A lecture on the pathology and treatment of epilepsy / by George Johnson.

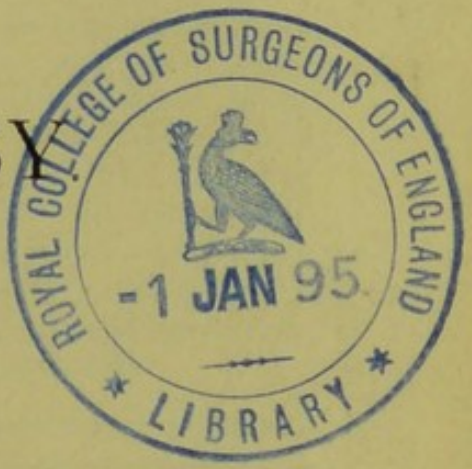
A LECTURE

ON THE

PATHOLOGY AND TREATMENT

OF

EPILEPSY



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THE PATHOLOGY AND TREATMENT OF EPILEPSY.

Epilepsy not the Result of Congestion, but of Sudden and Extreme Anæmia of the Brain—Experiments of Kussmaul and Tenner—Convulsions from Sudden Arrest of the Pulmonary Circulation by Air or Salts of Soda in Veins, by Embolism, by Acute Apnœa—Evidence as to Cerebral Anæmia resulting from Arterial Spasm—Two Classes of Cases, Nervous and Toxæmic—Epilepsy is Cerebral Collapse, Cholera Collapse is Pulmonary Epilepsy—Partial Epilepsy a Result of Spasm in limited Arterial Regions—Comparison of Epilepsy and Syncope—Rational Principles of Treatment in the Two Classes of Epileptic Cases—The Action of Chloroform and of Bromide of Potassium.

I PURPOSE upon the present occasion to attempt an answer to the following question—To what extent do the phenomena of an epileptic fit admit of explanation? In a fully developed epileptic fit, there are two chief phenomena to be explained: these are loss of consciousness and convulsions. The loss of consciousness, until within a very recent period, has generally been supposed to be a result of congestion of the nervous centres, and especially of the cerebrum. But this explanation is inconsistent with the fact, that the epileptic loss of consciousness comes on in a moment at the very commencement of the attack, when there is no evidence of congestion, and when the face is usually pale from anæmia. The congestion follows the loss of consciousness; and the explanation of its occurrence appears to be this. The convulsion implicates the respiratory muscles; the blood, therefore, being imperfectly aërated, is impeded in its passage through the lungs; it consequently accumulates in the right side of the heart and in the veins. The congestion is a secondary venous congestion; and, at the time when this congestion has reached its greatest height, there is often a commencing return of consciousness. Obviously, then, this congestion is not the cause of epileptic loss of consciousness. This retrograde venous engorgement is the cause of the ecchymoses beneath the skin and the conjunctiva, which often occur during a fit, and of the hæmorrhage into the substance

or upon the surface of the brain, which happily is a much less frequent accident.

A number of facts point to the conclusion, *that both the loss of consciousness and the convulsions of epilepsy are the result of sudden and extreme anæmia of the brain.*

In man, and in most, if not in all, warm-blooded animals, a rapid and very copious hæmorrhage usually causes convulsions. Kussmaul and Tenner state (*On Epileptic Convulsions from Hæmorrhage*, New Sydenham Society, 1859) that, in numerous cases of dogs, cats, and rabbits, they observed, without a single exception, violent and general convulsions preceding death from loss of blood. In order to produce this result, the hæmorrhage must be rapid. If it occur slowly, so that the vital powers are gradually consumed, death then occurs with swooning, drowsiness, and delirium, without convulsions.

The same observers found that an interruption in the supply of blood to the head of a rabbit, by ligature or compression of the arteries, produces epileptic fits as surely as hæmorrhage does. In about one hundred rabbits, they ligatured or compressed the carotids and subclavians, from which, be it remembered, the vertebrals proceed, and in every instance except that of one very old, lean, and feeble rabbit, convulsions occurred. In order to produce convulsions, it was necessary to close all the four arteries which supply the brain. If but one carotid or one subclavian remained pervious, the animal was enfeebled and more or less paralysed, but not convulsed. And, again, if, during the height of a convulsion, the ligature is removed from one carotid, the convulsions generally cease immediately, and there is a sudden change from the most frightful spasm to complete relaxation of the muscles. The description of the convulsions thus artificially produced in these animals, shows that they were essentially the same as epileptic convulsions in the human subject. There was the dilated pupil, the tonic spasm, quickly succeeded by clonic convulsion, so violent as to throw the animal forcibly forwards to a distance of one or two feet, and sometimes even over the shoulders of the experimenter. These experiments suffice to show the fallacy of the explanation which Dr. Brown-Séquard and others have given of the clonic convulsions in man. It has been supposed that the clonic convulsions are a consequence of the circulation of black blood which results from the tonic spasm of the respiratory muscles. Now it is manifest that, in these animals with ligatured arteries, no black blood could reach their brain. In them, therefore, the clonic convulsion, as well as the preceding tonic spasm, must be due to want of blood, and not to the altered quality of blood in the brain.

These experiments obviously cannot be repeated on the human subject; but Drs. Kussmaul and Tenner describe the effects of compressing the carotids in six men. In all, the face turned pale; the pupils first contracted and then dilated; the respiration became slow, deep, and sighing; then there was giddiness, staggering, and unconsciousness, and the patients would have fallen had they not been supported. "In two subjects, of weak intellect and moderately anæmic, in whom, notwithstanding the above symptoms, the compression was continued, a choking sensation, attended by vomiting and general convulsions, came on, which, however, did not attain an aggravated form; for, on withholding the compression, they disappeared in a few seconds." (*Op. cit.*, p. 28.)

Compressing the carotids does not, of course, entirely cut off the supply of arterial blood to the brain; but these experiments render it probable that sudden occlusion of all the arteries of the head will as certainly excite epileptic convulsions in man as in the lower animals.

There is a class of cases in which a sudden arrest of the blood in its passage through the lungs causes convulsions and speedy death. I mean cases in which the circulation is arrested by the admission of atmospheric air into the veins; cases of embolism of the pulmonary artery; again, cases in which the flow of blood through the lungs is stopped by the injection of certain salts into the veins; and, lastly, cases of acute apnœa. When animals are killed by blowing air into the veins, the breathing becomes hurried, the animal falls down, and usually dies in convulsions; the contents of the bladder and rectum being frequently expelled at the time of death. Dr. John Reid states that, "in a very few cases only, is death from this cause not preceded by convulsions." (*Physiological, Anatomical, and Pathological Researches.*) The immediate cause of death in these cases is the arrest of the frothy mixture of air and blood in its passage through the minute pulmonary arteries (the air rarely reaches the left side of the heart); and, as a result of this arrest, there is, of course, anæmia of the brain and of every other organ supplied by the systemic arteries.

In man, it appears that death from the admission of atmospheric air into the veins has been less frequently preceded by convulsions. Probably the chief reason of the less frequent occurrence of convulsions in the human subject is, that the amount of air accidentally admitted is less, and death, consequently, is less rapid than when air is forcibly driven into the veins of an animal. It would probably be found, on a careful inquiry, that the occurrence of convulsions in these cases depends upon the circulation being suddenly and completely arrested.

Convulsions are mentioned in only five out of fifteen cases collected

by Amussat ; but, Dr. Reid remarks, "several of the cases, as we might have expected, are very imperfectly reported; for it is not to be supposed that the surgeon or his assistants should possess the coolness and time to watch narrowly the phenomena, when their minds were agitated by the threatened sudden dissolution of their patient, and their attention distracted by anxious attempts to save him." For the same reason, the record of cases of pulmonary embolism is very imperfect ; but, in some instances, it has been observed that death was preceded by violent convulsions ; and Virchow noted, amongst the results of artificial embolism of the pulmonary artery in animals, convulsions and dilatation of the pupil. (*Des Emboles Pulmonaires.* Par B. Ball. P. 129.)

Again, Blake found that an injection of a solution of soda or its salts into the veins of a dog, destroys life by arresting the flow of blood through the lungs. The left side of the heart is found empty, and the right distended. Death occurs in about forty-five seconds, and is preceded by violent opisthotonos. (*Edinburgh Medical and Surgical Journal*, vol. liv, p. 343.)

Blake attributes the nervous symptoms to the venous pressure on the brain ; but they may, with much more reason, be attributed to the arrest of the arterial supply to the brain. When, from any cause, the blood is arrested in its passage through the lungs, it is obvious that distension of the systemic veins must have, as its necessary correlative, comparative emptiness of the systemic arteries ; the one will be an index and a measure of the other.

Lastly, we have the convulsions which occur in almost every case of acute apnœa or sudden suffocation. It is generally supposed that the convulsions of apnœa are excited by the circulation of black blood through the brain ; but they are more probably due to the rapid and extreme anæmia of the brain consequent upon the impeded transit of blood through the lungs. When the air is excluded from the lungs, the circulation is rapidly arrested by the contraction of the minute pulmonary arteries. That this is the true explanation of the convulsions of apnœa, is rendered highly probable by an observation of Kussmaul and Tenner (*op. cit.*, p. 75), to the effect that "the approach of convulsions in strangulation can be accelerated if the arteries are simultaneously compressed." It is obvious that, if the presence of black blood in the brain were the cause of the convulsions, their approach would be retarded, and not accelerated, by compression of the arteries which supply the brain. The facts are consistent only with the theory that the immediate cause of the convulsions in cases of suffocation is a rapidly increasing cerebral anæmia, resulting from the arrest of the pulmonary

circulation. Black blood, in so far as it is deficient in oxygen, is equivalent to no blood. Probably it is rather by its negative quality of being unoxygenised, than by any positively noxious properties, that it is unsuited to maintain the functions of the brain. It is probable, too, that the minute cerebral arteries resist the passage of black blood, and so increase the anæmia of the brain. If the circulation of dark blood through the cerebral vessels would excite convulsions, we should expect to find this symptom of common occurrence in cases of emphysema with bronchitis.

I have now referred to instances of epileptiform convulsions occurring under a considerable variety of circumstances, but all agreeing in this one condition, namely, that the convulsions are associated with a defective supply of arterial blood to the brain. Let us now proceed to inquire whether the phenomena of epileptic convulsions, as they ordinarily occur in the human subject, are consistent with the theory of anæmia. It is a matter of general observation that, at the very commencement of an epileptic fit, the face is pallid. There is anæmia of the superficial vessels; and with this there is probably associated anæmia of the intracranial vessels which supply the brain itself. The pallor is in most cases soon succeeded by lividity, owing to venous engorgement consequent upon the impeded respiration and pulmonary circulation. It is very remarkable that, while the face is pallid, the heart and the carotids are beating strongly. It is probable, therefore, that there exists some impediment to the flow of blood through the minute branches of the arteries. To explain this impediment, Kussmaul and Tenner suggest that the minute arteries, both the superficial and the intracranial branches, contract so as to bar the passage of blood. Hence arise the pallor of the face and the epileptic convulsion. In some cases, it is said that the face is more or less livid at the very commencement of the fit. The probable explanation of this is, that the respiratory muscles are convulsed, and there is a consequent venous turgescence before the spasm affects the facial arteries. The spasm of the *facial* arteries, though usually present, is obviously not the cause of the fit. The early implication of the respiratory muscles is clearly shown in those cases in which the "epileptic cry"—a result of spasm of the glottis—is the first indication of the fit.

Kussmaul and Tenner endeavoured to support the theory of arterial spasm by experiment, and to some extent they succeeded. In each of three white rabbits they ligatured the two subclavians and one carotid; the cervical sympathetic was then exposed and galvanised, with a view to excite contraction of the arteries by the stimulus conveyed through

the vaso-motor nerves. In two animals, no effect was produced; but in the third rabbit the background of the eye became completely pale; the pupil dilated, so that the iris could scarcely be seen; the neck was drawn back; and violent convulsions occurred. The electrodes being removed, the spasms ceased, the pupil contracted, and the background of the eye became red; but the animal continued in a swooning condition. After some minutes, electricity applied to the sympathetic nerve produced the same effects as at first. A third attempt did not succeed.

The authors suggest that these experiments deserve repetition, with the view of rendering certain what at present is probable; namely, "that epileptic convulsions can be brought about by contraction of the blood-vessels induced by the vaso-motor nerves."

According to this theory, then, epilepsy is a result of sudden anæmia of the brain; and this anæmia, when not caused by hæmorrhage or by a mechanical impediment to the circulation outside the cranium, is due to an extreme contraction of the minute cerebral arteries. With reference to this theory of arterial contraction, all cases of epilepsy may be arranged in two distinct classes: 1, cases in which the arterial contraction is the result of a purely nervous or reflex influence; 2, cases in which the arterial spasm is a result of blood-poisoning.

In the first class are included all cases of epilepsy which are due to emotional influence, sudden terror, or anger, or long continued anxiety and sorrow, perpetuated and intensified often by nocturnal dreams and frequently recurring nightmare; also cases associated with those disordered states of the nervous system which are the result of vicious sensual habits; cases, too, in which the disease is hereditary. This class also includes cases of epileptic convulsions from the irritation of the gums during dentition; of the kidney, or the ureter, or the gall-duct, by a calculus; and of the alimentary canal by worms. Again, the cases in which convulsions result from tumours or other organic disease of the brain are included under the head of epilepsy from a reflex influence. The structural change in the brain is not the proximate cause of the epilepsy; it excites the epileptic convulsions through a secondary reflex influence upon the blood-vessels. Dr. Brown-Séquard's guinea-pigs, rendered epileptic by injury to the spinal cord, are also included in this class of cases. The injury to the cord probably acts by increasing the reflex excitability of the nervous centres, so that a trifling external irritation suffices to excite a fit.

In the class of toxæmic epilepsy are included those cases in which noxious materials in the blood are the immediate exciting cause of the arterial spasm—uræmic convulsions, and all cases of convulsions from

retained excreta; convulsions resulting from the admission into the circulating blood of unwholesome and undigested food; the convulsions which result from alcoholism; the convulsions which sometimes occur during the initiatory stage of certain of the acute febrile exanthemata, more especially small-pox; the convulsions which are occasionally associated with pyæmic infection; and the convulsions resulting from a poisonous dose of prussic acid. In each and all of these cases, it is probable that the immediate cause of the convulsion is anæmia of the brain, resulting from contraction of the cerebral arteries; the arterial spasm being excited by the presence of morbid blood in the vessels.

In the hypertrophy of the muscular walls of the arteries of the pia mater, which we have recently observed in cases of Bright's disease, we have evidence of a continual resistance to the passage of the deteriorated blood through these vessels. This resistance probably explains some of the cerebral symptoms of Bright's disease; but a convulsive seizure must be due to a sudden temporary increase of arterial contraction—this sudden contraction differing as much from the continuous tonic contraction of the vessels, as the cardiac spasm of angina pectoris differs from the regular strong contractions of a hypertrophied ventricle. With reference to the action of prussic acid upon the blood-vessels, it is noteworthy that Blake, having killed a dog by injecting prussic acid into the jugular vein, observed that, after the animal had ceased to struggle, the dynamometer in the femoral artery still indicated a considerable increase of pressure. This fact is explicable only on the supposition that the prussic acid excited unusual contractions in the minute systemic arteries; and this contraction of the cerebral arteries would account for the convulsions in cases of prussic-acid poisoning. Again, the symptoms which result from an over-medicinal dose of prussic acid are such as might be occasioned by a less degree of obstruction to the cerebral circulation. These symptoms, as described by Pereira, are the following: "disordered and laborious respiration (sometimes quick, at others slow and deep), pain in the head, giddiness, obscured vision, and sleepiness. In some instances, faintness is experienced." These symptoms are remarkably like those of the epileptic vertigo, or *petit mal,* as it is called; and, like them, they are probably due to a temporary and partial interruption of the cerebral circulation by arterial spasm. The poison, being very volatile, is quickly exhaled by the lungs; and the symptoms soon cease. In accordance, then, with this theory of arterial contraction, epilepsy might be designated *cerebral collapse;* and, on the other hand, the arrest of the circulation by the contraction of the pulmonary arteries in the

collapse of cholera may be looked upon as a form of *pulmonary epilepsy*. Without doubt, the true key to the pathology of both these awful diseases, epilepsy and cholera, is to be found in this doctrine of arterial spasm.

The two classes of epileptic cases—the purely nervous and the toxæmic—have their analogues in the two varieties of laryngeal spasm. Spasm of the larynx may result from irritation of the brain, or of the gums, or of the alimentary canal; in short, it may be a purely nervous reflex *laryngismus stridulus*, which is closely allied to epilepsy. On the other hand, laryngeal spasm may be excited by a crumb of bread or a grain of salt, or other irritant, within the larynx; and this is analogous to the arterial spasm which is excited by toxæmia, and which may result in an epileptic fit.

There are cases of convulsions in which the purely nervous and the toxæmic elements are combined in varying degrees—cases of so-called idiopathic epilepsy, in which the immediate exciting cause of a fit is some blood contamination consequent upon disordered digestion or retained excreta; and again, cases of toxæmia in which a paroxysm of convulsion is provoked by some emotional or other nervous excitement. In many cases of puerperal convulsion there is a combination of the toxæmic and the purely nervous influences—a scanty secretion of albuminous urine and consequent uræmia, together with the exalted excitability of the whole nervous system which often accompanies the puerperal state, and which is highly intensified during the pains of parturition.

There are various forms of what may be called partial epilepsy: sudden and transient impairment of motor power, or irregular spasmodic movements limited to a particular set of muscles; various disordered sensations in limited portions of the skin; derangements of the special senses; sudden perversions of taste, or smell, or sight, or hearing; sudden impairment of speech; vertigo; confusion of thought; temporary delirium; and mental excitement. One or more of these symptoms may occur singly or variously combined in different cases, the onset and the departure being often equally sudden. In explanation of these phenomena, Dr. Hughlings Jackson has, with much ingenuity, suggested that they may result from a sudden temporary interruption of the blood-current through one or more branches of the cerebral arteries by spasm of their muscular walls; so that the brain-tissue within a circumscribed "arterial region", having its nutritive supply arrested or limited, would suffer a suspension or impairment of its proper functions. This appears to be a very probable explanation of the curious phenomena in question.

It must be borne in mind, that the brain is not one organ, having a simple function, like a lung or a kidney; but that it is a congeries of complex organs, having very diverse functions. It seems probable that the physiological cooperation of all these cerebral organs may require that the blood-supply to the various regions of the brain should be specially regulated by certain branches of the arterial tree, under the guidance of the vaso-motor nerves; and this regulating power residing in the arteries probably renders them liable to disorderly action under the disturbing influences of disease.

Dr. Hughlings Jackson, in his very interesting and suggestive papers on nervous diseases, has repeatedly directed attention to the fact that hemiplegia, hemispasm, and hemichorea must have the same anatomical seat in the corpus striatum or thereabout; but that the structural condition of the nervous tissue concerned must be different in the different classes of cases. In cases of paralysis, the nervous tissue is so far disorganised, either temporarily or permanently, that its functions are entirely suspended; while, in cases of irregular movement, the disordered and perverted functions are associated with a less degree of structural change, or with none at all.

During the disorderly convulsive movements of epilepsy nerve force is converted into motion, and this is followed by a state of exhaustion; so that for a time the reflex excitability of the nervous centres is lessened. When fits recur at short intervals they are usually less violent; on the other hand, a very violent paroxysm often follows an unusually prolonged immunity from the attacks. It would appear that the nerve force, which is stored up during the intervals of the attacks, is discharged during the convulsive seizure. The convulsive movements are probably, in part, at least, a result of the suspension of the functions of the cerebral hemispheres, whereby the controlling influence of the will is cut off from those lower centres which more directly influence muscular movements. There appears to be some analogy between epileptic convulsions and the involuntary reflex movements which are common in paralysed limbs, and which cease with the return of voluntary power over the limbs. Cases of hemi-convulsion, without loss of consciousness, may on this theory be explained by supposing that while the circulation through the hemispheres remains uninterrupted, there is a sudden temporary occlusion of the arteries which supply the corpus striatum, and the irregular movements then result from the controlling influence of the hemispherical ganglia being thus cut off from a portion of the motor tract. There is an immense reserve of latent force stored up in the nervous centres. This force is normally under the control of the

will; but it is liable to sudden and explosive discharges when the machinery of volition is out of gear, as it is during an epileptic fit. The influence of the will in warding off a fit is shown by the fact that, in most cases, the attacks are more frequent during sleep, while, in some instances, they occur only during sleep when the will is off guard.

An arrest of the cerebral circulation from any cause, of necessity, involves a suspension of the functions of the brain. For this suspension of function, it is not necessary that the vessels should be empty; they may be full, and even gorged with blood; but, if the blood be stagnant, the effect is essentially the same as if they were bloodless. Great venous turgescence may so retard the current of blood in the capillaries as to suspend the functions of the brain. Within the last few weeks I have seen two patients, in each of whom a sudden and complete loss of consciousness occurred during a violent fit of coughing. One man fell and cut his temple during the brief loss of consciousness. The explanation of such an attack is, that during the violent expiratory efforts of coughing the blood is driven back into the veins, which consequently become so turgid as to check the capillary circulation within the brain. So that, whether the suspension of the cerebral functions be the result of the arrest of the arterial or of the venous current; in either case, the symptoms are immediately due to defective capillary circulation. In like manner, the functions of the kidney are suppressed by ligature of the renal vein no less than by ligature of the renal artery. The functions of every organ require for their discharge a continual supply of moving blood. Great confusion has often resulted from a disregard of the undoubted fact that the activity of the circulation through an organ is often in an inverse ratio to the "congestion" of its small vessels. In other words, the vessels are gorged, because the blood cannot readily pass through them.

Epileptic loss of consciousness differs from ordinary syncope in the suddenness of its occurrence. We have already seen that anæmia of the brain, to cause convulsions, must be sudden and extreme. In cases of syncope the circulation *gradually* fails, in consequence of diminished cardiac power, and there may be complete loss of consciousness without convulsions. Syncope, in proportion to its suddenness, approaches in its character to epilepsy, and there are certain cases of fainting, with semi-convulsive shudderings, which show that the boundary between epilepsy and syncope is sometimes ill defined and difficult to trace.

During a severe and prolonged convulsion there is usually much rattling noise over the chest, resulting from the mixture of air and mucus in the bronchial tubes, and frothy mucus is forcibly driven from the mouth and nose. This mucus is an exudation from the bronchial mu-

cous membrane, and the explanation of it appears to be this. While the respiratory muscles are in a state of spasm—respiration being imperfectly performed—the movement of blood through the lungs is impeded by the contraction of the pulmonary arteries; there is consequently an accumulation of blood in the right side of the heart and in the veins; and this venous stasis, taking a retrograde course, distends the bronchial veins and capillaries, which relieve themselves by a mucous exudation into the bronchial tubes. Sometimes the capillaries are ruptured, and the mucus is tinged with blood, even when the tongue is not bitten and bleeding. Precisely similar phenomena occur in all the forms of apnœa; for example, during a fit of spasmodic asthma, during the death struggles of drowning and suffocation, during the coma and the consequent apnœa from brain disease or narcotic poisoning; and what is vulgarly called the "death-rattle" is a noisy mixture of air and mucus in the bronchial tubes, the mucus being a passive exudation from the bronchial capillaries, the result of an increasing accumulation of blood in the right side of the heart and in the systemic veins, while respiration and circulation are simultaneously failing.

Extreme dilatation of the pupil appears to be a constant phenomenon at the commencement of the epileptic seizure. This is usually attributed to spasm of the radiating fibres of the iris under the influence of the sympathetic nerve. It is a positive fact, that galvanising the cervical sympathetic dilates the pupil at the same time that it excites contraction of the minute arteries, and a similar influence is supposed to operate in epilepsy. It is, however, conceivable that the dilatation of the pupil at the commencement of an epileptic fit is a secondary result of the extreme anæmia of the retina, just as the pupils of a chlorotic girl are dilated until her natural colour has been restored by iron. The pupils are dilated in cases of ordinary syncope, when the retina is anæmic from failure of the circulation, and when there is no suspicion of spasm of the radiating fibres of the iris excited by a stimulus being conveyed through the sympathetic. And again, in Kussmaul and Tenner's experiments upon animals and upon men, it was found that when the brain, and of course the retinæ, were rendered anæmic by ligature or compression of the arteries, the pupils dilated as in an ordinary epileptic fit. Yet here there is no reason to suppose a primary influence of the sympathetic acting simultaneously upon blood-vessels and iris. In these cases the dilatation of the pupil was a secondary result of anæmia of the retina, and so it may be in ordinary cases of epilepsy. This is a point of curious physiological interest rather than of practical importance, but it appears to me to be one which requires further investigation.

It has been too much the custom to look upon epilepsy as a separate entity without reference to the physiological history of the disease, and the treatment has, for the most part, been a blind empiricism. The sources of epilepsy may be as numerous and as varied as the sources of apnœa, with which, both as a cause and a consequence, it is so closely correlated. To prescribe for epilepsy without reference to its cause, is as unscientific as to prescribe for loss of voice without first ascertaining upon what conditions within the larynx or elsewhere the aphonia depends. As aphonia has for its immediate cause a defective vibration of the vocal cords, so the proximate cause of epilepsy is a defective blood-supply to certain parts or to the whole of the brain. The problem in each case is to ascertain the antecedent conditions and the circumstances which have led to the functional defect.

A priori considerations might have taught us, what experience amply confirms; namely, that in the two classes of epileptic cases very different remedies are required. In the purely nervous cases, the indication is to remove all sources of irritation, whether mental or bodily, to soothe the nervous system by anæsthetics, and to impart vigour by a well regulated nutritious diet and by tonics. On the other hand, in the toxæmic cases, the primary object is to free the blood from noxious impurities. In effecting this object, active eliminative and depletory measures may be required; as, for instance, in cases of uræmic poisoning. A local anæmia, the result of a toxæmic influence upon the cerebral arteries, may co-exist with a general vascular plethora. The argument that epilepsy cannot be a result of cerebral anæmia because it sometimes occurs in plethoric subjects and is sometimes relieved by depletory treatment, appears to me to be based upon a misapprehension of the terms of this most interesting pathological problem.

Convulsions of the mixed character before referred to (page 10) may require a combination of the evacuant and the sedative treatment.

It is not now my intention to enter into details as to the treatment of epilepsy; but there are two remedies upon which I desire to say a few words; namely, chloroform and bromide of potassium. It is a well known fact that chloroform inhalation has a remarkable power of arresting epileptic convulsions. Its action in warding off a threatened fit, and in cutting short a violent and prolonged paroxysm, is uniform and certain; as uniform and as certain as the influence of sudden and extreme anæmia in exciting convulsions. I, for a time, supposed that the chloroform acts by relaxing the cerebral arteries; but Kussmaul and Tenner have shown that animals when etherised, get no convulsions while being rapidly bled to death, or when their arteries are ligatured. It is

probable, therefore, that anæsthetic vapours prevent or stop convulsions by lessening the reflex excitability of the nervous system, so that convulsions do not occur in etherised rabbits, even though the brain be rendered extremely anæmic by hæmorrhage or by arterial obstruction.

Recent experience has amply proved that the bromide of potassium, in full and frequent doses and sufficiently long continued, is of great value in the treatment of epilepsy. The known physiological action of this medicine renders it probable that its curative effect in epilepsy is a result of its soothing, sedative, anæsthetic influence upon the nervous centres, whose reflex excitability it lessens. In short, its action in preventing convulsions is analogous to that of chloroform, differing in being less powerful and rapid in its operation, yet, by frequent repetition, its influence may be rendered more durable and more permanently beneficial.

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