THE CROONIAN LECTURES ON THE INFLUENCE OF THE CIRCULATION UPON THE NERVOUS SYSTEM.

Delivered before the Royal College of Physicians.

By WALTER MOXON, M.D., F.R.C.P.,
Physician to Guy's Hospital.

LECTURE III.—PART I.

IN my last lecture, I endeavoured to show that certain special diseases, described in our text-books as congestion of the brain, must be referred to other causes, and, indeed, chiefly to conditions the very reverse of a state of congestion; and I gave my reasons for believing that there is not any such disease as congestion of the brain, in the sense of determination of blood to the head—or, in other words, congestion as a cause or onset of attacks of diseases such as described in some of our leading works under several distinct forms, does not exist. Neither pathological examination of persons dying under such conditions, nor the clinical symptoms described, afford any proof that a congested state of the brain even attends such illness, let alone its giving rise to it. The supposition that the blood causes such symptoms arose from views regarding the cerebral circulation, which views are obsolete.

Meantime, whilst such obsolete views of the cerebral circulation have entailed upon us the confusing doctrine of congestion of the brain, modern views of the vascular mechanism have in their turn given rise to speculative explanations of nervous disorders, which greatly and increasingly influence present teaching upon diseases of the nervous system. I refer in particular to the widely spread assumption that epilepsy and other convulsive disorders are caused, or initiated, by spasm of the arteries of the brain. I will, with your permission, now examine the grounds of such an opinion, and afterwards consider the relations of angina pectoris to high tension in the arteries.

Any explanation is so much better than none, that the explaining power of a new view is apt to purchase condonement of its unreality. Only let us, with Kussmaul, assume that he produced epilepsy in animals by ligaturing all the cervical vessels, and then—if, indeed, we can further justly persuade ourselves with him that the arteries of the brain are able to contract universally through an impulse sent to them along the vaso-motor nerves, so that the brain is rendered entirely anæmic, as if the cervical arteries were all ligatured—this would undoubtedly make epilepsy a very comprehensible thing: reducing the whole highly complex set of actions to dependence upon a single impulse from the vaso-motor centres. Even if we do not accept the theory of entire anæmia, but take the same idea as modified by Schröder, and now taught as the fashionable theory of epilepsy—namely, that the vaso-motor nerve carries forth a contractile influence to the arteries of the brain, which blanches it enough to produce unconsciousness, we make a step which invades the mystery of epileptic convulsions, and which would put at least a part of it into continuity with the paleness and motion, and other familiar things of the kind.

If we go further, and suppose with some writers who have devoted special attention to nervous diseases, that in an epileptic convulsion the tonic stage is caused by tonic spasm of the arteries, and that each jerk of clonic spasm in the subsequent convulsion is due to a corresponding short spasm of the arteries of the brain, we can head off the epileptic paroxysm in hydraulic changes; and this appears to afford a certain satisfaction, which may be taken for an explanation of the subject. Indeed, in the supposed power of rapid and entire closure of all the cerebral arteries, or the supposed power of rapid closure of any of them under the influence of the sympathetic, we have a handy method of unlocking almost any difficulties as to nerve-action; and, if you will only imagine that the arteries close or open to the play of sympathetic influence, like mysterious doors in enchanted forests, to the utterance of "open sesame," many otherwise impenetrable obscurities seem made free to the entry of light.

Now, there is no doubt that the arteries, and especially the smaller arteries, are provided with muscular fibres in their walls, and that these muscular fibres contract and cause the arteries to narrow their channels. But what we have to do with, is the degree in which this power of contraction is under the influence of the vaso-motor nerves. If it were

really the case that the muscular activity of the arteries is entirely dependent upon the action of the vaso-motor nerves, the powers of these nerves would stand in a much clearer light. But the contraction of the arteries serves purposes with which the nerves have nothing to do, and over which the nerves have no power; for it has been shown that the contractions of the arteries will proceed just the same when the vaso-motor nerves are destroyed. Thus the remarkable rhythmical contractions and dilations of the arteries in the rabbit's ear are independent of the vaso-motor nerves, for they continue when the cervical sympathetic is divided; and even, according to Dr. Michael Foster, when the cervical ganglion is extirpated. Again, Kussmaul relates that after section of the cervical sympathetic nerve he has seen the arteries of the rabbit's ear contract, and drive their contents into the veins. Yet no ganglionic mechanism has been found in the rabbit's ear to account for these effects by nervous action. From such facts it follows that the muscles of the arteries possess and exercise their powers in a way independent of the vaso-motor centres, and that their contractions are not organised only to answer to those centres, but that the muscularity of the arteries fulfils functions independently of the play of the vaso-motor nerves upon them. This would lead us to anticipate limits to the power of the vaso-motor nerves over the arteries.

And, indeed, if we study the results of the various experiments which have revealed the intricacy of the vascular mechanism, we find some points appearing which are important for our question. The first point is that, when arteries are subjected to irritation so as to cause them to contract, the contraction is not instantaneously rapid, but measurably slow. On this point there is a very general agreement among observers. Reid, in his *Physiological and Anatomical Researches*, p. 6, says:—"The contractility of the arteries manifests itself by slow contraction, followed by equally slow dilatation". Carpenter (p. 330, edition 1876), quoting Weber, says:—"The electric stimulus is most effectual when applied by the magneto-galvanic apparatus; when the minute arteries of the mesentery of frogs, between one-seventh and one-seventeenth of a Paris line in diameter, are thus stimulated, they do not immediately respond to the irritation, but begin to contract after a few seconds, so that their diameter in from five to ten seconds is diminished by a third, and their sectional area consequently reduced to about half; by a continued application of the stimulus, the calibre is so much reduced that only a single row of corpuscles can pass, and at last the vessels become completely closed, and the current of blood arrested, the original conditions being restored on the cessation of the electric current". Paget (*Surgical Pathology*, p. 225), says:—"If, as one is watching the movements of blood in a companion artery and vein, the point of a fine needle be drawn across them three or four times without apparently injuring them, or the membrane over them, they will both presently gradually contract and close.....their contraction, which follow at some interval after the application of the stimulus, and is slowly accomplished, it shows that their power of contraction is like that of parts with smooth or organic muscular fibres". Michael Foster says:—"By galvanic, and still better by mechanical stimulation, this muscular coat may, in the living artery, be made to contract. During this contraction, which has the slow character belonging to the contraction of all plain muscle, the calibre of the vessel is diminished". I have made many observations upon the contraction of arteries as caused by direct stimulation, in watching the development of the inflammatory process, and have observed them to contract slowly. I never saw complete contraction in less than six seconds; but I have never witnessed the effects of stimulation of the vaso-motor nerves. It is antecedently improbable that if the contractions of arteries are slow, when caused by direct stimulus, they would be rapid under indirect. Dr. Burdon Sanderson, in the *Handbook for the Physiological Laboratory*, p. 237, when speaking of the direct excitation of the spinal cord by galvanism in the frog, says:—"On opening the key for a moment, so as to allow the induced current to pass through the needles, it is seen that all the arteries of the web at once contract, the contraction increasing for four or five seconds, and then gradually subsiding. If the excitation is continued for several seconds, the circulation stops." I have here a copy of a diagram from Dr. Michael Foster's work, which is, perhaps, more to our purpose, as it shows an instance of the rate of arterial contraction in a mammal. It is a manometer-trace of the fall of blood-pressure caused by galvanising the depressor nerve in the rabbit; but it is said in the text that galvanising the excitator nerve will produce, sometimes, the exact reverse of this trace. The slow progress of the contraction is evident when we see it take thirteen seconds to complete the action, and that this action is nowhere sudden, but is gradual throughout. This corresponds with the usual characters of contractions of plain muscular fibre. My first proposition, then, is that the contraction of arteries is slow, requiring from four or five to thirteen seconds for its completion.

I will next inquire what is the degree of completeness of the contraction of arteries as caused through the vaso-motor nerves. We have seen that an entire closure of the arteries may be caused in the web of the frog's foot by stimulation of the spinal cord; but it must be observed that in cold-blooded vertebrates the several parts of the vascular system stand in somewhat different relations to those they bear in warm-blooded animals. In fishes, the veins have pulsatile sacs upon them. The veins, as well as the arteries, of the frog's foot will close on application of a direct stimulus. Frogs have lymphatic hearts. In short, there is less differentiation of the vascular mechanism in such animals: the heart does less, and the vessels more, in the work of circulation. If, instead of such remote creatures, we take examples from amongst warm-blooded vertebrates, the development of whose vascular mechanism is more like that which belongs to the human organism, we find that the powers of the vaso-motor nerves are moderate.

There appears evidently, in the description of the results of the stimulation of the vaso-motor nerves, a much less extent of the contraction produced, than there is of certain and large dilatation of the vessel on section of the nerve. Section of the vaso-motor nerves dilates the arterioles very decidedly; and when the vessels are thus dilated, if an interrupted current is sent through the distal segment of nerve, such a current as would tetanise a muscular nerve, the result, according to Dr. Michael Foster, is to cause the arteries to contract "to or beyond" their normal calibre. This would lead to the inference that stimulation of the vaso-motor nerves in the most violent way will do little more than counterpoise the effect of total paralysis of the nerve. In one of Ludwig and Cyon's experiments, cited by Dr. Burdon Sanderson, the normal arterial pressure was 90 millimètres; after division of the splanchnic nerve, the fall was to 41 millimètres; during excitation of the peripheral end of the nerve, 115 millimètres: thus the rise on stimulation did not exceed the normal by half the extent of the fall on division of the nerve. In the case of the rabbit's ear, after the vessels have been made very visible by the dilatation of them consequent upon division of the cervical sympathetic, the same authority says: "If now the peripheral end of the divided nerve is placed between the copper points, and the key opened, the artery contracts, and the congestion disappears". Dr. M. Foster says a galvanisation of the peripheral end of the nerve will cause the rabbit's ear to "become pale, much paler than normal; if the current be strong, the vessels diminish in size, so that the smaller ones disappear, and the temperature falls". But then it must be remembered that the visibility of the smaller vessels and the rise of temperature are due to total paralysis of the vessels by section of the nerve, and that the galvanism has to close up the vessels dilated by paralysis, and to cause a fall of abnormal temperature; also, the vessels have to be seen through the skin of the ear, and none of them are large, so that the continued visibility of these small vessels covered by skin and hair, when under tetanising stimulation of the nerve, shows how limited is the power of the vaso-motor nerves over the arteries. Now, these are but moderate results as compared with ligature of the cervical arteries; and if stimulating the arteries by tetanising the vaso-motor nerves only causes them to contract "to or beyond" their normal calibre, and if the muscular powers of the arteries are shown to act independently of the vaso-motor nerves, it is fair to ask what justification there is in this for supposing that any action of vaso-motor centres will equal Kussmaul and Tenner's proceedings, so that the arteries of the brain may be virtually ligatured by the cervical sympathetic. When the physiologist proceeds from his point of view to apply any conclusions from his experiments to practical medicine, he is very moderate: he has seen the extent of contraction and dilatation obtainable by cutting and galvanising the sympathetic, and he does not suppose that the sympathetic can ligature the cervical arteries. What he says is as follows: "The numerous pathological phenomena classed under sympathetic action, such as the affection of one eye by disease of the other, are probably at the bottom the results of reflex motor action"; and again: "The afferent impulses may arise anywhere, as, for instance, in the brain. Thus, an emotion originating in the cerebrum may either inhibit or exalt that portion of the vaso-motor centre which governs the vascular area of the head and neck, and thus call forth"—what? "either blushing or pallor". This is very moderate—it corresponds with the degree of change in the calibre of the arteries observed in experiment. But it gives no adequate grounds for any supposition of an action of the vaso-motor centres that would equal Kussmaul and Tenner's ligatures in depriving the brain of blood, so as to cause fits of epilepsy, or even fits of unconsciousness. Attempts to produce such fits by galvanising the cervical sympathetic ended in failure. The physiologist only claims for the action of the vaso-motor nerves such effects as blushing and pallor, and these would rather go with fits of laughter or crying. Even in the matter of blushing and pallor, we are not entitled to suppose

that the change of blood stream is altogether due to the vaso-motor nerves. It is true, of course, that bad news will make the face pale; but we must not forget that such causes influence the heart's beat, and thus a person suffering a mental shock has his circulation altered at its centre, and feels this effect universally, so that perhaps his stomach seems to turn, and he may have to go and eat his supper with what appetite he has. In fact, the only changes in the vessels of the face that can be certainly referred to the vaso-motor nerves are such changes as are more properly called flushing, as in the hectic of phthisis, or in the redness of the eye in neuralgia.

There is, of course, not the slightest doubt about the arteries possessing and using the power of contraction; but, in estimating the effects of their contractile actions, we have to note carefully the rapidity and the degree of their contraction, and the extent to which it is under control of the vaso-motor centres and nerves. It must, I think, be admitted that, so far as any evidence of sudden and entire closure of arteries, the arteries, if they possess such a power, are curiously sparing in the use of it. Indeed, I do not know a single instance in which the arteries put forth a power of sudden and total closure in parts exposed to view. The arteries appear to reserve all these quick and strong acts for exhibition, if I may so speak, in dark *stans* in such deep parts as are hidden within the cranium. But, if the arteries be capable of inducing sudden blanching by total contraction, why should the brain be the only part subject to such play? Why should not the arm, or leg, or ear, or nose, now and then show sudden paleness and redness from central vaso-motor play?

The kinds of instance which show the effect of contraction of the arteries are well known: thus the wax-like paleness of one or more fingers to which some people are subject; the drying of the mouth through depressing emotion, and its moistening through appetite; the flow of tears; the reddening of one ear "when people are talking of you at a distance". In all these cases, we have effects which are doubtless due to the power of the nerves over the arteries, and some which come on tolerably speedily. But, in all these and such instances, there is moderation in rate of accession, and also moderation in degree of the arterial change.

Let us now proceed to consider the onset of the epileptic convulsion, which it is customary to attribute to spasm of the cerebral arteries. I have already traced this theory through Schröder van der Kolk to the experiments of Kussmaul and Tenner, who produced epileptiform convulsions by ligature of the cervical vessels in rabbits, and who thereupon started a theory that spasm of the cerebral vessels would have the same effect. Schröder adopted a part only of this theory, supposing that spasms of the cerebral arteries produce the unconsciousness but not the convulsions of epilepsy; and this theory is adopted by our own authorities upon the subject. But, if we now refer to the published accounts of Kussmaul's experiments, we shall, I think, see reason in these to conclude that spasm of the arteries will not explain the onset of epilepsy. If we turn to Kussmaul's work, we find as follows. "General convulsions usually followed in from eight to eighteen seconds after complete withdrawal of arterial blood.....We found, in every instance, that the closing of all the four arteries was necessary to produce rapid convulsions. If but one carotid or one subclavian had remained pervious, we have never seen convulsions take place, even when the ligature has remained on the other three for several hours. The animals, however, have generally appeared enfeebled, and more or less paralysed."

It appears, then, that, when the arteries were closed absolutely as to three-quarters of their supply, no phenomena of epilepsy occurred, but only feebleness. We do not know what proportion of the remaining quarter of the arterial supply would have been sufficient to keep off the epileptic attack, since it was necessary to take this quarter away at one stroke by ligaturing the one remaining artery; but there were instances in which the fits did not occur, even when all the cervical arteries were ligatured, instances which Kussmaul explained by supposing some small artery to have reached the brain from an unusual source. Hence a very small remainder of arterial blood-supply prevents the onset of epileptic phenomena. But I think I have shown, from our best accredited works on physiology, that the most forcible stimulation of the vaso-motor nerves in mammals is far from causing complete closure of the corresponding vessels; and, although there are no numerical proportions given, yet I submit that the descriptions which I have quoted justify me in saying that it is at least very doubtful whether the arteries can be made to contract down to one-quarter of their natural calibre by any known mode of action of the vaso-motor nerves; whereas it requires a contraction beyond this degree to produce any part of an epileptic attack.

Again, it is not only on account of its insufficient degree that arterial spasm will not explain any epileptiform phenomena, but there is the

further lack of quickness in the act of arterial contraction. Let us take into consideration the slow rate of action of the arteries. When we realise the fact that even the partial closure which a vaso-motor nerve can cause in an artery requires from five to thirteen seconds for its production, we shall perceive that arterial contraction is too slow an affair to be credited with the production of the epileptic onset; for consider the frightful suddenness of the epileptic fit, and compare it with thirteen, or even with five, seconds. Let anyone count five seconds on his watch, and he will find it a considerable time. If a person, when taken with an epileptic attack, had thirteen seconds' warning, by changes in his brain through gradual loss of blood-supply, he would have time to do a great deal to protect himself. He could almost come in from the garden into the house, or at any rate cross his room to lie on a couch. Even if he had only five seconds, he could safely lie down upon the floor. But, instead of this, he falls downstairs, or into the water, or into the fire; a stroke of lightning is not more absolutely sudden. Surely it is not to be supposed that no feeling at all would arise in the brain during a gradual blanching of it, up to the degree which Kussmaul and Tenner showed to be required before the epileptic onset will appear.

Meantime, however, it remains true that, when a person is seized with epilepsy, the face becomes very pale, and so some interference with blood-supply evidently does occur. And, although I think it is our duty, in forming opinions on these grave subjects, to test the foundations and the reasonableness of our conclusions, yet I should not have spent so much time in showing the groundlessness of the theory of spasm of the cerebral vessels, if I could not offer facts of a kind to explain the paleness of the face, and perhaps the unconsciousness of the epileptic seizure.

But I will now relate some observations, which have convinced me that the cause of paleness of the face in the epileptic attack is stoppage of the heart's action. The first of these observations was as follows. Seven years ago, I was standing by a patient who was in bed with cardiac dropsy, in Philip Ward at Guy's Hospital, and I was feeling his pulse carefully; the beats were regular, and of moderate strength. I was looking towards the middle of the bed, and my attention was given to the pulse. I missed one, then another beat, and then found the pulse to have stopped so entirely, that it made me look up suddenly at the man to see what was the matter. His face had become very pale; his head was turned towards his left shoulder; and the lip and cheek were twitching; the spasms then affected his arms, and he passed into a severe epileptic convulsion. At the time when I first noticed the pulse to disappear, the man's arm was quite still. I tried to feel the pulse during the convulsion, but there was so much muscular movement, that I could not distinguish it.

I had never before chanced to have my fingers on the pulse at the moment of onset of an epileptic convulsion, nor have I ever done so since, until January 13th of the present year, when I was at No. 1 bed, Philip Ward, in which bed was a man who had come in with uræmic vomiting, and had been seized with convulsions. At the time of my visit, he had come out of the insensibility which followed each convulsive attack, and was in an excited state, staring about rather wildly, and endeavouring to raise himself in the bed, and calling loudly the word "Beer!". He was not a temperate person. He then lay quite still, and looked quietly about him, his arms lying outside the bed-clothes. I took his wrist and felt his pulse; it was forcible and regular. I was feeling it, when it quite disappeared from under my finger, many beats being missed. I looked at him; he had become very pale in the face; twitching set in, and he at once went into a very severe epileptiform convulsion. Mr. Lane, my house-physician, then told me that he had chanced to be feeling the pulse at the moment of onset of convulsions in the man during the morning of that day, and that the same thing had happened on two occasions in which he had his fingers on the pulse at the moment of the commencement of the attack.

Since then, Mr. Lane has made a similar observation upon a girl with eclamptic convulsions in Mary Ward.

So far, however, it might be said that the failure of the pulse was due to spasm of the arteries of the arms. Now, such a sudden universal arterial spasm would be an otherwise unprecedented thing, and my next observation shows that the supposition of it is not wanted. Three years ago, I was examining the heart of a young gentleman, who was asking my advice on account of attacks of epilepsy. He was standing, and I had my stethoscope over his heart, and was attending to the quality of the sounds. These sounds stopped suddenly and so completely, that, in great surprise, I moved to look at him, and saw him with exceedingly pale face evidently quite unconscious, his balance just lost, and he falling forwards. He went forthwith into severe epileptiform convulsions.

Dr. Hughlings Jackson has kindly informed me that he has observed

on several occasions the pulse to disappear during the paleness of the face in the onset of attacks of *petit mal*, and Dr. Hilton Fagge was good enough to send me the following notes of a case.

"John W., aged 23, consulted me for epistaxis. While I was listening to his heart, it suddenly stopped beating. His face turned very pale. He said, 'I am going to faint', and sat down in the chair, from which he had risen that I might examine him. There was, of course, no pulse at the wrist. Then his face flushed, and his pulse started again. I suppose the attack lasted half a minute. There was distinct twitching of the muscles of one or both hands. I could not observe very well, having to get water, and being alarmed at his condition. On recovering, he said that he often had 'fainting fits'; explaining this by adding, when he cut himself badly, more than ten years previously, he had had epileptic fits."*

A medical friend, whose observation I can well trust, and who has a daughter subject to epileptic seizures, with frequent attacks of "*petit mal*", writes to me that "her heart appears to stop in every attack".

Now, in inquiries amongst my colleagues and friends, these are all the cases with which I have been able to meet, in which the state of the circulation has been observed at the first moment of the epileptic onset. There are, of course, very numerous observations upon the state of the pulse when the patient has already fallen; the heart's beat has, however, recommenced, as I suppose to be necessary if life is to continue. I do not know how many beats can be entirely omitted without death resulting, but the number must be limited, because the circulation depends on tension in the arteries, and the whole arterial tension must be due to but few heartfuls of blood, so that the loss of few beats would take the tension quite away. I would, however, ask your notice of the fact that most observers who have felt the pulse just after a person has fallen in an epileptic fit, agree in describing the pulse as small, sometimes imperceptible. It is said, in descriptions of the first stage of the fit, to be sometimes unaltered; but I think this must refer to the later part of the first stage, during which the pulse recovers itself, but the insensibility continues. I have found the pulse present again when the clonic spasms have just commenced.

The observations which I have adduced, showing failure of the pulse in the first onset of epileptic attacks, tend to take away the significance of a very wonderful case which is quoted by Kussmaul from Pereira, as some evidence of spasmodic closure of the cerebral vessels. In this case, the pulse at one wrist was observed to disappear in the onset of an epileptic attack, and *post mortem* examination showed that the radial artery received its supply through the vertebral from the circle of Willis, a very remarkable anomaly indeed. It is inferred by Kussmaul that, in this person, the arteries of the brain went into spasm, and the spasm shut off also the blood from the arm which had the misfortune to derive its supply from so irritable a source. But I think it is reasonable to conclude that, in this very wonderful case, what really happened was a failure of the heart's action, as in my cases.

Thus, so far as I have been able to learn, the unconsciousness in an epileptic fit is of just the same nature as the unconsciousness in a fainting fit. It has happened to me—as, I presume, to most physicians—to be able to notice the first onset of a fainting fit. Time will not allow me to describe the several occurrences; but in one young candidate for service at a bank, who fainted under examination, the heart's beat ceased as I was listening, and the lad fainted. He was pale throughout the attack; and he retched as he was recovering, and told me that nothing of the kind had ever happened to him before. In the case of a young lady subject to "fainting fits", I observed that the heart stopped beating. She used to fall very suddenly, and slight twitching of the face appeared. Nothing more like epilepsy ever occurred. But such attacks are very like epilepsy; and I think that sudden faintings are always to be regarded with suspicion when they take place without sufficient cause of the nature of violent emotion or shock from some horrifying occurrence. Practically, the presence of adequate cause of this kind is the readiest distinguishing character of what one might call normal faintness, as contrasted with epileptiform fainting. Other distinctions lie in the more sudden onset, and the earlier return of colour in epileptiform fainting. These sudden fits of unconsciousness, occurring without shock to explain them, appear to bear the same relation to the *petit mal* of epilepsy as eclamptic convulsions bear to the *haut mal*. I mean that, in either of the two parallels, the character of the attacks is the same; and the difference is due to the cause of the attack, which in fainting, as in eclampsia, is temporary, but in *petit mal*, as in epilepsy, tends to produce a recurrence of the attack.

I do not know how the cessation of the heart's action is caused; but

* At the conclusion of the lecture, Dr. Southey informed me that, in the only instance in which he had his finger on the pulse at the beginning of an epileptic convulsion, he observed the pulse to disappear during the onset of the attack.

I suppose it is through the pneumogastric nerve. It is quite certain that this nerve is capable of inhibiting, as it is called, the heart's action so as to stop it for many beats; so that the power of the pneumogastric to cause the change is beyond question; whereas I have shown that there is great reason to doubt that spasm of arteries will stop the circulation in the brain so as to produce insensibility. Another fact, that points to the pneumogastric as the probable cause of the change, is, that the gastric branches of this nerve are evidently involved in minor degrees of the condition of fainting; as in the former experience of many of us, when the witnessing of sanguinary operations, before we were accustomed to such scenes, caused nausea and sinking sensations more or less embarrassing.

Again, Mr. Thomas Bird, whose experience in the administration of anæsthetics is very large, and whose care as an observer is well known, tells me that, during chloroform-exhibition, sudden pallor is accompanied by retardation of the pulse, and followed by sickness or retching.

Such lesser degrees of faintness evidently refer to a change of action in the pneumogastric system of nerves. Now it is well known that one of the most common of the immediate causes of sudden fainting fits is the sight of blood, or rather of bleeding; and this sudden fainting, which in those who fall at the sight of a cut finger is abnormal, is an extreme example of a depressing action of the pneumogastric nerve, which is, in a lesser degree, so far natural that a large proportion of young men suffer a feeling like sea-sickness when first they witness a protracted sanguinary cutting operation. Hence the blanching and insensibility of the epileptic onset may be reasonably regarded as an effect of pneumogastric inhibition, the initial step being thus an act of the nervous ganglia.

And, indeed, it must be clearly seen that any explanation of the epileptic seizure thus comes ultimately back, not to the hydraulic mechanism of the circulation, but to the seats of origin of the nervous power, in whose endowments lie the mechanisms of epileptic convulsions, and in whose action the first impulse must originate when the fit is brought about. But, in the cases I have described, the next step following this incomprehensible impulse is a very comprehensible stoppage of the heart, with the result of a very comprehensible paleness and insensibility. It is said that paleness does not always occur in the onset of epilepsy. I have observed a livid appearance in the onset of the fit in a very red-faced person. The natural red appearance is, I believe, never retained. There is now under my care an epileptic girl who also has an extreme degree of inveterate psoriasis, through which her face is reddened intensely with inflammation; yet in the onset of her fits this girl's face becomes very pale.

Starting, then, with a pneumogastric impulse of unknown origin, we can, through the cessation of the heart's beat, abundantly satisfy any desire for a hydraulic explanation of unconsciousness; but we must note that the entirely unknown source of the pneumogastric impulse stands behind our explanation, throwing on it the doubt whether the same unknown impulse which discharges the pneumogastric may not play among the centres where consciousness is seated, and itself involve a loss of consciousness. But, admitting this necessary doubt, we are able to say that the stoppage of the heart's action is itself sufficient to produce the unconsciousness and pallor; so that it is not necessary for us to seek any other explanation of them. And even in the minor degrees of epilepsy, in which there is only a momentary hesitation, and as if a blank were suddenly introduced in the continuity of the person's course of action, the stoppage of the heart's action will explain it, especially as a sense of giddiness accompanies the smallest degrees of such attacks, as in epileptic vertigo; for giddiness is, when certainly referable to any change of circulation in the brain, always referable to a diminution of force of the circulation. It very commonly occurs on sudden removal of stress or tension from the venous circulation in the brain, as instanced in the giddiness which is felt on going out of chambers of compressed air, or on rising suddenly from a stooping posture. Very violent giddiness has other and different causes. The extreme giddiness one sometimes meets with, as when a gentleman came to me and begged relief from a giddy attack so violent that, when seized, he had to call to his daughters to hold him on the floor, as he seemed to be falling upon the ceiling. This man's attacks ceased on the occurrence of a paroxysm of gout. Menière's disease offers a frequently applicable explanation of severe giddiness. But there is ample evidence to show that slight giddiness occurs through slight blanching of the brain; so that when, in an epileptic subject, the face grows suddenly pale, and at the same moment a sense of giddiness is felt, it is reasonable to ascribe all this to failure of the heart, if we have reason to know that the heart fails; and I have endeavoured to show reason to think so.

But a fully developed epileptic attack offers us at least three quite distinct occurrences. The first is the initial loss of consciousness; the

second, the outbreak of convulsions; and the third, the state of stupor which succeeds to these. It is a natural and fair question to ask, Will the hydraulic explanation, which suffices for the loss of consciousness, go further, and explain the other two terms of the series of events that compose an epileptic fit? Now, either of the three portions of the complete attack may occur almost alone, or perhaps quite alone. In the so-called *petit mal*, a partial faint and momentary loss of consciousness is all that happens. In some cases of convulsions, a partial degree of epileptiform muscular jactitation is similarly present; and Dr. Hughlings Jackson's name will be always honourably identified with our best thought and knowledge upon this most interesting phase of epilepsy.

In other cases, the stage of stupor may come on with little evidence of faint or of convulsions; and I think Trousseau has claim to be honourably held in recollection, on account of the clearness with which he showed the truly epileptic nature of attacks of this kind, which were—and, indeed, by some still are—obscured under the designation of apoplecticiform cerebral congestion.

This separate occurrence of the three distinct parts of the epileptic attacks proves that there is no direct dependence the one upon the other; and yet, they are so constantly found united, in serial order, in the complete epileptic fit, that it is natural to look for some causative connection between them; and to find some way of showing how, upon the profound fainting attack, convulsions would ensue; and how, upon convulsions, stupor would follow. If this were positively desirable, it would not be difficult to bring in Kussmaul's convulsions from anæmia, to explain the onset of the spasmodic movements: for if, indeed, it be a law of the vertebrate organism, that a discharge in convulsion occurs when bloodlessness is on the point of becoming fatal to the life of the brain, then a prolonged stoppage of the heart would fall under such law, and convulsion be the result. Nay, we may further here observe, that, in a certain degree, there is in such a law that kind of adaptation to purpose, which it is so great a satisfaction to perceive, seeing that, when the heart's beat stops, the arteries empty themselves, and then tension is done away; and it requires at least six or seven heart-fulls of blood to restore arterial tension. Meantime, with the removal of arterial tension, the *vis à tergo* on the whole circulation is lost, and the blood is no longer pushed round in a continuous stream, so that it would therefore tend to stop in the veins, and in the abdominal veins alone there is room for the whole of the blood. Therefore, with all the blood in the veins, and with no *vis à tergo* to act on them, where should the heart get its six or seven charges of blood to start arterial tension once more? But the veins of the limbs have valves that prevent reflux of the blood; and convulsions compress the veins, and drive to the heart the necessary amount to keep the arteries full.

And, indeed, it is quite probable that this is the purpose or use of the convulsions. Neither does it gainsay such a use that the convulsions become extreme, and pass beyond the service for which they are adapted. For, take the parallel case of the plainly conservative spasm of a cough, whose object is beyond doubt to expel undesirable contents from the windpipe, do we not find cough sometimes so vigorously acting in excess of its duties that we are at our wits' end to find some means of keeping this otherwise beneficent contrivance within due bounds.

Likewise, also, if we ask why stupor should succeed to convulsions, we may think this also a natural consequence of the convulsions; and we may also perceive that it favours recovery. For convulsion is no mere exertion, as in healthy exercise; but it is the exertion of otherwise dying nervous centres, called out, not by the wholesome influence of sufficient or redundant supply, but brought to pass by desperate starvation; and, therefore, in the matter of exhaustion, not to be put on a par with normal fatigue. Then, what more likely than extreme exhaustion? and what better than a short deep sleep, in which the senses are, if I may so speak, closed for repairs? An insensibility which is thus negative, and whose service is plainly to allow time for the centres to recover themselves, is not of itself to be dreaded nor thought of as a terrible thing. It need not to be set down to any overpowering positive cause, on the baseless old-fashioned theory of congestion of the brain. But here, too, in a more dangerous way, we may find ourselves troubled with an excess of what is designed for good; and as such may need checking, and convulsions prove extreme, so stupor, negative of power as it is, may be protracted; and the sleep, which was intended to refresh, may not restore excitability, but become longer and deeper until the person dies in a state of coma. And the normal stupor of the epileptic attack becomes thus extreme and fatal under conditions that sufficiently explain the occurrence. For such fatal coma never happens in the epilepsy of young or vigorous subjects, when the brain is well nourished and capable of quick recovery. It is in old age that it occurs, or at the close of a long course of cerebral exhaustion, especially when an impure state of the blood adds another cause of

stupor, as in cases of protracted jaundice, or diabetes, or chronic renal disease, which not unfrequently terminate by a sudden epileptiform coma; or in old alcoholism in unstable brains. In these cases, essentially epileptic attacks occur, in which there is little or no convulsion, and the person appears seized with sudden coma. But, as a proof of the epileptic nature of such attacks, it is remarkable that, in the description of them under the erroneous title of apoplectiform cerebral congestion, it is stated that the pulse disappears at the wrist in the onset. Now, after the cases which I have given, I consider this to be evidence of the epileptic nature of these attacks. For clinical evidence of the connection of such attacks, in individual cases, with the history of epilepsy, I have already referred to Trousseau's lecture on the subject. But, if I had time, I could give many cases of the kind from my own experience.

ABSTRACT OF LECTURES

ON THE

ANATOMY, PHYSIOLOGY, AND ZOOLOGY OF THE CETACEA.

Delivered at the Royal College of Surgeons of England.

By W. H. FLOWER, F.R.S., LL.D.,
Hunterian Professor of Comparative Anatomy.

LECTURE II.

THE cetacea are divided into two distinct groups, according as they possess whalebone or teeth. To those which possess whalebone, the name *Mystacoceti* is given, and those which have teeth are called *Odontoceti*. The classification of whales in this manner may seem to be an arbitrary one, but in reality it is not, as differences in the anatomical characters—both of their skeleton and of their soft parts—separate them into two groups exactly corresponding to the above.

As a type of the whalebone whales, the lesser rorqual (*Balenoptera rostrata*) may be taken. It was first described by Fabricius, who called it *Balena rostrata*. Hunter identified the one he received as being of the same species, and gave it the English name of piked whale. The animal caught and preserved, in 1689, at Bremen is the earliest one of the species known. Since Hunter's time, several have been preserved and described. A very excellent specimen was procured for the museum by Mr. J. H. Gurney, M.P. This whale was stranded at Cromer, in 1864, and carefully buried till the soft parts were completely decomposed; the skeleton was exhumed under Professor Flower's superintendence, its exact length obtained, and the curves of the spine and other particulars noted. After being brought to London, it was mounted, and now occupies a place in the western museum of the College. The skeleton is very complete, all the small bones having been secured, as well as the large ones; and the whalebone is also preserved *in situ* on one side of the jaw.

The *Balenoptera rostrata*, or lesser rorqual, inhabits the northern parts of the Atlantic Ocean, the fiords of Norway, the English and French coasts; and in two instances it has been found in the Mediterranean. It occurs on the American coasts; and an animal very similar, if not identically the same, has been found on the coast of New Zealand. Its food consists of fish about the size of the herring, small cod, and octopods or other invertebrata; it does not eat the very minute food which the Greenland whale lives upon. The greatest length to which the species attains is probably about thirty feet; the skeleton in the museum, which is that of a nearly adult animal, measures twenty-five feet, and, when alive might perhaps have been slightly longer. The body is long and narrow; the mouth wide and gaping, to use Hunter's simile, "like that of a goat-sucker"; the pectoral fin is small; and a dorsal fin of small size is present. In colour, it differs from that of the other *Balenoptera*, being black on the back, while the belly is white; the pectoral fin has a characteristic white band running across it, other parts being black. The extremity of the tail consists of two lobes placed horizontally. The upper surface of the one is concave, while that of the other is convex; on the under surface, the reverse is the case, the lobes being alternately convex and concave. Thus, the tail-lobes correspond to the blades of a screw-steamer, and probably propel the animal through the water in

the same manner as the screw does a ship. There are no external ears or projections on the surface of the body, to obstruct the passage of the animal through the water; only a small slit, about two millimetres in length, situated on each side of the head, represents the external opening of the aural passage. There are two nostrils, situated longitudinally on the top of the head. The double nostril always present in the whalebone whales is an important distinction between them and the toothed whale, which possesses only one. The soft parts superficial to the muscles are arranged in layers: the first consists of a thick cuticle, containing pigment-cells; under which is the true skin, with its long papillæ; the third layer is formed by the blubber—a fibrous meshwork of connective tissue the interstices of which are filled with oil-globules; under this layer are the muscles. The skin of the throat and breast is thrown into curious folds, between which are furrows running in a longitudinal direction from the chin to where the chest and belly join, giving the anterior part of the under surface of the animal a fluted appearance. The furrows number about sixty on each side, and are usually quite distinct, though sometimes they join. The folds consist of skin, with a layer of blubber underneath. The object of this arrangement is, to allow considerable distension of the floor of the mouth. This is necessary, to enable the animal to obtain its food, which it does, not by seizing the fish on which it lives, singly, but by swimming along with its mouth open, and taking in large quantities of water, which it sifts of all fish contained, and drives out between the plates of whalebone by contracting the floor of the mouth. The barnacles and parasites of various kinds which frequently attach themselves to the anterior limbs, tail, and other external parts of the body of most whales, do not seem to attack this species, though it is often infested by internal ones. In the foetus of this animal, Eschricht has shown that a row of bristles is situated along the lips and on the chin, but never retained till the time of birth. The skull is very curiously modified, though not so much so as that of the Greenland whale. The enormous size of the mouth, as compared to the size of the cranium, is very striking. The foramen magnum is situated at the back of the head, and is directed backwards and upwards; the basisphenoid and presphenoid are hollowed out; the ethmoid is greatly prolonged; underneath it is the vomer, which is trough-like and very long. The parietals are situated beyond the frontals, but cannot be seen externally, on account of the upward and forward prolongation of the occipital bone, which covers not only the parietals but also the greater portion of the frontal. The nasal passages, instead of being horizontal, as in man, are directed upwards. Not only is the skull greatly elongated, but, to give breadth to the mouth, and so permit the animal to cover a larger area when feeding, it is drawn out laterally by lateral elongation of the frontal bone, and of the under surface of the temporal bone where the lower jaw articulates. The position of the lacrymal bone is also much modified. On the under surface of the maxillary bones may be seen several grooves; these contain the blood-vessels going to nourish the whalebone. Two round bones project from the under surface of the temporal bones; these are the tympanic bones, which are situated one on each side. They are loosely connected to the portion of the temporal bone which contains the organ of hearing, and are easily broken away; being very hard and dense, they exist long after other parts of the skeleton have disappeared. From these circumstances, they are frequently found in the fossil state in beds such as the Red Crag at Ipswich, and may often be picked up on the seashore. They correspond to the tympanic bullæ of the carnivora, but are isolated to a much greater extent from the skull than in any other animal. The right tympanic bone may be distinguished very easily from the left, if held with the open portion upwards and inwards; the thin end with the notch for the Eustachian tube anteriorly, and the thick end posteriorly. The notch is always directed to the mesial line, and is anterior; while, to the outer and upper surface, is attached the periotic, which arches across the tympanic opening. The openings between the tympanic and periotic bones are covered over by the tympanic membrane, which is of irregular shape. The mandible consists of two rami, which do not touch one another in front, but are united by fibrous tissue. Each ramus is arched or bow-shaped, the convex surface being the external one. The mandibulo-cranial articulation is peculiar; there is no synovial membrane present, but the mandible is attached to the skull by dense fibro-cartilage, permitting a considerable degree of movement. The power of the jaw is, on account of the looseness of its attachment, very slight, the animal being able to do little more than to approximate it to the maxillæ. The hyoid bone is large and flat, and consists of a body, two thyro-hyoids, and two stylohyals.

Mr. ARTHUR FLINT, L.R.C.P., of Westgate-on-Sea, has been elected a member of the Board of Guardians and Sanitary Committee of the Isle of Thanet Union.