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ORIGINAL ARTICLES.

ON THE CLINICAL STUDY OF EPILEPSY.

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MR. CHAIRMAN:—When the eminent President of this Association, seconded by yourself, asked me to prepare some remarks for opening a discussion before the Section on Medicine, I felt that the desire was not so much that I should give a formal paper, as it was that I should bring before the body some subject of general interest, and one in which all could take part.

The subject of chronic convulsive affections, grouped under the name of epilepsy, is one of such large medical and social importance, that I felt that I could not do better than select it, large and vague as it is. In what I say, I shall simply bring before the Section the notes of a few cases from my case books, and make them the subject of brief practical comments as I pass along.

The conception that I think we must form of epilepsy, is a very broad one. For studying the disease to the best advantage, if we could entirely exclude cases where any anatomical change has occurred, it were desirable. While it is perfectly possible to do this in cases where definite symptoms of cerebral tumor are associated with convulsions, and where we recognize that the attacks are purely symptomatic, there are many cases which we have to call epilepsy, where doubtless there are minute changes in the meninges or in the motor centres of the cortex, undistinguished by other symptoms. Prominent among these are the cases of epilepsy following sunstroke, of which I shall give illustrations later.

But there may be difficulty, even when a distinct history of traumatism exists, and unquestionably, if autopsies could be secured in every instance, it would be found that all collected series of cases of epilepsy which have served as the basis of statistical calculation, contain many cases of purely symptomatic epileptiform character. The following is quoted, partly as an illustration of these remarks:

CASE I. — *Epileptic Attacks following Injury to Head, Recurring after long Interval of Immunity. Abscess in Anterior Lobe of each Hemisphere.*

Dr. O. A. R., age 44 at the time of his death; married; excellent habits, and without hereditary predisposition. At the age of 23 was thrown out of his wagon, striking his head against a tree, producing a depression of the frontal bone to the right of the median line, over the frontal sinus. He had convulsions at the time, but recovered perfectly, continued his practice successfully and served as an army surgeon very creditably, and had no convulsions, nor any cerebral symptoms whatever, for 18 years, until August, 1881, after which time he had severe convulsions at intervals from four to six weeks. There was no albuminuria. No cardiac disease. No symptoms of pressure, nor any sensations referred to the spot of injury. The recurrence of the convulsions seemed to be connected with sustained mental anxiety and worry and the effects of continued hard work, as he had no vacation for 16 years. Ophthalmoscopic examination gave negative results. Bromides failed to influence the spells. The actual cautery was applied to the neck, and a strict diet was ordered, with cessation from work. There was marked temporary improvement, the intervals between the attacks being increased to six months. He then became so much better that he insisted upon resuming practice, when immediately violent convulsions occurred, which were uninfluenced by any measure. Trephining was at last performed by Dr. Agnew at my request, when slight depression of the internal table, with a fissure and displacement inward of a minute spicula of bone were found, with an old abscess of considerable size in the anterior lobe of the right hemisphere, and a small abscess in the anterior lobe of the left side. There can be little doubt that a slow irritative lesion had existed all these years, and the brain became habituated to its presence, and it was not until a large area and overwork established convulsions, which thenceforth were uncontrollable.

Equally difficult and yet important is it to recognize and eliminate all instances of hysteria. In typical cases of hystero-epilepsy, such as the following, this is readily done.

CASE II.—*Hystero-Epilepsy, Consequent on Membranous Enteritis.*

Mrs. McC., age 30. Several of her family have presented nervous symptoms, neurasthenia, etc. She herself was in perfect health until, after over-exertion nursing a relative, she was seized with severe dysentery, which passed into a subacute form and has been followed by membranous enteritis, continuing now for more than eight years. The membranous formations are peculiarly numerous, long, thick and firm.

Gradually a condition of marked neurasthenia developed, with excessive mobility of the system, and in the course of two years decided hysterical tendency showed itself. She became easily moved to tears, although her usual state remains one of great animation and cheerfulness. Excessive spinal tenderness was developed and, indeed, the entire body has become morbidly sensitive. Attacks of hystero-epilepsy first appeared five years ago, and still continue, though less frequent and severe than formerly. They are very characteristic and interesting, and can be induced at will by pressure on the spine or over the ovaries; also by fatigue or excitement, and are most common at the menstrual period. Ovarian compression will not check them. There is no erotic manifestation. Her character remains frank, generous, self-sacrificing and courageous. General nutrition has been well preserved. Menstruation is regular, but scanty and somewhat difficult. Obstinate constipation, connected with atony of the lower bowel, is a chronic condition. Prior to the passage of a membrane the general hyperæsthesia is exaggerated, great abdominal distress exists, and crops of from four to twelve dark-bluish stigmata appear on different parts of the body, chiefly on the legs and thighs. These spots look like bruises, are often a half inch in diameter, and persist for five to twenty days, gradually fading and changing color as when a bruise is passing away. At such times the convulsive attacks are frequent. Bromide has done no good. No remedy, either by the mouth or rectum, has cured the membranous formations. Repeated application of the actual cautery at different parts of the body has been made, and each time with marked and somewhat enduring good results; and repeated changes of scene, and wholesome, cheerful influences, have had an excellent effect.

Contrary to the teachings of Charcot, my own experience would be that in the cases of hystero-epilepsy seen in this country, compression of the ovaries does not arrest the attack, nor are erotic manifestations prominent. In this respect the disease as it occurs in this country and in England, is in marked contrast with the disease as it is said to occur in France, particularly in hospital patients.

While such cases as the above are easily distinguished, there are many others where the convulsions occur in such manner, and exhibit such coördinated movements, as to render it difficult to say whether they are hysterical or epileptic. Indeed, I think that it must be conceded that there are cases of epilepsy and hysteria combined. In both of these affections, we must undoubtedly recognize some rooted morbid sensibility and instability of nerve-tissue, probably distinct in its seat in these two affections. In epilepsy it would seem that there is a supremely unstable condition of one or more areas, minute or extensive, of the gray matter within the encephalon, rendering it liable to sudden and violent discharges which it may possibly be that in hysteria, in hystero-epilepsy and in the combined forms to which I have alluded, it is the ganglionic nerve centres which are in this unstable condition. Instability of the encephalic gray matter may co-exist, so that an attack may be induced through violent disturbance of the ganglia controlling intracranial circulation, with consequent discharge from an unstable centre in cortex or elsewhere.

Without entering farther on this abstruse question, the nervous instability which must be recognized as the fundamental element, is brought about in various ways. The influence of heredity is so well known that no further allusion to it is necessary. The effects of nervous exhaustion as from overgrowth, over-strain or exhausting illness has long been recognized. The exhaustion which follows typhoid fever, furnishes a good example of the influence of this factor, as in the following case:

CASE III.—*Epilepsy following Typhoid Fever.*

Mrs. B., age 38, had been in good health until five years ago when she had a very severe attack of typhoid fever. This was complicated with hemorrhages, phlegmasia alba dolens of both legs, and a relapse of the fever kept her in bed four months. On the first occasion of going out to walk after the fever she fell in severe convulsions of epileptic character, with tongue biting. This was the first convulsion she had presented. For three years subsequently, convulsions of an epileptic character continued to recur at varying intervals, sometimes as frequent as twice a week and of severe and typical character. They were often induced by exertion in the sunlight or by sudden exposure to very bright light. Gradually, as her health improved, the attacks became less severe and less frequent. For the last twelve months she has had no general convulsions but has merely had minor attacks with momentary unconsciousness preceded by sharp praecordial pain; at increasingly long intervals.

In the same way epilepsy frequently developes after scarlet fever, but in reference to the relation of epilepsy and scarlet fever, I shall speak more fully, later. Shock to the nervous system, whether this be purely psychical, as from fright; or whether it be from such influences as exposure to the violent action of the sun; or, whether it has been connected with an actual injury, with or without distinct lesion of the cranial bones, frequently appears as the exciting cause of the disease. I have already alluded to the great frequency with which heat-exhaustion, or actual sun-stroke, figures in this relation, and the following case is quoted partly to illustrate this, and partly with reference to certain points of treatment.

CASE IV. Mr. G. W. W. His mother has borne a number of healthy children, but is herself, at the menopause, highly anæmic and neurasthenic with tendency to asthma. He is 29 years old, and has been subject to severe sick headaches from boyhood. Four years ago, while working on a raft, was overcome by the heat and was attacked with violent headache and nausea, loss of consciousness and afterwards had fever with delirium. His general health was much impaired, and in addition during the next eighteen months, he had six severe attacks and a number of minor attacks. The severe attacks began with convulsive pain in the left side of the head, about the left mastoid region, increasing in intensity

until it became unindurable in from two to five minutes, and he then became unconscious. In the minor attacks the pain would be on the right side of the head and around the right eye, and increase until he goes into a stupor and becomes almost, if not quite, unconscious, and afterwards passes into a heavy sleep for hours. The severe attacks are preceded by confusion of vision, objects look blurred, move and close in upon him until all gets dark. This takes twelve or fifteen minutes, then the speech becomes affected; he thinks of objects he wishes to mention, but calls them by the wrong name, this increases until he cannot speak at all, though his tongue is not paralyzed. These changes are going on during the latter part of the vision changes, and reach their height about the same time, and then a numbness or tingling begins in the left biceps extending down to the hand and left side to the foot. The left side feels cold and numb and tingles, and he then loses power of moving left arm or leg, though he can still move right members freely. This is the last he remembers. He is usually able to reach some place to lie down. After this there are convulsive movements with tongue biting, and very excited action of the heart. The urine is normal. No cardiac murmur, but heart's action easily excited.

When I first saw him in 1882, there was marked tenderness over the left suboccipital nerve behind the mastoid process where the pains begin at the attack.

There has been great excess in sexual intercourse, which had probably aided in producing his constitutional susceptibility. Bromides and repeated blisters failed to produce marked effect. The actual cautery was applied February 8, 1882, and the effect of this, with care in diet and avoidance of severe exercise or much exposure to the sun, were followed by immunity until the summer of 1883, when he began to expose himself too much and to have headache; and in September, while driving in the hot sun, he had an attack beginning with flashes of light, sense of surrounding objects drawing in on him, though he was able to guide his horse home and reached his room, where he broke three capsules of nitrite of amyl, which did not prevent the attack from going on to complete unconsciousness and convulsions, followed by stupor for three hours.

Renewed application of cautery was again made, and restriction of habits has been insisted upon, and he is again improving. The beneficial effects of the cautery, and avoidance of exertion, was very marked here for many months, in the absence of both minor and major attacks, as well as of headache and uncomfortable sensations about the head.

Again, instability of the circulation with associated disturbance of the nutrition of the brain, as in heart disease, anæmia, etc. plays an important part as a predisposing cause in many cases. And I would call especial attention to the remarkable frequency with which careful physical examination will show the existence of organic cardiac disease in cases of epilepsy. In this connection allusion is also to be made to the possibility of minute capillary embolisms and interference with the nutrition of small areas of the brain in cases of valvular disease of the heart. Prolonged peripheral irritation may gradually bring about this instability and establish the habit of convulsive seizures. This peripheral irritation may be from prolonged morbid dentition, from severe and protracted genital irritation, or from long continued gastro-intestinal irritation, especially in the form of chronic catarrhal irritation of the mucous membrane with the resulting anæmia and malnutrition.

Such general considerations help us to realize the condition of those whom we group under the name of epileptics, and I think show us that they are not affected by a single definite disease, presenting a vast variety of type and severity, but that they exhibit in common, merely a state of impaired nutrition and of morbid instability of the gray matter within the encephalon, however induced or maintained and varying in degree to an immense extent in different cases, and thus rendering them liable to explosive discharges or convulsions upon provocations of correspondingly varying intensity. There are in all probability cases where progressive minute molecular changes in the nervous tissue induce the outbreaks at certain irregular periods. Such may be the cases of recurring status epilepticus with elevated temperature, and followed each time by evidences of advancing deterioration and also such cases as exhibit distinct progressive impairment of cerebral function. Such convulsions may then be as truly symptomatic as in the case of intracranial tumor. But far more commonly the irregularly recurring convulsions are connected with not an advancing morbid tendency, nor an irregularly progressive anatomical change, but with the occasional and irregular operation of those widely different causes which in different cases are calculated to disturb the weak centre and induce the explosive discharge. Unquestionably, it is true that the evil effects of habit are seen here as prominently as in other chronic conditions, and if the tendency or instability can not be reduced and the provoking causes be avoided, then the mere continuance of the attacks induced by such provocations will develope a more and more facile response on the part of the weak centres until at last attacks may be induced by almost imperceptible causes.

It is important to recognize the degree of this instability in each case, and to recognize that we are not dealing with a radical morbid condition, but merely with an exaggeration of the normal condition. Every one is liable to convulsions: it is merely a question of the provoking cause required. Take the following case of single convulsive seizure in an adult consequent upon intense dental irritation:

CASE V.—*Single convulsion, induced by intense pain.*

Mr. — 25 years old, with gouty diathesis, plethoric and somewhat intemperate in habits. Was seen in 1879, after he had a violent convulsion with tongue biting, followed by confusion of mind for several hours. It had been preceded by violent toothache lasting 36 hours, preventing sleep or eating, and finally becoming so severe that he felt he was losing his self-control, which is the last he can recollect. No subsequent spell has occurred.

Or the following case where alcoholic excesses induced the convulsions:

CASE VI.—*Epileptic convulsions apparently induced by Alcohol.*

W. J. F., age 45, had drank whiskey to excess and used tobacco to excess. Had primary syphilis 25 years ago followed by no constitutional symptoms. His general health was excellent until 5 years ago when he began to have attacks of acute dyspepsia following his hard drinking. Hemorrhoids ensued which were operated upon, and five weeks afterwards he had epileptic convulsions occurring in one of his attacks of dyspepsia. He had four more epileptic convulsions in the next two years, always occurring immediately after an attack of acute dyspepsia. Two and a half years ago he fell down stairs in an epileptic fit, and on recovering consciousness had complete loss of motion and sensation in the legs, following the blow upon the lumbar region. There has been a gradual return of power, though not complete. Since then he has stopped drinking to a great extent and has no more dyspepsia and no return of epileptic fits.

At the other extreme, we see cases where the tendency is so great that a sudden noise or a slight indiscretion in diet will induce severe attacks. It is not pretended that in all cases of epilepsy such provoking causes of the attack can be detected, but merely to bring into prominence the fact that the more closely we seek, the more frequently we shall find them, and that the avoidance of the attack is so important, and the recognition of the cause so valuable as a guide to restorative and preventive treatment, that a most critical and prolonged study in this direction is required in every case.

Turning now to the character of such provoking causes, I should mention as among the most frequent in my observation, first, indiscretions in diet, or improper food. I have already stated that this cause seems frequently to develop the tendency to the disease by impairing nutrition and inducing instability of nerve tissue, but also when such instability exists it very often serves to provoke the attacks. It may excite them by causing increased local irritation of the mucous membrane which will act in a reflex manner, or it has often seemed to me that it acts in some cases by inducing a condition of toxæmia from the admission into the blood of elements imperfectly elaborated, or from the failure of the emunctories to remove some product of malassimilation.

We are becoming more familiar with the wide range of symptoms, largely nervous in type, associated with the condition inadequately named lithæmia. In a considerable number of my epileptic patients it has seemed to me that their attacks bore a close analogy to the attacks of vertigo, for instance such as we see induced in lithæmic patients by indiscretions in diet. I believe it is difficult to lay too much stress upon the part which disorders of assimilation and nutrition, and the derangements of the associated functions of the great abdominal organs, stomach, liver and kidneys, play in the production of instability of nervous centres and the varied demonstrations or phenomena of such disorders as epilepsy and hysteria.

In this connection, it is interesting to again revert to the frequency with which scarlatina seems to have a causal connection with epilepsy. This may be explained in some cases by the tendency to wide spread tissue-change in this disease so that impaired nutrition of the gray nervous matter might be expected sometimes to occur. But it seems to me probable that in many other cases it may act by leaving such a degree of renal insufficiency as will under comparatively slight causes lead to toxæmia, due to the retention of malassimilated materials. It does not seem necessary that such a condition should reveal itself by the presence of albumen in the urine, although the statement of Huppert (Archiv für Psychiatrie, 1877, p. 189) that immediately after epileptic attacks albuminuria is almost invariably present and hyaline tubecasts can frequently be found, (while certainly exaggerated and not fully confirmed by subsequent investigators), calls attention to this interesting question. The cases of epileptic convulsions to which I especially refer, are those where previous to the seizure some derangements of digestion or secretion make themselves evident for a short time. Failure, impairment, or alteration of appetite, dullness, languor, with heavy aching sensations through the limbs; changes in the appearance with heaviness of expression, coating of the tongue and foul breath; constipation with alterations in the appearance of the urine, this and other similar premonitory symptoms will be found of frequent occurrence if carefully inquired after, and point in the direction of what I have been saying.

Among other provoking causes are excessive excitement; sexual excesses, inducing exhaustion; alcoholic excesses; great intellectual exertions, especially if associated with the excitement of competition; and excessive muscular exertion. A single severe muscular effort may suffice to induce the attack (see case 10). The importance of this is especially marked in cases where there is cardiac weakness. In the following case, where I could detect no disturbance of the heart's action, fatigue induced by muscular effort certainly increased the attacks of petit mal.

CASE VII.—Marion S., age 22, no hereditary tendency, began at the age of 19 after a course of excessive study at college combined with injudicious habits of life, to have characteristic minor attacks marked by palor and brief but complete loss of consciousness. She was a girl of strong will without the slightest hysterical tendency. No cardiac lesion or albuminuria. Attacks recurred at irregular intervals and close study revealed that they were apt to occur in connection with menstruation or to follow decided muscular exertion, for instance when driving her horse became startled, and while she was not at all alarmed it was necessary to use powerful exertion to control the animal. Following this there was a series of minor attacks at short intervals. Rest in bed for several weeks with gradual return to exercise but with complete rest at the menstrual period, carefully regulated diet and dialyzed iron in ascending doses broke up the spells which have now been absent for more than two years.

In those cases in which the morbid state of the

nervous system has been brought about by sunstroke, the attacks will often be induced by exposure to excessive heat, or to intense light. Of course, all these causes become operative on account of the morbid sensibility and instability of the nervous tissues.

It is often said that many epileptics are in full health. This certainly does not coincide with my own observations. Rarely has it failed to be the case that a critical study has not revealed either some organic trouble or some marked functional disorder. We do not speak of a lithæmic patient who has vertigo or other lithæmic symptoms on trifling indiscretions as being in perfect health.

The principles of rational treatment would seem to follow from a consideration of the points to which I have briefly referred. In no disease is routine treatment less permissible, and in no disease is attention to hygiene and general influences more essential. In each case the treatment should be adapted to the special peculiarity, to the character of the primary cause, if such can be discovered, and to the character of the provoking cause if it can be detected. Among the general principles which could be dwelt upon, I shall briefly allude to the importance of relieving anæmia and neuræthenia and the morbid susceptibility by diet, by change of occupation and residence, and by rest. I would dwell upon the special value of prolonged rest in certain cases, which in my own experience have been chiefly met with among growing children. As for instance in this case:

CASE VIII.—*Epilepsy apparently cured by prolonged rest, careful diet, with small doses of bromides.*

F. S., age 15, a boy of unusual intellectual powers, and very ambitious. He had convulsions while teething and at the beginning of scarlatina, at 3 years of age. Healthy until 8, when epileptic attacks made their appearance, and proved obstinate, yielding only when he was removed from school and sent to a farm, and kept under the influence of small doses of bromide. He was then allowed to return to school, but after again devoting himself to his studies minor attacks began, and they were soon followed by frequently returning nocturnal severe attacks. These recurred with increased frequency. They were not controlled by bromides. The urine contained neither albumen nor sugar; no cardiac lesion. Appetite was good, but previous to this last outbreak of the attacks and during it, he had too frequent movements of the bowels—not actual looseness, but two or more occurring in the day, and usually with some little mucus. The minor spells occurred during the day, the severe convulsions in the night or early morning, and finally by February 1, 1883, more than one occurred in a single night. Congenital phymosis existed, but as no irritation appeared it was allowed to remain, He was then put to bed and kept there constantly, with very carefully regulated diet, 10 grains of bromide of potassium and 2 grains of quinine twice daily, substituted later by cod liver oil. He remained in bed strictly for two months and a half, during which time his attacks, both major and minor, ceased entirely. He grew and gained weight while in bed. The disposition to looseness was controlled entirely by diet. The bromide was suspended, cod liver oil continued. He was then cautiously allowed to rise for a short time each day, and gradually increased until he returned to his ordinary mode of life. There has been no relapse in fifteen months, and a cure is apparently complete.

It is especially in these cases where there is anæmia, defective nutrition, and neurasthenia, that cod liver oil, iron, strychnia, arsenic, and quinine have proved so valuable.

Gastro-intestinal irritation should be relieved. Possibly this is more especially applicable to cases where the aura is in the pneumogastric area, although it is applicable where the aura is absent or is in the course of other nerves. It is certainly applicable in cases of gastro-intestinal catarrh. In some of these cases, extreme benefit will be obtained from an absolute milk diet. The following case is of great interest in illustration of this statement:

CASE IX.—Charles Wilkinson, family not strong; disposed to be anæmic and somewhat nervous. One sister had marked anæmia; a brother had incipient phthisis of right apex, cured by long treatment and change of climate, and his father suffered a great deal from gastric trouble. Charles is now 19 years old, and was apprenticed to a plumber at 16½, when he seemed very well. He worked hard, and when two years older he had a severe pain in the back, which was either rheumatic or spinal, probable the former, and was treated under the name of meningitis, and was confined to bed for five or six weeks, and did not recover entirely until he came under my care. This trouble in the back seemed to be lumbago; it was in the lumbar region, and affected him so that when he was in a sitting position he could scarcely rise, but after he had walked a little he grew easier. There was no vomiting. He ate rapidly and heartily. The pain in back began quite severely, though he kept about for two or three weeks. Then, when at work on a roof, in March, 1881, he was seized with such intense pain in the back that he had to be helped off the roof, and was brought home and put to bed. While in bed, two or three weeks later, he had his first epileptic fit (the first one followed administration of a dose of oil, and was more like a syncopal attack). Then he had for at least three months (every day, or at most every third day) similar spells of sudden unconsciousness, eyes fixed and staring, no convulsive movements—lasting a few moments or two or three hours as though dead—occurring by day or by night. Face pale, but not as in a faint. Breathing very gentle during attack, and as he came out of it, very deep and heavy. He would have no consciousness of it afterwards. When they came on he would say, "I am sick; I am sick," and then go off into unconsciousness. Towards end of the two months these spells grew more frequent, even several in a day, always the same. When suddenly while lying in bed, to which he was confined then from pain in the back (lumbago?), he was seized with a fully developed violent epileptic attack (no apparent cause, and no excitement).

He was then using bromide of potass. and bell., and had eaten that day two bananas. [It is to be noted

that there was no vomiting, but his tongue was coated. He often complained of being sick all over. Appetite poor, flatulent, constipation, for which purgatives were given (senna and prunes), and the attacks were always preceded for an hour or so by great distress in the stomach. The stools contained mucus, jelly-like, greenish.]

After this he continued to have violent epileptic attacks every few days, until by September 1, 1881, they were two or three times a week. In the intervals he would have the peculiar petit mal as above described. The spells were all complete. He would feel they were coming—not always, for he had them during night in sleep—pain in stomach, like a cramp; often sudden flatulence, the stomach swelling up in two or three minutes. He would fall if not caught. Convulsions very long and severe, followed by stupor; frothed at the mouth; never bit his tongue; had no consciousness or recollection of the attack. Treatment by large doses of bromides and laxatives continued. He gradually got better of pain in back, and in course of ten or twelve weeks from the beginning he could leave bed, though he did not walk out before August, and then always with a companion.

I saw him in September, when he was looking poorly—pale, thin, and anæmic. Marked gastric irritability; connection of attacks with gastric disorder suggested itself prominently. He was then having his attacks very frequently, nearly every night just after falling asleep, and not rarely, also, one or two during day.

He was ordered strict milk diet, ℥iss every two hours, gradually increasing to two quarts. This was continued for three months; not a crumb of bread during this time. Nitrate of silver and opium, inunction, twice daily; also nitrite of amyl, which stopped the attacks, if there was a chance of using it. The attacks continued for three months, growing less and less severe and frequent, and then stopped, and he had none for nearly a year. He regained his health; was able to return to work. Subsequently he has had six or eight attacks at long intervals, when careless in diet or when he overworks.

No bromide at all was used. On one occasion he had two or three attacks at shorter intervals, and I confined him again to milk and gave him a course of silver, after which he had no spell for a long time.

No treatment now for a year; no spell during that time, except once after mental worry he had a slight attack.

CASE X.—B. F., age 11. His father was of a highly nervous temperament, and had one uncle an epileptic, who died aged 38, of rapid phthisis. The maternal grandmother had heart disease, and for 20 years before her death had convulsions frequently, about once in two weeks. One grand-aunt had paralysis. His mother had two sisters, both of whom died with kidney disease with frequently recurring convulsions. One cousin had convulsions. While his mother was pregnant, she was much worried about her mother, who was then having convulsions, and died three months before the patient was born. He had distinct carpo-pedal contractions with fever when teething. His stomach was always very sensitive, and the least excess excited fever. At such times, curious spells of rapid, sighing breathing would occur, and would recur until an emetic was administered, after which they would promptly cease. In August, 1880, when seven years old, he fell from the second story, striking his head upon two nails projecting from the wall and then striking the floor, but not losing consciousness. Soon after this, he began to complain of minor attacks, consisting of transient rapid vibration of the tongue, which he called "quivering," without loss of consciousness. I saw him first in October, 1881, and detected organic mitral insufficiency. He had been cutting teeth recently, and the minor attacks had been more frequent. His first severe convulsion occurred in November, 1881, connected with dental irritation. Bromides were given in small doses, extreme care was used in diet, and no convulsions occurred until the following summer, when dental irritations about the incisor teeth presented themselves, and quivering attacks began at once, occurring frequently; and he soon had two severe convulsions. After dentition was established, his attacks became much better, but in September, 1882, in spite of bromides in large doses, minor and major attacks recurred. I then found tightly adherent prepuce, with much irritation. This was separated, and the attacks again ceased, although bromide was now abandoned, as it had failed to control attacks. There was now a period of immunity for a number of months, during which time excessively careful diet was used, and, whenever there was evidence of irritation of the system, chloral enemas.

Since then there have been several outbreaks, the minor attacks of quivering growing frequent, and full convulsions occurring at short intervals. On one occasion, such a state was directly induced by violent straining muscular effort trying to lift a heavy weight, which brought on severe cardiac disturbance. Strict rest in bed, digitalis and very careful diet, with a few chloral enemas, broke this up. On several other occasions, the development of these periods of activity has been preceded for a few days by evident disorder of assimilation, the complexion growing thick, dark rings under the eyes, breath heavy, stools less natural. Under such circumstances, neither bromide or chloral enemas have stopped the convulsions. An emetic of ipecac in warm water, with very rigid diet and almost complete rest, have broken up the spells, and have secured immunity for a number of months. In this way, there has been a great improvement during the past 18 months without any bromide, and with the use of only occasional chloral enemas. Moderate study with a tutor has been permitted. The boy is developing finely, physically and mentally. The organic heart disease remains the same. The attempt has been made to arrest the quivering by a clamp which he carries to apply to the tongue, but without definite results. Nitrite of amyl always succeeds when opportunity is given. Bromides in any form, even in small doses, have lately seemed to disturb digestion, and do more harm than good.

It seems that it is in these cases, where the connection between the gastro-intestinal mucous membrane and the nervous system is so prominently marked,

that nitrate of silver is especially useful, either with or without the conjoined use of the bromides. I would also call attention to the importance of the occasional use of emetics in cases where the occurrence of attacks is accompanied by evidences of increased gastric disturbance. It has happened in my experience that, when spells of unusual severity were occurring with unusual frequency, despite the use of various anticonvulsives in full doses, the use of an emetic has been followed by an abrupt cessation of the attacks, and by immunity for a considerable period. (See case X.)

Over-exertion should be avoided in all cases, and especially in cardiac cases as will be seen from several cases which I have reported. It is in these cardiac cases that digitalis seems to be of value. Excitement, over-work and excessive study is to be avoided. Study in private should be preferred.

Counter-irritation and the removal of local irritation. In the first place I should allude to the value of the actual cautery, especially when intra-cranial irritation is suspected, as after sunstroke and after injury without demonstrable lesion, and even in organic cases, although we cannot think that the actual cautery could influence the gross cerebral lesions, yet its effect on the nervous system is sometimes such as to secure immunity for a considerable period.

Trephining. It is my conviction that this operation is not performed often enough for epilepsy. We may ask ourselves if it would not be proper to do the operation when the position of the discharging centre can be located by the aura, the character of the convulsions or from a history of an injury, even though no positive lesion of the cranium exists? When there is a demonstrable lesion, it should be performed.

Adhesions of the prepuce are to be broken up by dilatation, and the redundant prepuce is to be removed by circumcision, although from my own experience, I should say that the importance of this has been greatly over-estimated.

Lastly, the arrest of the attacks. Ligature and nitrite of amyl are useful in preventing individual attacks and in checking the formation of a habit. I have purposely confined myself to these general measures, omitting any reference to the drugs usually employed, notably the bromides. I have done this not because I fail to appreciate the enormous value of the bromides in epilepsy, or the fact that the discovery of the anti-convulsive powers of these agents has actually modified the whole question of prognosis in epilepsy, and rendered the disease more manageable than it formerly was, but because I think that there is reason to believe that the profession has drifted too much into depending upon the bromides, prescribing them in every case without studying the individual peculiarities sufficiently, and as a moderate dose fails, to increase it and continue it for a long time, until this course finally produces results injurious in themselves, and calculated to hinder the restoration of nervous power and the removal of the fundamental condition of nervous susceptibility and instability upon which the disease essentially depends.

Bromides do not cure epilepsy, except in rare instances. More frequently they control the disease, sometimes indefinitely, but more commonly losing their effect gradually, and this is the worst feature about them. In other cases, they fail to control the convulsions, and there are a few cases where the bromides have seemed to be actually injurious by depressing the digestive power, if they did not actually cause gastro-intestinal irritation. I shall not discuss the selection of the special form of bromides or any particular method of administration. The bromide of potassium has seemed to be the most valuable. Occasionally, it has been desirable to substitute this or combine it with some of the other bromides. As to the mode of administration; they should be given at first in minimum doses, and no more than is sufficient to control the convulsions should be administered. In many cases, I have found it necessary, after using the bromides, to either abandon or modify the treatment, and I have concluded that far more value is to be attached to hygiene, diet, and the more general remedies, than is to be attached to the bromides.

In like manner, I shall not attempt to allude to the other drugs, such as belladonna and other antispasmodics, which have been recommended, but will close with a brief allusion to the effect of enemas of hydrate of chloral, as having given good results in some cases where the bromides have failed. Often far better results are secured from enemas of chloral, than can be obtained from the administration of the remedy by the mouth. This method is of particular service where the administration of the bromides and chloral by the mouth seem to be disadvantageous on account of their tendency to disorder the stomach and digestion.

DISCUSSION.

Dr. Austin Flint, Sr., of New York, opened the discussion on the subject of Dr. Pepper's paper as follows:

An argument for the toxical pathology of epilepsy is not, as it seems to me, out of place in a discussion of that affection from the standpoint of the medical practitioner, inasmuch as our inquiries with regard to the prevention and treatment of diseases must, in a great measure, receive their direction from pathological views.

By the phrase "toxical pathology," I mean to express the doctrine that the manifestations of epilepsy depend on a toxical agent of some kind, produced somewhere within the body.

The first point in the argument is the absence of any generally received or satisfactory pathological doctrine. I assume, as a postulate, that epilepsy has no established anatomical characteristics. It may be associated with different lesions, and in a certain proportion of cases no lesions are discoverable. A rational inference from these facts is that different associated lesions, when present, have only an incidental pathological connection with the disease, if the connection be not purely accidental, and that the disease is essentially one of the neuroses. It may also be assumed that the manifestations of the disease generally occur without being preceded or accompanied by any apparent morbid conditions which may

be supposed to be causative. As a rule, epileptic manifestations have no distinct premonitions. They occur wholly irrespective of any causative agency from without. The varied sensations which have been described under the name *aura epileptica* are in most instances wanting, and the diversity in their character divests them of any special pathological significance. Pathological explanations such as are expressed by the terms "discharging lesions," (Hughlings Jackson) and "excitability," (Brown-Séquard) are not only purely hypothetical, but they convey nothing clearly explanatory. It may be said of the etiology, as well as of the pathology, that there are no obstacles to be removed in order to make way for a new doctrine.

The second point in the argument is the analogy between the phenomena of epilepsy and those known to be produced by toxical agents. The closest analogy exists between epileptic paroxysms and uræmic coma with convulsions. No one at the present day, doubts that the coma and convulsions referable to renal disease, are caused by retained urinary principles. Yet, within my recollection, uræmia not being recognized as a morbid condition, the coma and convulsions dependent thereon, were as indeterminate in respect of their pathology and causation as are the phenomena of an epileptic paroxysm at the present day. An attack of uræmic coma and convulsions may simulate a paroxysm of epilepsy so closely that the latter could not be discriminated from the former without the information to be obtained from an analysis of the urine. I have known several attacks of uræmic coma and convulsions to have occurred, and been considered to be paroxysms of epilepsy. On the other hand, in some cases of epilepsy in which the paroxysms recur after short intervals during many hours and even days, there is so close a simulation of uræmia that an examination of the urine is essential for the discrimination. Reasoning on the principle of like effects being referable to like causes, I submit as a reasonable conclusion that the pathology of epilepsy, in the same sense as the pathology of uræmia, is toxical.

As a fourth point in the argument, certain facts pertaining to the clinical history are more rationally accounted for on the supposition of a toxical causative agent, than by any other explanation. The facts referred to are, 1st. The absence of any definite ailments prior to the occurrence of an epileptic paroxysm, there having been no ailments, or those which may have existed being of an indefinite character expressed by the term *malaise*. This fact is alike applicable to certain cases of uræmic coma and convulsions. 2nd. The short duration of the epileptic paroxysm. 3rd. The immediate recovery in many instances of the general condition of health which existed prior to the paroxysm, and not infrequently a feeling of improved health. These facts are more consistent with the supposition that the epileptic paroxysm is due to the transient accumulation of a toxical agent, than with any other pathological consideration which may be supposed to exist. It is to be remarked, however, that the toxical pathology by no means excludes the probability that certain local morbid conditions are more or less important, and may be essential, or auxiliary, constituting a susceptibility to the action of the toxical agent.

As a fifth point in the argument, facts pertaining to therapeutics uphold the toxical pathology. Nothnagel in his treatise on epilepsy, opens up the consideration of the treatment in these words: "Small indeed is the actual encouragement derived from looking through those chapters of medical literature, from the oldest to the most recent times, which refer to the treatment of epilepsy. The methods and the remedies change; but the final result always remains the same beggarly one[1]." I presume no one will deny that this statement expresses truthfully the actual state of our present knowledge of the therapeutics of epilepsy. From this statement it may be inferred that up to the present time pathological views have failed in giving a successful direction to therapeutical observations. Of the remedies which within late years have been found to have a certain amount of influence over the disease, namely, the preparations of zinc, belladonna and the bromine salts, the most rational explanation is that they enable the nervous system to tolerate, to a greater or less extent, the toxical agent on which depend the manifestations of the disease.

It is needless to say that in this brief paper I have merely touched upon the general points in the argument for the toxical pathology of epilepsy, and I have omitted points which might be adduced. The limitation as to time (10 minutes) has prevented a fuller consideration of the subject. Accepting the toxical pathology of the disease, the pathological inquiries to which it gives rise are, what is the *modus operandi* of the toxical agent, what is its nature, whence is its source, and what is the mode of its production?

The questions which relate to the practical applications of the doctrine are, how can the production of the toxical agent be prevented, how can its morbific effects be neutralized, and how can it be eliminated from the body? In answer to these questions at the present time, nothing could be offered but conjectures.

Dr. P. O. Hooper, of Arkansas, remarked that little or nothing can be offered or suggested or added to the exhaustive paper of Dr. Pepper, for no subject in medicine has received more elucidation than epilepsy. It is interesting to note the steps of progress in the numerous investigations which have led to results nearly as clear as absolute demonstrations; so plain as to be accepted with singular unanimity by all who have carefully given the subject a thought. The one fact received, that anæmia of the brain and spinal cord is the direct cause of epilepsy, explains its phenomena in all their varied phases. Leaving out the classifications of *grand mal, haut mal, petit mal*, and the like as unnecessary, if the enquirers will regard both the disease and its direct cause as unities, it seems that a great stride will have been made in the direction of the simple truth as to its nature, causes, pathology and treatment.

C. J. B. Williams gave the key-note in vascular

[1]Ziemssen's Cyclopædia Vol. XIV., p. 279.

pathology when most of us were pupils. He has been followed by Addison, Waller, and Conheim. In another direction, Claude Bernard, Brown-Séquard and others were pursuing their studies of the nervous system. Others have been giving special attention to phenomena affected by the results of investigations in these same directions. Notice, epilepsy, as has been said, is the immediate effect of a sudden anæmia of brain and spinal cord. Analogy indicates as much, as shown in animals when bled to death. Post-mortem examinations and vivisections afford almost conclusive confirmation. Now, what superinduces the sudden anæmia? Constriction of vessels supplying the nervous centres. Then, an irritation anywhere, or from any cause, in certain predisposed constitutions may be sufficient to effect it, and the usual central and peripheral causes; whether the irritation exists in one tissue or another; whether it is found in brain itself, in the alimentary canal, uterus, or muscular tissue, or wherever it may be, the result is exactly the same. The irritation is conveyed by a sentient medium to the nerve centres, and reflected. In its course the motor filaments are influenced by the distribution of the sympathetic, and the vascular supply of the part to which the reflection tends is modified. If the point of tendency happens to be in the line of the nerve centres, and the modification should be a sudden constriction, epilepsy in some of its protean phases would be looked for, because the circulation would be suddenly shut off, so to speak, and the consequent anæmia give rise to epilepsy.

A late very excellent work by Dr. E. C. Mann, says in substance, that it seems very probable that owing to functional disturbance of the vaso-motor nerves which are distributed to the cerebral blood-vessels, we have during an epileptic paroxysm, primarily the vascular tonus just referred to, causing sudden anæmia of the brain, followed immediately by congestion and hyperanæmia.

The whole motor tract of the cerebrum, and also of the spinal cord, is undoubtedly connected with the production of epilepsy; and probably, also, the motor nuclei in the medulla oblongata and on the floor of the fourth ventricle, and the corpus striatum, as one of the centres of motion, is also concerned. The conclusion of this writer is irresistible, the *rationale* is clear, and the theory most plausible. Accepting this line of reasoning as true, a flood of light is thrown upon both the endless variety of manifestations in which the malady shows itself, and the efficacy or want of it of the almost endless list of remedies and remedial agencies that have challenged the consideration of practitioners.

The manifestations will depend briefly as follows: Upon the constitution of the patient and the nature and locality of the irritation. In respect to the former, an unstable, nervous organization, whether hereditary or acquired, is of the first importance as tending to a predisposition; as to the latter, they are endless in variety. In the treatment, any means or measures that will create and maintain a steady tone of the vessels which supply the nervous centres would be esteemed as most useful, whilst an agent sufficiently sedative and tranquilizing to guard and protect those tissues against the assaults of irritating influences, would rank as desirable adjuvants. Nitrate silver has long maintained a reputation based no doubt upon its tonic powers, but lately ergot has attained a recognized supremacy. In fixing attention to the bromides as almost a specific, Sir Charles Locock takes the attitude of a great benefactor to the race.

It would be futile to look for the remedies indicated without such an examination as would serve to locate the prime cause or irritation. Wherever found, its removal is a matter of first consideration. The agencies and methods must be determined by the facts and circumstances of each particular case.

Dr. Eugene Grissom, of N. C., remarked as follows:

Epilepsy as an *organic* disease is usually inherited, either as such, or as a sequel of some other neurosis. It is preceded, for the most part, by one or more eclamptic seizures in childhood, and commonly makes its onset at the period of puberty.

It may be the result of conception during drunkenness, or after any other prolonged depression of the system, leading to its enervation. It may serve as the representative of chorea, or of partial paralysis in the father, or of hysteria, or any wasting uterine or ovarian affection in the mother. It may accompany such disorganization of the brain as results from syphilitic cranial changes. It may appear in the child of phthisical, or scrofulous, or rickety parents. Finally, it may be the last expression of an ancestral history of degeneration, traceable, perhaps, for many generations (like the royal house of Spain), on its way downward, to extinction in cretinism.

The influence of hereditary bias in developing the unstable nervous condition which eventuates in epilepsy, from a parentage either already affected by some recognized neurosis, or else so far weakened and degraded from a normal vital standard by a succession of either moral or physical causes of depression, as to fail of nervous nutrition; this influence, I say, is acknowledged by all who have studied the statistics of this interesting subject. It is clearly exhibited by Echeverria in a table of 532 cases, and conceded by all writers of note.

It is hardly to be expected that any course of therapeutics can create a man anew, and restore in a single lifetime, the losses resulting from misused minds and bodies. It is impossible to repair in a day the long-continued breaches of systemic integrity made by persistence in evil habits, mental or physical. When, therefore, organic or idiopathic epilepsy is recognized, as clearly the result of gradual and profound degeneration, the prognosis cannot be hopeful.

The extremity of the difficulty lies in the very fact that the channels of restoration are themselves choked. If *pain* is the expression of *mal-nutrition*, the epileptic movement is believed to be, in the highest degree, the result of *in-nutrition* of the nervous centres. The very circulation through the vaso-motor apparatus, is disordered, irregular and fitful, and the stimuli to which the system would ordinarily respond with advantage, whether of food, or exercise, or sensory irritations of whatever character, are capable of becoming transformed to excitants, through the trophic

changes, by which the normal action is driven into tortuous and abnormal influence upon nerve centres, in the victim of epilepsy.

The deep-seated obstacle to a scientific therapeutics would seem to be the inability to control the channels of communication, in the case of the confirmed and inherited epileptic. The actions and reactions of ordinary organic life are modified by the introduction of a mysterious element, which capriciously breaks for a moment the usual interdependence of nerve or muscle, or nervous filaments and their appropriate centre. The expression of the face is gone, the voice changes, every muscle has taken upon itself strained and forced, perhaps unnatural uses; more than all, and most significant of all, the mental manifestations are those of another and apparently foreign creature—everything denotes that the fountains of the depths of being are broken up. We are not to suppose that all this irregularity of muscular and nervous movement on the surface, and this utter change of psychical expression, can be unattended with deep-seated change in glandular action, in the nutritive movements of the system, and, indeed, in the chemicovital processes in general.

It will be necessary to discover the secrets of the transmission of nervous force, before we can pronounce authoritatively upon the genesis of epilepsy, whether considered as the ultimate result of *lives* which disorder nutrition and disarrange nervous distribution, or as an immediate consequence of any agency of depression.

But that branch of the subject which is more fruitful of hope for the medical man, is the consideration of *functional* epilepsy—of that symptom, perhaps, rather than disease, which may be present in almost every disease, as some writer has remarked—which may accompany what is apparently a slight intestinal irritation, as from the parasites which abound in childhood, or from dentition, or the changes incident to menstruation, or the change of life; or, again, from such traumatic causes as a blow on the head or spine, a sunstroke, great cold, and certain moral causes like excessive fright, which sometimes strike down the system with such force that life itself hardly rallies after the blow.

The functional form of epilepsy is by far the most frequent, and its alleviation or cure demands, and ought to receive, much more pains-taking care and attention than it usually receives from the general practitioner. It is protean in its development, and must be met by protean efforts. Its two sources may be broadly defined as:

First. That which proceeds from any great draught upon the vital powers; as, in a physical sense, from a great loss of blood, leaving the nerve centres anæmic, or in a moral sense, from an overpowering fright, in some way producing the same anæmia by the shock, which leaves the nerve centre for the time being unable to appropriate the food required for momentary nutrition.

Second. That epilepsy which is set up by the suffering of the peripheral nervous trunks, from some perhaps minute but ever-irritating cause of disturbance of the terminating nervous filaments, either of the cutaneous or of the splanchnoid distribution.

If one is tempted to ask whether, in the latter case, the *post hoc* has been taken for the *propter hoc*, let us ask ourselves how we can account for the terrific consequences of tetanus as a result of an insignificant wound, or the propagation of fatal erysipelas from a mere scratch, which has really injured nothing of importance to the system, provided the nervous and circulatory distribution were unharmed; or, again, the spectacle of hydrophobia from a nervous injury scarcely visible, and perhaps long forgotten?

But this subject has been set at rest by the results of modern treatment—by the brilliant practice of Dr. Sayre, in relief of epilepsy from irritation of the genito-urinary apparatus—by the successful application of the trephine to relieve epilepsy from the entanglement of nervous filaments in a cicatrix—by the effects of nerve stretching in diseases of a choreic or epileptic character, confined to the track of the nerves of voluntary motion—by the cases of Battey's operation in which relief has been obtained from epileptic disorder, and such other evidence as medical science is daily supplying.

It is characteristic of *functional* epilepsy that it expends its morbid power chiefly upon the nerves of voluntary motion, and those most frequently used;—as, for example, upon the great toe in the foot, or the index-finger and muscles of the forearm, or again of the tongue, lips and eyelids. How often does the interruption of the delicate movements of the tongue in precise articulation betray the first weakness in the power of coördination!

Therapeutic measures, therefore, which are to be adopted in addition to constitutional treatment, should be directed to the tonic regimen of the voluntary muscular system, which is in danger of losing the inhibitory control over its vaso-motor circulation, by reason of nerve-weakness.

The connection of epilepsy with anæmia has been shown by Van der Kolk and others. It is emphatically an affection associated with weakness; with atrophy, or with loss of tonic ability to maintain control over the circulation, and *with that*, and in a much more significant sense, want of control of those manifestations of the brain power which accompany the *conscious* exercise of will, memory or judgment. This topic, if pursued, would open the intricate, and as yet unsolved problems of moral responsibility within the circle of the epileptic eclipse.

Dr. Hughlings Jackson (West Riding Asylum Reports) affirms that the epileptic paroxysm is a sudden, excessive and rapid discharge of gray matter of some part of the brain. The gray matter, which is the seat of what he determines a discharging lesion, is in a constant abnormal state of nutrition, and hence it is constantly abnormal in its function. He represents the primary attack of epilepsy as the discharge of a part which has been for some time in a state of mal-nutrition.

Certainly it is perhaps *not* a rash supposition that the brain, acting as a whole, with its allied ganglia controls the operations of systemic and voluntary motion by the harmonious action of the forces whose

origin proceeds from the nerve centres, inspired under proper nutrition, when of normal construction at birth and without subsequent injury, with the *vis vitæ* that will probably forever elude investigation, however thorough and delicate. If this harmony is conceived to be maintained by the original propelling energy of each centre, and also by the inhibitory control which each possesses upon the others, to a certain extent, we can perceive that a failure of nutrition in any centre, may leave its reciprocal or corresponding centre without the checking influence essential to the due marshalling of the whole movement in voluntary action, under the command of the will, if we may venture upon a metaphysical term in considering a physical problem.

The epileptic affection is essentially a stoppage or a transformation of the movements, not of muscular centres only, which are the mere epiphenomena which attract our vision, but it is deeper and more important; the irregular, involuntary, and exhaustive discharge is most effective in its consequences when the upper sensory and cortical centres are shaken and disordered, and intellectual perception and moral responsibility are tottering, or for the time being lying prostrate under the physical storm which has spent itself upward, rather than merely shaken the victim with choreic muscular spasm.

Whether nervous movement proceeds from one molecule to another by chemical adaptation and momentary molecular change induced by affinities paralleled by isomerism, or through some as yet unknown development of force akin to action by electrolysis, or any corresponding method of the transmission of force, it would seem that in epilepsy there is an interruption to the transmission of normal movement, a separation of the links, a stoppage in the even flow of nutrition for the cells, which harmonious organic life demands.

In regard to the therapeutics of functional epilepsy, what we have said will, in a measure, indicate the treatment.

The most careful attention to diet is required to build up the system, and supply the nerve centres with a sufficiency of pure blood. Regular and systematic, but above all things *un*fatiguing exercise follows as a matter of course. Fatigue simply invites exhaustion, anæmia of the medulla, and the paroxysm. How often we witness the epileptic convulsion as a sequel of exhaustive effort. Such climatic tendencies and daily occupations and educative influences as tend to build up the whole man healthfully and happily, and thus to erect a brakewater against all that would harrass, oppress, or wear the bodily or mental frame, are imperatively indicated. Much must be left to rest, to patience, to time, to consolidate new habits, and preserve new channels of normal nervous nutrition.

The topical effect of drugs and medication I need not dwell upon. In epilepsy from syphilis the iodide and mercurial preparations have been of value. The bromides, digitalis, atropine—all these are favored by those who seek to reduce the susceptibility of the medulla and allay erethism, if present, after the proximate cause has been removed. *Zinc* preparations have been useful in irritations of the pneumo-gastric, *iron* in cases after chlorosis, *quinine* in general anæmic states, and *electricity* in primary affections of the sympathetic system.

Dr. T. B. Lester, of Kansas City, Mo., said, the clinical advantages afforded the general practitioner, are not such as enable him to throw much light upon a subject so obscure as epilepsy. Yet the comparatively few facts which he finds in his limited field of observation are important in making up the great aggregate of facts which we may reasonably hope will one day dispel the darkness which enshrouds the true nature and proper treatment of this disease.

The paper of Dr. Pepper to which we have just listened with so much pleasure and profit makes a strong plea for a rational treatment based upon the constitutional peculiarity and special causation of each individual case.

This course of treatment cannot fail to commend itself to every one, save those who pertinaciously cling to some cherished plan of routine treatment.

There can be but little doubt that a large majority of the functional derangements of the nerve centres, which give rise to the pathological phenomena of epilepsy of every grade, have their origin outside these centres themselves. The great difficulty which lies in our way in treatment is two-fold—our inability in many cases to remove the primary cause—and to restore the reflex and convulsive centres to their normal degree of excitability. It is comparatively easy to comprehend how a purely local peripheral cause long continued by its irritation of the reflex centres may ultimately result in such histological changes, so that, should we succeed in removing the primary factor, the epileptic seizures would still continue to recur. Such is the familiar history of epilepsy, and it teaches us the important lesson to be vigilant in our search for every probable source of irritation, and prompt in removing it by every means in our power; for it is positively beyond our ability to estimate the measure of damage there is in delay.

It is probable that those cases dependent on some constitutional disease—with rare exceptions—are developed through the *intermediate* influence of the malnutrition which they produce, and not from any *direct* toxic influence which may be exerted upon the nerve centres, through the agency of retained excrementitious elements or other products of mal-assimilation. This, I think, may be predicated of those cases having their supposed origin in syphilis, lead-poisoning, frequently recurring hæmorrhages, renal insufficiency, or other defective elimination, chronic malarial infection, mental or corporeal exhaustion, etc.

Any constitutional disease which produces general anæmia is competent to develop an epileptic seizure, upon the supervention of slight peripheral irritation owing to the increased excitability of the reflex, and convulsive centres, which anæmia is well known to produce. If this be true, how futile must be our treatment, if alone directed to the sedation of this increased excitability. This end can be oftentimes accomplished by the influence of the potassium bromide, but the relief must at best be temporary and

partial unless conjoined with the use of such restoratives as iron, arsenic, cod liver oil, nutritious diet, good sanitary surroundings, and freedom from overtaxation of body and mind.

Diminished arterial blood pressure, with its concomitant increased venous blood pressure, is competent to establish the same, or similar condition of malnutrition, as the other constitutional conditions before mentioned. I have a case under treatment at this time, in which the first seizure occurred at the age of 64 years, without any assignable local or constitutional cause, except the enfeebled action of the heart, incident to the degeneration of old age.

In this case potassium bromide has been tried, with the effect only of lessening the frequency and severity of the attacks while the patient was kept strongly under its influence, but under the conjoined influence of the bromide digitalis, iron, and cod liver oil, the case has been much improved.

If the paper of Dr. Pepper does nothing more than to direct the attention of the profession, in a forcible manner as it does, to the importance of a just discrimination, between the varied constitutional conditions which exist in epilepsy, to the end that we may treat it more rationally, it will have accomplished great good.

J. J. Caldwell, M.D., of Baltimore asked what is Epilepsy?

Epilepsy a disease—or a deficiency—or a disturbance of the brain and nervous system, whereby there is a sudden and deadlike arterial anæmia of the brain and nerve centres.

These phenomena may indicate functional or organic lesions, hence the disease may be curable or incurable. As a proof of the above proposition I will cite the following theories, facts and authorities:

PONS AXIS.

No. 1.—Von Der Kolk on the Medulla.

No. 2.—Kroons on Asymetry of the Medulla Oblongata.

No. 3.—Solberg on the Cord.

No. 4—Lebut and Delasauve and Meynert on Ammonhorn.

No. 5.—Kussmaul on Ammonhorn.

No. 6.—Pathological Anatomy, Anæmia—Hyperæsthenia of the pons.

No. 7.—Traumatic lesions of brain and cord, electrical and toxic irritation of the same.

No. 8.—Sir Ashley Cooper's experiments on ligation and compression of the vessels.

No. 9.—Marshall Hall & Travers on excessive hæmorrhage in warm-blooded animals. Kussmaul & Tenner on the same.

No. 10.—Kussmaul & Tenner, experiments with watch glass on the brain and compression on the Carotids.

No. 11.—Landois on Venous Hyperæmia and Epileptiform Convulsions momentarily obliterating the superior Vena Cava in Rabbits.

No. 12.—Herman on the same, thus substantiating Von der Kolk's experiments.

No. 13.—Nothnagel on the roll played by the pons and medulla in general. Convulsions by irritating the brain with a needle to locate the true boundaries of the convulsive centre. Substantiates experiments of Schiff and Deiter.

No. 14.—The relation of lesions of the cord or of certain spinal nerves to epilepsy were noticed for the first time by Brown-Sequard who has cleared up the question and proves a hereditary sequence. Westphal has shown that in Guinea pigs, blows upon the head may immediately give rise to the production of epileptiform attacks.

No. 15.—Ferner's recent researches on Epileptiform shows convulsions may be produced upon the opposite side of the body by passing strong induced currents through one of the Cerebral hemispheres.

No. 16.—Bartholow's experiments upon a woman's brain by the induced current gave rise to epileptiform convulsions upon the opposite side of the body.

No. 17.—Brown-Sequard has shown contraction of the vessels of the "Cerebral-pia-mater in epileptic animals."

No. 18.—Travers, Nothnagel and others have shown that irritation of the peripheral nerves act in the same manner upon the cerebral vessels.

No. 19.—Dr. Howe on Epilepsy from sexual excesses.

No. 20.—Longet on irregular muscular action due to epilepsy demonstrating that the tuber anulare of the pons controls sensation and motion mandates.

No. 21.—Dr. Hammond on spasmodic contraction of the muscles, and his operations for clitordectomy.

No. 22.—Dr. Baker on effects of epilepsy on the genital organs.

No. 23.—Dr. Flint on case of female masturbation.

No. 24.—Baker Brown, of London, corroborates opinion of Dr. Flint.

No. 25.—Incurable case.

No. 26.—Terrier, Surrurier. Case of a soldier.

No. 27.—Zimmerman, case of convulsions produced by every emission.

No. 28.—Hospital records of ven. exceps.

No. 29.—Symptoms of epilepsy or falling sickness.

No. 30.—Duration of fit, etc.

No. 31.—Feigned epilepsy or malingering.

No. 32.—Epilepsy a nervous affection.

No. 33.—Ceasar, Bonaparte, Petrach.

A NEW METHOD OF TREATING ASIATIC CHOLERA PROPOSED.

BY S. S. TODD, M.D., PROFESSOR OF DISEASES OF WOMEN IN THE KANSAS CITY MEDICAL COLLEGE.

I do not wish in this brief paper to go into the subject of natural history of this disease. Thanks to modern investigation we seem to stand on pretty solid ground now with respect to its cause and mode of propagation, and the best means of warding off the disease, lessening the ratio of its mortality, and possibly its *fatality*—or the ratio of deaths to the number attacked.

More light is needed on the subject of the patholo-