A LECTURE ON RECENT PROGRESS IN THE PATHOLOGY AND TREATMENT OF CEREBRAL PARALYSIS.

Delivered at the Hospital for Epilepsy and Paralysis, Regent's Park.

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I NOW proceed to consider the palsy which arises from lesion of the parts constituting the corpus striatum. This peculiar form of paralysis is of the greatest practical importance, for I have calculated that on the average about 25,000 persons die of it annually in England and Wales, while at least twice as many people living in this country suffer from the effects of it. I have already stated that this form of paralysis is always owing to disease of the blood-vessels supplying the corpus striatum, and I must therefore now say a few words on the peculiarities of these latter.

Fig. 3.—A C. Sphere of the anterior cerebral artery. M C. Sphere of the middle cerebral artery. P C. Sphere of the posterior cerebral artery. C N. Caudate nucleus. L V. Lateral ventricle. I C. Internal capsule. L N. Lenticular nucleus. CL. Caudate nucleus. Ch. Chiasma of the optic nerves. I R. Internal carotid artery. 1. Internal carotid artery. 2. Middle cerebral (Sylvian) artery. 3. Lenticulo-striated artery.

The arteries which nourish the central ganglia arise from the circle of Willis, the middle cerebral or Sylvian artery being the most important of them. This vessel supplies the lenticular nucleus, the internal capsule, and the larger portion of the caudate nucleus; indeed, only the head of the latter receives blood from the anterior cerebral artery.

Cohnheim has drawn attention to the circumstance that most of the smaller cerebral arteries are what he calls "Endarterien", or terminal vessels; *i.e.*, they do not anastomose with others, but turn straight into capillaries. This is the reason why we meet, in the central ganglia as well as in the cerebral cortex, with extremely localised softening, one vessel being quite independent of the other. Indeed, they can all be separately injected, but the whole of the corpus striatum cannot be injected from any one of them. Softening of the entire caudate kernel has, for the same reason, never been observed.

That hæmorrhage should occur so frequently in the central ganglia is explained by the fact that their arterioles are under the direct influence of the pressure of the left ventricle, the road from the heart to the corpus striatum being short and straight. Moreover, as these vessels do

not anastomose, but are terminal, an excess of pressure in them cannot be well relieved, but must expend its force on the coats of the vessels, thus causing, in the end, aneurysmal dilatation, with eventual rupture and escape of blood into the brain-matter. Embolism occurs also, for anatomical reasons, with preference in the middle cerebral artery on the left side. An excrescence, detached from the valves of the heart, is driven from the aorta straight into the left carotid, proceeds unchecked through the circle of Willis, but becomes impacted in the middle cerebral, as the calibre of that vessel is not large enough to allow it to pass further on.

The peculiar distribution of the blood-vessels in the corpora striata explains likewise why hæmorrhage *within* their structure is rare, and why it takes place so frequently just *outside* them. The artery which is chiefly in fault here is one of the lenticulo-striated arteries, coursing between the lenticular kernel and the external capsule, and is so frequently affected with aneurysmal dilatation, leading eventually to apoplexy, that Charcot has called it the cerebral-hæmorrhage-artery. When it is ruptured, the blood which escapes from it is found to perform a kind of natural dissection of brain-matter in the direction of the least resistance. The external portion of this region, which consists of the island of Reil, the claustrum, and the external capsule, is spared by reason of its greater cohesion; while, on the inner side, the grey nuclei are pushed back towards the lateral ventricles, into which the blood will make its irruption whenever the hæmorrhage is very profuse. In such cases, we find generally, in addition to the ordinary symptoms of apoplexy, early rigidity of the paralysed muscles, epileptiform convulsions, and vomiting—which have the worst prognostic significance. Such patients often die within a day or two, without recovering consciousness.

A second class consists of those in which there is localised softening in the grey kernels, or where the hæmorrhage is so limited in extent that, although it displaces and pushes them against the ventricles, it does not tear up the internal capsule. In some cases, indeed, the pathological lesion is so slight, that there are hardly any, or no symptoms at all, during life. Thus the head of the caudate nucleus has been found softened after death, without there having been loss of motor power. But where the lesion is somewhat extensive, crossed incomplete hemiplegia is the result. In some cases, only the lower branches of the portio dura have been found paralysed, while the arm and leg escaped; in others, there have been paralysis of the arm and paresis of the leg. In all cases, however, it is found that the paralysis is transitory, and that after a few weeks or months the patient recovers the power over the affected side to a great extent. We are at present unable to distinguish, during life, lesions of one grey nucleus from those occurring in the other; but where hemiplegia remains more or less permanent, and is after a time followed by late rigidity of the paralysed muscles, it is certain that the anatomical changes have not been limited to either of the grey kernels of the corpus striatum, but have also invaded that portion of the internal capsule which contains the pyramidal strands, thus severing the connection between the Rolandic convolutions and the extremities.

It seems at first sight singular that disease of the grey centres should cause less severe symptoms than destruction of white matter, which does not generate, but only conducts power. This fact, however, becomes easily intelligible, if we consider that there are several grey centres, while there is only one conducting strand. The principle of compensation or substitution applies to the several grey kernels. If the influence of the lenticular nucleus be removed, the patient may fall back on the caudate nucleus and the hemispheres. No doubt there is a loss, which is more or less seriously felt at first; but, as time goes on, and the will of the patient is more directed towards the remaining sources of motor force, these latter undertake to supply the missing influence, and the patient is ultimately found not to be much worse off than he was before the attack. As soon, however, as the internal capsule is destroyed, a chasm is made between all the grey centres generating motor influence on the one hand, and the portio dura and the extremities on the other hand, which cannot be bridged over. Similarly, in a galvanic battery, a few pairs of plates may become rusty and unserviceable, yet the instrument may continue to be of service if there are other plates which continue in a proper electro-motive condition. But let the conducting wires of the battery be destroyed at any one point, and no more influence can be transmitted to the electrodes, however perfect may be the condition of the generator of force itself. Destruction of the internal capsule by hæmorrhage or softening is unfortunately a very frequent occurrence; and only regeneration of nerve-fibres in the capsule could, by re-establishing the conduction between the centre and the periphery, lead to the cure of the paralysis in such instances.

A description of the attack of paralysis or apoplexy does not enter into my present purpose. I therefore take up the patient at the time

when he has recovered from the more immediate and threatening symptoms, and where it appears plain that his life is not in any immediate danger, but that he is disabled by paralysis of the arm and leg, to which is added loss of power in the inferior branches of the *portio dura*. To these initial symptoms, however, others are gradually added, which impart a peculiar and characteristic aspect of the case.

A feeling of stiffness, which differs from the powerless feeling of the first few weeks, becomes noticeable in the paralysed muscles a month or two after the stroke. This is particularly experienced in the flexors of the forearm; and these muscles gradually assume a rigid condition. The fingers then appear flexed into the palm of the hand; and sometimes this contraction is so extreme, that the nails cut into the flesh. The thumb is flexed, and at the same time so strongly adducted, that it almost or quite disappears under the other fingers. When this rigidity is once fully established, it is difficult to overcome it by force, as the fingers offer the greatest resistance to any attempt at opening the hand of the patient. The forearm is generally pronated; the elbow is in a state of semiflexion, by rigidity of the biceps and brachialis internus; the arm is adducted to the side by contraction of the coraco-brachialis and the pectoralis major. The shoulder is sometimes raised by contraction of the trapezius, or depressed when there is paralysis without rigidity in that muscle. A certain degree of contraction, however, is mostly present in all the muscles. For instance, when the elbow is in a state of semiflexion through rigidity of the biceps, the triceps is, for that reason, not relaxed; for, on attempting to produce complete flexion of the forearm, so as to place the fingers in the acromion of the same side, resistance to this movement is experienced, showing that the triceps is likewise rigid. It is therefore simply a question of degree between the flexors and extensors; and this accounts for the circumstance, that it is sometimes difficult to make any considerable change in the position of the limb.

The contraction is generally less in the lower than in the upper extremity. It may appear in the hamstring muscles of the thigh, and the flexors of the leg; and where this reaches an extreme degree, it renders walking quite impossible, the leg being flexed on the thigh, and the thigh on the pelvis; so that the heels touch the buttocks. More frequently the rigidity invades the extensors and adductors of the leg, and causes the appearance of *pes equinus*. The patient then walks on tiptoe, and, in order to diminish the dragging of the foot on the ground, instinctively swings the leg from without inwards, which constitutes a peculiarly characteristic kind of gait.

In the face there is also a certain amount of rigidity, which is more particularly noticed when the patient is talking, laughing, or crying. While at first, when there is simple palsy, the commissure of the lips is depressed, it becomes later on raised on the paralysed side. The nasolabial sulcus appears more marked; the nostril dilated; and the eye looks smaller, from rigidity of the orbicularis.

Muscular stiffness is greater when the limbs are cold than when they are warm. For this reason, the hand appears more open when the patient is in bed, and the arm is kept under the bedclothes, than when he is up. It is also greater in the waking state than during sleep; and this I believe to be due to the circumstance that emotions have a tendency to increase it, so that their absence during sleep renders the contraction less marked. The rigidity is also exaggerated by efforts to use the paralysed muscles; by injuries such as a blow or a fall; by faradisation of the skin; by the old-fashioned method of using electro-magnetism, which consisted of making the patient take hold of two metallic handles with both hands, and thus sending a powerful current right through the body. Duchenne has related the case of a student who treated himself in this fashion, and in whom the exciting effect was so great as to cause a fresh paralytic attack. The administration of strychnia, which was formerly much in vogue for the treatment of this form of paralysis, likewise tends to increase the rigidity; and, if it be pushed so far as to cause convulsive movements in the muscles, these are more violent in the paralysed than in the healthy side. Anything and everything, therefore, which has in a general way the tendency to exaggerate the reflex excitability of the cord will increase the rigidity of the paralysed muscles.

What is the cause of this peculiar symptom? It was formerly believed to be owing to inflammation of the brain at the seat of the lesion, which was thought to follow habitually upon an attack of apoplexy; but the changes which really follow an effusion of blood are not of an inflammatory character. Nor is it due to the shrinking of the cerebral cicatrix, as Dr. Todd believed; nor to peripheral neuritis, which has been assumed to occur by other observers. Neither can we explain it by an increase of reflex excitability, owing to the removal of the inhibitory influence of the brain; for the influence of the brain upon the paralysed limbs is removed immediately after the attack, while rigidity comes on a month or two afterwards. Pathological anatomy

has, however, clearly shown that destructive lesions of the motor centres, and more particularly softening and hæmorrhage affecting the conducting paths of motor power in the white internal capsule, give rise to a peculiar form of chronic myelitis leading to sclerosis of the pyramidal strand corresponding to the affected side, in which, after death, wasting of nerve-tubes and increase of interstitial connective tissue are discovered.

The degree and extent of this sclerosis varies. In some cases, the whole texture of the strand has been found utterly destroyed, there being no nerve-tubes left, but only amyloid corpuscles and proliferated connective tissue; in such, the muscular rigidity is extreme, and affects all the muscles of the paralysed side indiscriminately. In other cases, the degenerative process appears to be more limited, there being white patches interspersed in the grey mass, which contain healthy nerve-tubes; in these, the contraction is not so marked, and the limbs are proportionately more useful to their owners.

The sclerosed appearance of the pyramidal strands is, on the *post mortem* table, seen to commence immediately behind the lesion, and then follows the anatomical distribution of the strand as I have sketched it above. It appears particularly striking in the *crus cerebri*, which is altogether smaller than the one on the healthy side, the alteration being chiefly marked in the middle portion of the *crus*. In the *pons Varolii* it is not so obvious, except at its lower surface where it joins the medulla oblongata, but becomes clearly apparent again in the anterior pyramid, where it crosses over to the opposite side of the cord, and runs down in the lateral column until it reaches the lumbar enlargement. This sclerosis, which is really a chronic inflammatory process, leads to irritation in the large motor cells of the anterior horns of the spinal cord, and thus causes that contracted condition of the muscles, which is in reality an exaggeration of their normal tonicity, this latter being under the direct influence of those ganglionic masses. Irritation of the horns, however, is a very different thing from actual disease or destruction of them; and it is only in exceptional cases of cerebral paralysis that these horns become really diseased in the further course of the affection.

In the vast majority of cases there is no muscular wasting, except what may be accounted for by disuse. It is indeed singular to see patients who have been deprived of the power over one side of the body more or less completely for five or ten years, and more, and in whom, nevertheless, the muscular tissue is fairly nourished, and where faradisation causes at once a ready response by inducing electro-muscular contractions. Compare with such cases those of infantile paralysis, where the muscles waste away, and lose their faradic excitability, within a few weeks; and an enormous difference is at once seen, which is, however, satisfactorily explained by the anatomical data which I have just given.

In some cases, however, the sclerosing myelitis spreads from the lateral column to the anterior horns; and we then notice a corresponding change in the symptoms. As the centre of tonicity is gradually being destroyed, the rigidity of the muscles diminishes *pari passu*, and ultimately vanishes altogether. When the muscles become more relaxed, the patient is apt to think that he is getting better; but such hopes are delusive, for the limbs become, on the contrary, more feeble as time goes on, and the muscles are found to lose their faradic excitability. This process goes on much in the same order as we see it in progressive muscular atrophy, affecting first the muscles of the shoulder and the hand, and afterwards other groups of muscles in succession. This is generally protracted over two or three years; and shooting pains are often felt in the muscles when the wasting commences. All grey cells being connected, the process may spread from the anterior to the posterior horns, or to the anterior horns of the opposite side by means of the anterior commissure; and we have then no longer hemiplegia, but paraplegia with muscular atrophy. In *post mortem* examinations of such cases, the anterior horns have appeared shrunk in size; some of the large cells were gone altogether, while those which remained looked sickly, yellow, and granular, and had no proper nuclei or Deiters's elongations. The gelatinous matter surrounding them likewise appeared disintegrated, granular, soft, and unresisting. The so-called intermedio-lateral cells, which are believed to be centres of vaso-motor action, have likewise occasionally been found destroyed.

I must now ask your attention for a short time to a set of symptoms which are produced by experimenting upon the patient in a certain fashion; and which, although they are often thought to have only a theoretical and scientific interest, are also, as I hope to be able to show, of importance in a diagnostic and therapeutic point of view. This inquiry takes us into the region of the so-called tendon-reflexes, which have of late years absorbed a considerable amount of professional attention, and on which I will now only touch in so far as they concern the particular condition which is now under consideration.

By examining the paralysed side of the patient in a peculiar manner, we may discover the first onset of sclerosing myelitis before actual

muscular rigidity has commenced. This is done in the lower extremity by quickly lifting the fore part of the foot with one hand, while the flabby leg is supported with the other hand under the knee-joint of the patient. We thereby produce what is called ankle-clonus, or foot-phenomenon, or induced trepidation, or spinal epilepsy; that is to say, a series of rhythmic muscular movements of the paralysed leg, which go on for a time varying from half a minute to five minutes and more. Such movements cannot be induced in a healthy subject, and in the hemiplegic only about a month or two after the attack. They may be increased by the administration of strychnia, and anything else that causes an exaltation of reflex excitability; while they are diminished by bromide of potassium, and anything which has a tendency to subdue that faculty. We also find that, in the knee, the patellar tendon-reflex is increased; and in the upper extremity, percussion of the tendon of the biceps causes flexion of the forearm; of the triceps, extension; and of the tendon of the extensor communis digitorum at the wrist, extension of the latter.

That these phenomena are owing, not to mechanical stimulation of the muscles, but really to a reflex action which takes place in the cord, has been so firmly established that I do not think it necessary to dwell on this point. I will only say that the time which is required for the stimulation to travel from the tendon to the cord, and from the cord back again to the muscle, amounts in the healthy man, on the average, to 50-1000ths of a second; while whenever there is irritation in the cord it is a good deal less—something like 34-1000ths. Researches on this point have been made by Tschirjew, Gowers, and Brissaud, the results of which do not quite agree; and the subject is worthy of further investigation. An interesting fact is that in hemiplegia with late rigidity, the time required for the reflex to become visible appears also to be less in the healthy limb, so that the healthy side does not appear to be entirely healthy.

Another point of interest connected with this subject is, that apart from the reflexory movement itself, certain other effects are apt to occur, from percussions of tendons in hemiplegic patients; viz., pain in the lumbar, dorsal or cervical region, a feeling of constriction in the throat and chest, tremor, and a general sensation of *malaise*. Brissaud, who has written an able monograph on late rigidity, has even seen it lead to fainting, aura epileptica, and true epileptiform convulsions. In hysterical women who are in a constant state of exalted reflex excitability, the same proceeding may, if frequently repeated, lead to permanent muscular contraction; and it is, therefore, incumbent upon the practitioner to exercise great prudence in using this test as a means of diagnosis, for fear of doing harm to his patients. Some time ago, when I had to examine a patient suffering from spinal disease, he begged me to take it for granted that the patellar reflex was increased, as the repeated knocks he had had on the patellæ, generally made him quite ill for the whole day.

Are there any cases of permanent hemiplegia without rigidity? This may well be doubted. Of course I do not speak here of lesions of the grey kernels simply, where the internal capsule remains unaffected; for in these cases the paralysis mends so much soon after it has been established, that we can hardly call them hemiplegic two or three months after the attack, when there remains simply a degree of awkwardness in using the fingers, but no actual paralysis. The patient then uses his Rolandic convolutions where formerly he used the lenticular and caudate nuclei; and more conscious attention and trouble is therefore required than before the stroke. Where, however, the internal capsule has been injured, and the affection is, therefore, more severe, there is probably always at least some degree of contraction, which is sometimes, indeed, barely appreciable when the limb is quiet, but which becomes at once obvious when the patient is requested to make an effort, however slight. Thus, for instance, he may be able to move his arm and hand in all directions; but when told to pick up a pin, or to unbutton his coat, the fingers and hand are seen to contract, rendering such manipulations difficult or impossible. On examining the state of reflex excitability, that of the tendons is found to be increased, and there is no difficulty in inducing spinal epilepsy. The time of menstruation in women is particularly favourable for the production of these phenomena—no doubt owing to the excitability of the cord being still further exaggerated during that period.

Time warns me that I must not tarry on this subject any longer; nor shall I be able to speak to-day of disease of the Rolandic convolutions, and the cortical paralysis which is owing to it, but will at once proceed to consider the prognosis and treatment of paralysis from corpus striatum hæmorrhage.

The *prognosis* of this form of paralysis is only favourable when the affection is limited to the grey kernels of the corpus striatum, while total destruction of the internal capsule generally leaves the patient permanently disabled. The amount of damage done, either by soften-

ing or hæmorrhage, is of course of the greatest consequence, as lesions of slight extent may be more easily repaired than those occupying a large area. Age and constitution of the patient have that influence which must be accorded to them in all diseases; and, finally, much depends upon the treatment which is adopted at an early stage of the malady, and chiefly before sclerosis has been fully established. With regard to this point, the occurrence of ankle-clonus after an apoplectic or paralytic seizure should be carefully watched for, as it is a diagnostic sign of the utmost value, leading us to predict the probable speedy appearance of late rigidity in the paralysed muscles; and, as soon as that symptom has made its appearance, no time should be lost in resorting to special therapeutical measures.

The *treatment* of cerebral paralysis would, *prima facie*, appear to have but slight chances, as in one class of cases there is a natural tendency to recovery of function by compensation of allied structures, while in another the destructiveness of the original lesion and its consequences would seem to render all therapeutical effects futile. The question of treatment has, therefore, been disregarded by many of those who have been most forward to investigate the pathology of these conditions. Nevertheless this must, for all time to come, be the chief point of interest for the practitioner. No doubt some cases are incurable; yet I think I shall be borne out by those most conversant with this subject when I say that few cases are met with in practice which may not be improved by judicious treatment.

Are there any means at our disposal to repair the lesion in the internal capsule, which we have seen to be the chief obstacle to recovery? That repair of destroyed nerve-fibres is not actually impossible has been shown not only by experiments on animals, but also by cases of neurectomy for facial neuralgia, in which at first anaesthesia was caused by the operation, from loss of nerve-substance, amounting in some cases to as much as three-quarters of an inch; and where this was eventually recovered from, and the paroxysmal pain, which had disappeared after the operation, returned in the same sphere after a time. This points in the most unmistakable manner to repair of nerve-fibres. Can part of the capsule be repaired in a similar manner, and thus the influence of the volitional centre of motor power in the hemispheres again be brought to bear upon the paralysed extremities? This question cannot be at present answered, as there is not yet sufficient clinical and pathological evidence for that; yet I have had a very considerable number of cases under my care in which such an occurrence appeared to have taken place to some extent.

The remedies I have used for this special purpose have been: first, phosphorus, sometimes simply dissolved in a sufficiency of oil, and administered in a capsule or perle twice a day, in doses of one-thirtieth of a grain, and sometimes in cod-liver oil, where the general state of the patient seemed to render the latter medicine advisable; and, second, the constant galvanic current, sent transversely through the brain by one electrode being placed to the right and the other to the left mastoid process, and also longitudinally by one being placed to the forehead and the other to the occiput. The cathode, which is the more stimulating of the two poles, should be nearest to the seat of lesion; so that, for instance, in right hemiplegia, the cathode is placed to the left and the anode to the right mastoid process. Such applications are made daily, the current being allowed to pass for from five to ten minutes at a time. Later on, local applications of the current to the paralysed parts are combined with this. These latter are not so much intended to act on the motor nerves and muscles as on the sentient nerves, by which the stimulation is reflected to the suffering centre. Faradisation is also sometimes useful, and should be employed to the antagonists of the contracted muscles.

Our second object should be to counteract the sclerosing myelitis which is apt to follow the attack. For this we have several remedies, which, if used at a sufficiently early stage, have appeared to me useful. These are principally the salts of gold and silver, and ergot of rye. Gold is given either as chloride of gold, or better still as chloride of gold and potassium, in doses of from one-eighth to one-half a grain, in pills. Silver is given as the oxide, nitrate, or phosphide, in doses similar to those of gold. Ergot of rye I generally give in the form of the liquid extract, in doses of from half a drachm to a drachm three times a day. A good plan is to give a gold-salt for a month, a silver-salt for a month, and ergot for a month. That remedy under which most improvement appears to take place may afterwards be continued, according to the special features of the case.

Iodide of potassium does no good in embolic softening, but appears useful in promoting the absorption of the clot after hæmorrhage. This pathological process is generally finished in about six weeks after the attack, and it is rare to see much good resulting from that remedy at a later stage of the disease. Bromide of potassium, on the other hand, is useful when reflex excitability appears much increased.

A word must be said about the employment of strychnia in these cases. This is a remedy for paralysis hallowed by tradition; yet nothing could well be more unphilosophical, or indeed hurtful, to our patients than to sanction or to continue such a practice. Sclerosing myelitis being the natural sequence of paralytic attacks strychnia is certain to increase the exalted state of excitability in the spinal cord, and thus to render the muscular rigidity more intense. It is therefore a medicine which should never, on any account, be given in such cases.

The general health has, of course, carefully to be attended to at the same time. We must remember that some of them—viz., the subjects of embolism—suffer from heart-disease; while others, in whom the attack has been owing to hæmorrhage, are probably affected with small aneurysms in different portions of the brain, which may burst subsequently, and give rise to fresh attacks. Straining at stool is dangerous for such persons, and the action of the bowels should therefore receive great attention. The *morale* of the patient is often improved by change of air and scene, as soon as he is sufficiently well to bear a journey. We cannot expect much from mineral water cures in the further progress of these cases. Wildbad, Gastein, Buxton, and other spas have had a reputation in the treatment of cerebral paralysis, which has, however, not stood the test of time and strict criticism; and it is, on the whole, better to send such patients to places which are easily accessible, where all comforts can be procured, and which possesses well known climatic advantages. A moderately dry bracing air is found to be particularly suitable for these patients; but high elevations must be avoided.

Shall we, then, ever succeed in removing the shadow of that dire disease—

"Paralysis—grim foe to men of mind—
That lies in ambush for them treacherously,
And lays them prostrate down as with a mace?"

Such, no doubt, would be too much to expect; yet, even now, when a treatment really in accordance with pathological conditions has only just been sketched out, it is found that, with scarcely any exceptions, our patients are very much the better for it; and that great further progress will be made in time to come in our management of these cases, is a hope which will surely not be disappointed.

ABSTRACT OF LECTURES

ON THE

ANATOMY, PHYSIOLOGY, AND ZOOLOGY OF THE CETACEA.

Delivered at the Royal College of Surgeons of England.

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LECTURE VIII.—THE ANATOMY OF THE PORPOISE (*continued*)— DOLPHINS, ETC.

THE larynx and trachea are somewhat similar to those of the whalebone-whales, but the superior extremity of the arytenoid cartilages are flattened out into a club-shaped end, and there is no air-pouch in front of the trachea, such as is found in the *Balanoptera rostrata*, with which the larynx communicates; or, if present, it is quite rudimentary. It is believed by many naturalists that the opening of the larynx remains grasped by the posterior nares behind the velum palati, not only during the act of respiration, but also during deglutition; and that the food is passed backwards into the pharynx on each side of the larynx. If such is the case, it is difficult to understand how the grampus, which has a larynx very similar to that of the porpoise, is able to swallow large animals—such as seals, etc.—entire, or nearly so, as it frequently does. The olfactory nerve is absent in the porpoise, and there is no cribriform plate, both of which structures are very fairly developed in the whalebone-whales, especially in the Greenland right whale. The vascular system is arranged in exactly the same manner in the porpoise as in the *Mysticeti*. There are large arterial plexuses situated in the neck, in the expanded foramen for the entrance into the mandible of the inferior dental branch of the fifth nerve, below and on each side of the dorsal vertebrae, especially in connection with the intercostal arteries, and in the caudal region in the arches formed by the chevron bones. The kidneys are extremely lobulated, resembling bunches of grapes. The lobulated form of the kidney is a character found in many mammals, as the bear and seal, but is more exaggerated in the cetacea than in other members of the class Mammalia. The organs of generation differ in no respect from those of the *Mysticeti*. The penis is very much curved, and is held in that position by a well-developed retractor muscle. The prostate gland is large; the vesiculæ seminales and Cowper's gland

are absent. The mammary nipples in the male are situated in a cavity, which opens externally as a small median slit, situated between the anus and the opening through which the penis is protruded externally. In the female, the nipples are larger, and are situated in two small cavities, which open externally on each side of the vulva. The mammary glands have large ducts, which act as reservoirs to contain milk; by this arrangement, the difficulty of lactation in the water is overcome, the animal being able to inject a large quantity of milk previously collected in the ducts rapidly into the mouth of its young. The placenta is diffuse and non-deciduate, as in the pig and horse, with some slight modifications.

The family of toothed whales most nearly allied to *Phocena* is the *Delphinidae*, or dolphins. The common dolphin (*Delphinus delphis*), which is much more rarely caught on our coasts than the porpoise, closely resembles the latter, and is frequently mistaken by fishermen for it. It differs from the porpoise in having a longer head; the rostrum is much more elongated, and projects suddenly in front of the lump of fat on the top and anterior part of the head. It has a number of very sharp conical teeth, which are exactly alike at all parts of both upper and lower jaws. The anterior limbs are longer than those of the porpoise, and the dorsal fin is high and sickle-like. The largest member of the family is the *Delphinus tursio*, an animal which was known to Hunter, and of which there is an articulated skeleton and several skulls in the museum. The geographical distribution of the *Delphinidae* is very wide, they being found in all seas, and even in some rivers. The chief subdivisions into which they may be separated are *Steno*, *Tursiops*, *Prodelphinus* or *Clymene*, and *Delphinus*.

The *Globicephalus* is another member of the group of toothed whales which has very distinct characters. It is very common in the northern seas, where it is usually found in shoals. It occurs on the shores of the Orkney and Shetland Islands occasionally, but is frequently found around the Farøe Islands, where large numbers are killed almost every year by the inhabitants. When a shoal approaches the coasts, it is at once pursued by the fishermen, who frighten the animals, naturally shy timid creatures, till they drive them aground, and then kill them with lances. The name *Globicephalus* was given to the animal from the external appearance of the head, which is of a very round shape, from the contour of the lump of fat on the anterior part of the upper surface of the skull; the skull itself is flat, however, and broad. The dorsal fin is high; the anterior extremities appear to be very long and narrow, from the great length of the manus, the central digit of which has as many as thirteen or fourteen phalanges. There are five bones in the carpus and five digits. The teeth are very small in proportion to the size of the animal, and confined to the anterior half of the upper and lower jaws. They are very loosely set in their sockets, and, when the skull is macerated, are frequently lost. Strong teeth are not required, as the animal lives mostly on squids and cuttle-fish. The cervical vertebrae are ankylosed together; the sternum is well formed, and has several sternal ribs articulated to each of its sides. The *Globicephalus* is found in all seas, from Australia and New Zealand to the north of Europe; and all forms seem to be very much alike.

The *Grampus* is a toothed whale resembling the *Globicephalus*. Its anterior extremity is not so long, and is less modified; the lump of fat on the top of the head is not so large as in the *Globicephalus*. It is peculiar in having no teeth in the upper jaw, and only a few confined to the anterior half in the lower jaw, and in being of a peculiar colour, with a number of whitish lines on the under surface of its body unsymmetrically arranged.

The largest and fiercest form of toothed whale is *Orca*. Hunter dissected a specimen of this animal, which was taken in the mouth of the Thames, and is now mounted in the Museum. It is occasionally caught on the British coasts. The dorsal fin is sword-shaped. The manus is very broad, all the individual bones being broader than they are long. The animal is white-coloured below and dark above. The skull, which is very similar to that of the *Globicephalus* and *Grampus* in its general characters, is usually slightly unsymmetrical, and the premaxilla of the right side is usually somewhat higher than that of the left. The teeth are large and well developed. The animal lives on seals and large fish; it even eats other animals of its own order. The stomach is very large, and has been found to contain several seals and porpoises at the same time.

Nearly allied to the *Orca* is the *Pseudorca crassidens*, which was first described as an extinct animal by Owen from a semifossil skull found in Lincolnshire, which has unfortunately been lost. Some years after Owen described his specimen, Professor Reinhardt obtained the skull of an animal caught in the Baltic, which he found to be identical with the one described by Professor Owen. Nothing further of this species was seen till a few years ago, when Dr. Crowther sent home two skulls,