritis as is met with in chronic meningitis. Curiously enough, the necropsy showed that although there was a tumour, there was also slight—very slight—meningitis contiguous to the optic nerves, and the microscope revealed changes in the nerves, proving that the neuritis was really “descending.” The case illustrates the need for caution in assigning too much weight to exposure to the sun as a cause of cerebral symptoms, should they come on after such exposure.

FIG. 3.

Section through the crura cerebri, seen from before. The tumour conceals the corpora quadrigemina. The highest layer of the tegmentum is seen to be damaged in the outer part, more on the left side than on the right.—*a*, Tumour. *bb*, Superior cerebellar peduncles. *cc*, Tegmenta of crura. *dd*, Crustæ of crura.

FIG. 4.

Section through the anterior quadrigeminal tubercles—the point of their greatest invasion by the growth, which has destroyed the left and part of the right. Viewed from before.—*a*, Position of iter. *b*, Right anterior quadrigeminal tubercle. *c*, Tumour invading left anterior quadrigeminal tubercle.

The most noteworthy symptom in the case is the paralysis of the auditory nerves. The sixth nerve was probably damaged by pressure. Paralysis of the sixth is of less value than that of any other cranial nerve, on account of its exposure to pressure by its course over the most prominent part of the pons. As regards the auditory nerves, no lesion of the trunks could be found to explain it. They were perfectly normal, and there was no trace of lymph or of pressure. A possible explanation may be that the paralysis was due to the damage to the most superficial fibres of the tegmentum of the crus cerebri. If these contain the fibres from the auditory nerve, the symmetrical paralysis would be explained, since the most superficial fibres of each were damaged. The point is mentioned as worthy of attention in any future cases of the kind.

COLLEGE OF PHYSICIANS, IRELAND.—There will be no course of scientific lectures delivered this year at the College; but it is probable they may be resumed in 1880.