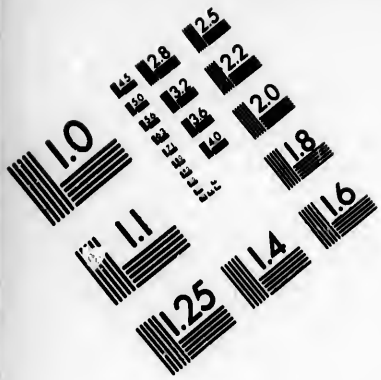

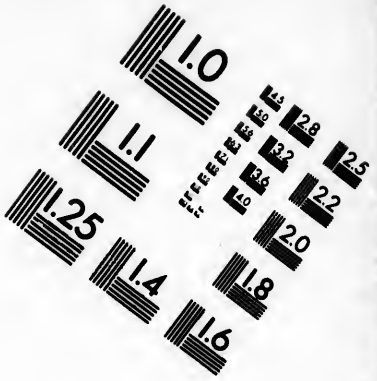

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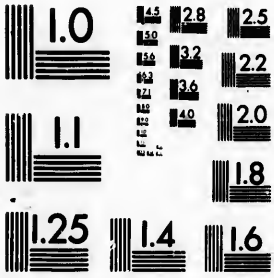

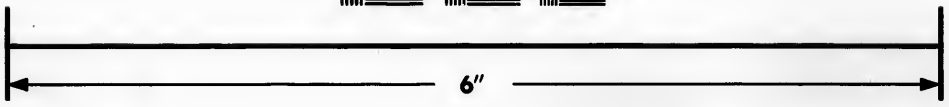

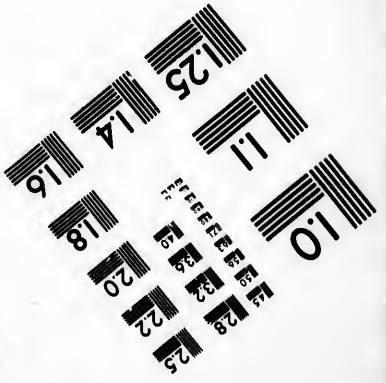

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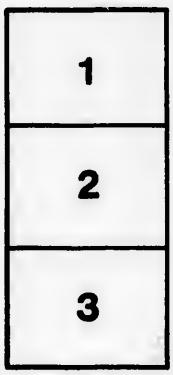

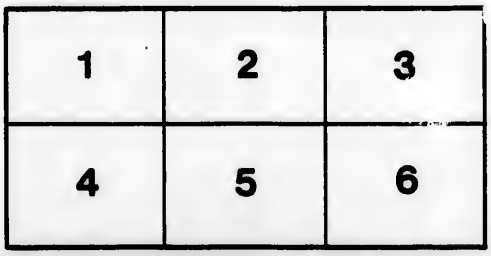

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A PAPER READ BEFORE THE MEDICO-CHIRURGICAL SOCIETY OF MONTREAL.

BY

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THE DIAGNOSIS AND TREATMENT OF EPILEPSY.

By James Stewart, M.D.,
Professor of Pharmacology and Therapeutics, McGill University.

(Read before the Medico-Chirurgical Society of Montreal.)

The diagnosis of epilepsy, as a rule, presents but little difficulty. It is, however, not unusual to meet with cases where very considerable trouble is found in concluding whether we have to do with true epilepsy or some allied affection. Hysteroid and motor disorders more frequently and closely simulate epileptic convulsions than any other affection. It is more frequent to find cases of epilepsy treated as hysteria than the converse. The following case, which presents many extremely interesting features, was diagnosed by more than one physician as hysteria.

A girl, when 12 years of age, began to complain of fits, which were variously diagnosed as epileptic and hysterical. Her father was an inebriate. Her mother and only sister suffered from neuralgia. There is a history of insanity on the mother's side. The "fits" begin nearly always with the expressions "Oh! I'm sick," or "I want my mother." "Take me home." For a period of two or three minutes she has a frightened look, but is not convulsed. She then falls into a deep sleep, lasting half an hour. She wakens up complaining of headache, and for an hour or two afterwards is, according to her mother's statement, "not quite herself." On one occasion she was requested to set the things for the tea-table, an hour after having a fit. She placed the plates on the table upside down. She generally passes urine during the fit, but has never bitten her tongue. She has no remembrance of what she says or what passes during

these " turns." On one occasion she had convulsive movements and turned over on her face. This fact itself is sufficient, I think, to establish the diagnosis that the " turns " are genuine epilepsy and not hysteria. It is certainly a matter of great importance to be able to draw a distinction between these two diseases.

The following case was also variously diagnosed as epilepsy and hysteria. A female, aged 25, with previous good health, two month after a severe mental distress, had a " fit," which she describes as a " stiffening of all her muscles." For the past year she has been troubled every two weeks with fits, which she characterizes as " severe" ones, and in addition, she has almost daily attacks of minor fits. The former are preceded by blindness and ringing in the ears, and followed by fits of " crying" and " laughing." During the attacks, which last about a minute, she is rigid. She has no remembrance of what passes during these fits. She bites her tongue and passes urine in the fit. In the slight fits she says that she has " strange feelings coming over her and then I loose consciousness, but only for a moment."

I think there can hardly be any doubt but what we have to do here with true epilepsy and not with hysteria. The hysteroid symptoms following the attacks do not exclude this. It is not uncommon to find such states in both sexes. The passing of urine and the tongue biting during the paroxysms are, however, sufficient to establish the diagnosis of epilepsy. The tonic character of the convulsions and the hysteroid state after they have passed away were, I have no doubt, the features in this case that led to the diagnosis of hysteria; and further, there was a history of those conditions which are so productive in giving rise to hysterical attacks.

It is not very rare to find genuine epileptic attacks alternating with hysterical attacks in females. Should such a patient happen to be observed only during the hysterical seizure, it is very likely that even a close observer would be misled, and would go on treating the case as one of pure hysteria.

If a patient, either male or female, bites the tongue and passes

urine involuntarily during a paroxysm, it may be set down as a certainty that we have to do with epilepsy. Patients who only have attacks of minor epilepsy followed by hysterical seizures present great difficulties for diagnosis, but nearly always, on close questioning, we are able to obtain a history of minor attacks without any post-epileptic hysterical seizures. If in the minor attack urine is passed involuntarily, the nature of the case is clear.

Unusual forms of megrim sometimes closely simulate attacks of minor epilepsy. The following case of ophthalmic migraine was mistaken by more than one observer for true epilepsy: — A married woman, aged 24, in the summer of 1888, had three attacks at intervals of a week, which began by blindness of the temporal half of each visual field. This lasted for one minute, and on one occasion the blindness extended completely over both fields. She felt a "peculiar numb sensation" spreading up the right arm; this was followed by a painful feeling in the right eyeball, which passed away in a few minutes. Altogether these attacks lasted about ten minutes, and were on every occasion followed by a state which she describes as "being foolish and saying and doing stupid things." She would use words wrongly, a condition of sensory aphasia. After taking Indian hemp she was not troubled again until a few weeks ago. During the past four weeks she has had six in all, closely resembling those she previously had, except that in place of a pain in the eyeball she has a compressed feeling in the head. It was the latter attacks that were mistaken for epilepsy. The resemblance is, indeed, close, but from the distinct history obtained of the previous attacks, there can be no doubt, I think, but what we have to do with ophthalmic migraine.

Ordinarily the migrainous paroxysm is so prolonged, even in the ophthalmic varieties, that the diagnosis is not a matter of difficulty, but in the case related the time was comparatively short. It should be remembered that we sometimes meet with migraine and epilepsy in the same subject, paroxysms of each taking place irregularly or more rarely alternately. Cases have been recorded where migraine has ceased after the development of idiopathic

epilepsy, but to reappear after the cessation of the epileptic fits. Such combinations and relations of these two diseases is a subject of great interest.

In the diagnosis of epilepsy, it is well to remember that loss of consciousness is not an essential part of the epileptic paroxysm. It is commonly believed that loss of consciousness always attends true epilepsy. In those text-books of medicine most popular at the present time, some state that loss of consciousness is an essential part of the fit, while others hold that it is not essential. In the vast majority of all cases, and in every case of severe epilepsy, there is loss of consciousness. A recent writer contends that loss of consciousness is an essential part, and that without it we have no epilepsy. In what are called abortive attacks of epilepsy it is very frequent to find consciousness present throughout. The following case must be taken as an example of genuine epilepsy, and yet consciousness in a certain number of fits was never lost.

A man aged 22, seen in 1885, had been subject to fits for some months. They were of two different varieties. (1) In one form he would suddenly begin to rub the palms of the hands against each other; these movements were continued for about one minute, and would cease as suddenly as they began. The patient would then proceed with the work he was engaged in, entirely unconscious of what he he had passed through. (2) In the other variety he was usually seized with flexion movements at the wrist and elbows, sometimes on the right side, sometimes on the left, and on a few occasions simultaneously in both upper extremities. During these muscular movements consciousness was maintained, never lost.

Now both of these attacks were epileptic in character. Cases illustrating this point might be easily multiplied, but that would serve no useful purpose. One undoubted case is sufficient to prove that loss of consciousness is not necessary to epilepsy. In the diagnosis, it is not enough to be satisfied with having determined that we have to do with epilepsy; we have to go deeper and find out what is the active factor in the case in question which has brought it about. Unfortunately, in the

majority of cases our investigations in this direction are fruitless; still, cases now and then are met with where the reward more than compensates us for our time and trouble.

It will be necessary, for the sake of clearness, before considering this subject, to take a survey of the nature of epilepsy. It is necessary, for intelligent treatment, to have a working hypothesis to go on. From the earliest times the nature of epilepsy has been a subject of the greatest interest. It is unnecessary to refer here to the numerous and often crude ideas that have been advanced in the past to explain its origin. Even now, after centuries of speculation and work, we cannot say that we know anything positive as to its true nature. Through the labors of that great medical philosopher Hughlings Jackson, and also the admirable work done in this connection by Gowers, we may consider it as proved that the epileptic paroxysm is dependent upon an instability of the nerve elements in the gray matter, which has as a consequence a proneness to discharge violently. The intimate changes that bring about this instability are unknown. In many cases the influence of an indirect heredity is marked—*i.e.*, an heredity to various functional nervous affections. Such a history is obtainable in 35 per cent. of cases.* A large number of epileptics are born, then, with this instability. The above proportion seems, after considering everything, small. It is so difficult in these cases to get a full and true history that we may be safe in saying that probably in 50 per cent. of all epileptics a neurotic history is obtainable, and we must consider the fact that these histories only refer to marked disturbances. The numerous silent but potent forces ever at work in the generation of being are beyond questionings. When we consider how few people live up to a high moral or physical state, the wonder is not that there are so many, but that there are so few born with unstable ganglion cells. Many epileptics would no doubt escape if their training and habits were not vicious. A slight instability becomes, as the results of bad training, a confirmed and marked instability. Some of the causes that lead to this, in a measure, acquired instability will now be shortly considered.

* Gowers, Diseases of the Nervous System.

First, as to the influence of *traumatism* in establishing ganglionic instability. It is only within a comparatively recent period that the influence of traumatism in establishing functional neuroses has been fully recognized. Its marked effects in bringing about hysteria has been specially insisted upon by the Charcot school. The profound and chronic neuroses following railway injuries are seen and recognized everywhere. That genuine epilepsy arises from traumatism of a general nature is also an undoubted fact. I do not mean traumatic epilepsy from injury of the brain or its coverings, but an instability originating as part of the general disturbance or shock attending falls, etc. The following case I look upon as an example of genuine epilepsy originating in this way:

A woman, now aged 43 years, enjoyed good health until two years ago, when she had her first fit. The fit followed twenty-four hours after a severe fall on the ice. Her nose bled freely at the time of the accident, but otherwise there was no wound of any kind, neither was there any loss of consciousness. Her family history is good. Mother died at 62 from dropsy; her father is 90, and in the enjoyment of good health; four sisters and four brothers all in good health. Her first fit, as mentioned, followed closely on the fall; the second four weeks after, and four months after the second she had seven fits within twenty-four hours, and did not recover consciousness between them—a true status epilepticus. Since then she had several fits at intervals of two and three weeks.

There can be no question that here we have a genuine epilepsy, and that it was in all probability induced by the severe commotion from which she suffered. There is not the slightest evidence of any internal injury, and there certainly is no necessity to assume any gross change to account for the epilepsy. From the literature of epilepsy it would be easy to multiply cases of this kind. Another form of traumatic epilepsy is one of very great importance. I refer to that which follows, and is due to, the cerebral concussion produced by the application of the forceps at birth. I have seen several cases where this was, in my opinion, the probable cause. A few months ago I saw a boy,

7½ years old, who had his first fit twenty-four hours after birth. He remained for three days in an epileptic state. Since that, fits have recurred at irregular intervals. He is the eldest of three children. Family history good. The delivery was accomplished with the forceps. In any one case it is difficult to prove that the concussion and compression of the brain caused by the forceps is the cause of the fits which follow, but the considerable number of cases where epilepsy has followed the application of the forceps leaves no room to doubt that it must be set down as one of the rarer exciting causes of this disease. This is a further warning to those who needlessly use these at times indis pensable instruments.

I wish now to refer to a way in which cerebral instability is brought about by the poisonous action of alcohol. That alcoholism is able to bring on true epilepsy is beyond doubt. I have met with two cases of undoubted alcoholic epilepsy. In one, the patient became a confirmed drinker after his 30th year. His first epileptic fit occurred after a three days spree; he had given up everything in order that he might drink. Now this patient at times was able to keep sober for weeks and even months, but he never had any fits except when he went to great excess, and then he almost invariably had them. Now this patient did not drink because he was an epileptic, but he became epileptic from the action of alcohol in destroying the central zone of his higher nervous mechanisms. In the other case the patient had two distinct attacks of the status epilepticus following two prolonged bouts of drinking. One occurred in 1884 and the other during the present year. In the intervals he was perfectly free. This case is a very striking example of the influence of alcohol in bringing about that instability of the cerebral gray matter which is at the bottom of epilepsy. How alcohol induces these changes is unknown. From the history of the cases reported, and from others also, it is shown that alcohol seldom induces permanent instability if the habit of drinking is given up. I do not include here that numerous class of cases where during infancy convulsions have been present, and where they have developed into confirmed epilepsy as the result of alcoholism afterwards. In

the cases reported both patients were over 30 years of age, and both had always enjoyed good health, never having had convulsions during infancy. We have to do with an acute dissolution of the centres and not with a permanent disability. No doubt if the habit of drinking is continued at short intervals for a lengthened period the centres become permanently unstable, and then we have a state not different from that we find in ordinary epileptic states.

The Treatment.

The first and most important indication to fulfill in the treatment of epilepsy is to endeavor to diminish that instability of the cerebral cells which in all confirmed cases of this disease is present to a greater or less extent. It is especially necessary that this should be before us while the disease is not as yet confirmed. By diligent attention to this indication there is no doubt that many children might be saved from becoming epileptics. In from 10 to 15 per cent. of epileptics the fits have begun in infancy, and there can be no question that careful treatment of the recurring convulsions of childhood might greatly diminish this proportion. To notice the apathy and indifference of patients as I have several times in such cases is distressing. A very great responsibility devolves on the medical attendant in these cases. The time for treatment is before the cells have acquired the vicious habit of unduly discharging. The influence of habit as related to the cells of the cerebral cortex is of paramount importance in the treatment of epilepsy. Nervous tissue, above all other tissues, is prone to easily acquire and retain organic memories. Tissues are resistive to disease in proportion to their maturity, and as the tissues of the cerebral cortex are the last of all to reach complete functional power, it follows that they are longer exposed to injurious influences. This is the reason why epilepsy may not show itself for a number of years. It is comparatively rare for epilepsy to originate after the 30th year. Fully 90 per cent. of cases begin before the 25th year. This period may be taken as about the time when the cells of the cortex have reached their maturity. The slow development of

the cerebral cortex in its functional parts at least has an important bearing on the treatment of its disordered states. Dr. Clouston says*: "The unique fact about the nerve cell is the extreme slowness with which it develops after its full bulk has been attained. In this it differs from any and every other tissue. We may say that after most of the nerve cells of the brain have attained their proper shape and full size, it takes the enormous time of 18 or 19 years to attain such functional perfection as they are to arrive at. This striking fact of such extreme slowness of development of complete function no doubt shows, as no other fact could, the complexity and importance of the functions which the brain tissue subserve. . . . . It is during this long period of gradual coming to perfection of the nerve cell that its hereditary influences for good or for evil come most into visible play. No doubt they exist before, but as yet we have no certain means of detecting them."

If we are to attack epilepsy successfully, we must go to the root of the trouble, and that is the unstable nerve cells. Really the foundation treatment of this disease is nothing more or less than an education of the cerebral cells.

Now we must for a moment consider the means through which the metabolism of these structures is carried on. The way in which they receive impressions is two-fold—(1) Through the blood, and (2) through the countless thousands upon thousands of afferent tracts which connects the cortex with the entire peripheral system. The endless changes taking place in the world about us are being constantly felt, and are ever constantly influencing for good or for evil our higher centres. There is no doubt that the functional power of the cerebral cells is more influenced by the sense impressions than it is by the chemical changes carried on through the influence of the blood. Both, of course, are necessary. Advantage should be taken of the preponderating influence of sense impressions on cerebral metabolism.

For some time I have been convinced of the importance of removing a young epileptic patient from his old surroundings.

* Morison Lectures, Edinburgh Medical Journal, January, 1891.

What can be worse for such a patient than to be brought into constant contact with those who are probably of the same nervous organization as himself? I had a case illustrating this in a very striking manner. A girl aged 12, whose mother, although healthy, was an excitable, irresolute, nervous woman, had recurring epileptic attacks for several years. Owing to an accidental circumstance the child was away from home for upwards of a year, and during the latter ten months of that period she was free from fits. On returning home they recurred with greater frequency than before. The child, of her own accord, desired to go back where she had been visiting, and with the result that she again improved. Now although this is only one case, it teaches us something useful—viz., in the treatment of these cases the environment of the patient is of great importance. It can be used as a means of educating the nerve centres. The many ways in which the environment of the patient can be influenced, and through it the metabolism of the cerebral centres, are too numerous to refer to. I will here only mention one other. It is well known that certain people have one special sense developed inordinately—developed at the expense of the other senses. In some, for instance, the hearing is acute, while the sight is dull. As frequently the converse is true. The nearer the different sense centres in the brain come to the physiological norme, the less likely are we to have unbalanced mechanism. When certain areas are inordinately acute, we have an unbalanced state; and by careful training I have no doubt much might be done in the direction of bringing about a more perfectly balanced afferent system. It is not my intention to deal to-night with the connection of ocular defects and certain ovarian and uterine operations as methods of treating epilepsy.

Ever since the treatment of epilepsy by the different bromides has become the general practice, it has become with most of us a mere matter of routine to give these drugs day after day and month after month, paying but little heed to the many other ways which, individually, are not of great moment, but when taken together are of the greatest possible importance. It will no doubt appear to many to be very heterodox to question the great

utility of the bromide treatment. That it does good in many cases, that in some cases it is indispensable I freely grant; but I as firmly believe that it does harm sometimes, and that it can be entirely dispensed with to the advantage of the patient in certain cases. I will first refer to the pharmacology of the drug. How does pot. bromide act in epilepsy? Some still maintain that it is through the blood-vessels, but there is no foundation whatever for that view. The experiments of Albertini have, I think, proved conclusively that it acts simply by depressing the motor activity of the cerebral cortex. Everything that we know of the action of the drug supports the experimental investigations referred to. Now there can be no question that an agent that depresses the reflex activity of the nerve centres cannot fulfill the first and most important indication in the treatment of epilepsy, *i.e.*, increasing the stability of the nerve centres. To neglect this indication is to neglect the first step in the proper treatment. Where the bromides are specially called for and operative are those frequently recurring attacks, say one or two weekly. Here we can with these agents break the habit—in other words, lessen the unconscious memory of the disease. The dose and mode of administration of the bromides is a matter of some importance. As to the quantity necessary, this, as is well known, varies greatly. As a rule, it is seldom advisable to order more than one drachm in the twenty-four hours; the smallest quantity sufficient to break the habit is the best. Formerly I followed the usual practice of giving the bromides after meals, but being influenced by the writings of Seguin, who strongly recommends that it be administered before meals in a weak alkaline water, I followed this practice for some time, but some recent experiences have taught me to rely on the usual method of administration after meals. The only gain in giving an agent on an empty stomach is that it enters the blood more rapidly. Now in these cases there is no necessity for this rapid absorption, and further, I feel satisfied that it is much more apt to disorder the functions of the stomach when given before meals.

Another important point is the frequency of administration. The object aimed at is to keep a sufficient quantity of the drug

in the circulation at all times to keep up the depressant action on the nerve centres. If the fits recur only during the night or morning, one dose in the twenty-four hours of ss or ʒi is all that is necessary. If they come at irregular intervals in the day it is well to give the bromide twice in the twenty-four hours—after the evening meal and after breakfast. As the elimination is not very rapid, being seldom complete even within twenty-four hours, we may feel assured that two doses in the twenty-four are all sufficient in any case. The elimination is, of course, more rapid during the day than during the night. It is never necessary to administer it three times in the twenty-four hours. If given too frequently we are certain soon to have bromism with its disagreeable consequences. We have a cumulative action; *i.e.*, more is taken up than can be eliminated. To prevent the disagreeable effects of a bromide on the skin, we have in arsenic a very efficient remedy.

As already mentioned, there is a class of cases of epilepsy better treated without bromides. It is that class where the fits recur at considerable intervals, say once every three or four months, or even less frequently. It has been well, and we believe truly, said by Broadbent*: "The epileptic who enjoys a certain degree of immunity from attacks in virtue of large doses of bromide is on a lower platform than one who obtains such immunity by means which raise the tone of his nervous system, and the immunity may be purchased too dearly, not to speak of the disfigurement produced by the bromide rash, or of the derangement of digestion, or of the anæmia, which are common results. I have seen patients reduced to a condition scarcely distinguishable from general paralysis by the bromides, and all minor degrees of intellectual and nervous debasement, from which condition they have been rescued by discontinuing the drug and adopting a different treatment, the fits in several instances also ceasing. The legitimate use of bromides I conceive to be for the purpose of palliation, of staving off attacks which have become too frequent, and so of gaining time for a study of the peculiarities of the case, its true causation and ultimate pathology, and for the application of remedial measures which

* The Pulse, p. 299.

shall go to the root of the disease. When the fits are separated by months, I can see nothing but harm in giving bromides regularly. If, in such a case, an exciting cause can be identified, bromides can be usefully employed to parry its influence and avert the attack; but no such identification is possible if the bromides are being taken constantly. Only by careful inquiry into the antecedent circumstances of each fit can this be done, and attention to the administration of the drug take the place of attention to other matters."

A great many other agents besides the bromides have been used with alleged success in the treatment of epilepsy. I have had very little experience with any of them. Borax in several cases failed utterly. There are good reasons for believing that a combination of a bromide with digitalis may be more effective in certain cases than when the bromide is given alone. Gowers considers that this combination is especially valuable in night epilepsies. It is altogether likely that the action of digitalis for good is effected through the circulation. It is not uncommon to find the epilepsies of adolescence to be attended with a lowered blood-pressure, and from what we know of the action of digitalis on the central and peripheral nervous system it appears very probable that its action is through the circulation and not through the nervous system. A combination of the bromides with atropine is said sometimes to succeed. On physiological grounds I should require positive proof of this statement before resorting to this treatment. Atropine has a stimulating action on the cerebral cortex in both the lower animals and man, and the combination of it with a bromide is, as far as I can see, the union of agents pharmacologically incompatible. I have had no experience of the treatment of epilepsy with nitro-glycerine or simulo.

Long ago it was the custom to recommend epileptics to abstain from nitrogenous food. A recent writer (Dr. Jno. Ferguson) has with great ingenuity insisted on the importance of attending to this. He even goes so far as to say that the treatment of epilepsy is mainly dietetic. This is, however, a very partial view of this great and deep subject. The diet should, of course, be carefully attended to, but not mainly in the direction referred

to. As a matter of fact, experience does not confirm the statement that nitrogenous food is injurious. That it may even be very necessary is more than probable in the great number of cases of essential epilepsy during adolescence, for it is here that we meet with the cases attended with low blood-pressure. For such cases a nitrogenous diet is certainly indicated on physiological grounds, and I believe experience confirms this. In cases of senile epilepsy, it is not uncommon to find high tension, and here it may be very necessary to curtail or deprive the patient altogether of nitrogenous food.

In the present paper I do not touch on the subject of epilepsies from gross cerebral changes. I have met with only one case where surgical measures were of great benefit. Two years ago Dr. Roddick operated on a case in the Montreal General Hospital with very gratifying results.

Although the treatment of epilepsy with drugs and modification of the diet in certain cases is of great importance, neither are of prime importance. The foundation treatment of epilepsy is certainly not a drug treatment, neither is it mainly a dietetic treatment. It consists essentially in the proper training of the ganglion cells—it is, in the broad sense of that term, an education of the ganglion cells and attention to all those general measures which conduce to the proper performance of function in every organ and tissue of the body.

In his description of the functions of the cerebral convolutions, Wesley Mills says*: "The quality or functional capacity of the individual elements, especially of the cortical cells, both as the result of innate, inherited powers, and as altered by education, is, of course, a matter of great importance. By education we mean all those influences that have been brought to bear npon these cells from without, of whatever kind. Apart, too, from all these considerations, it must be clear that what any set of cells can accomplish, be they brain cells or other, must depend largely upon their capacity to appropriate nourishment, which in turn will be modified by blood supply, the behaviour of excreting organs, etc. The brain and other parts are so mutu-

* Text-book of Animal Physiology.

ally dependant that they cannot be understood by any isolated consideration of the one or the other. It is not to be supposed that an individual with a poor respiratory, circulatory and digestive system, no matter what the possibilities of his cerebrum, can ever rank with an organism admirably balanced in these respects."

Those broad general principles so ably enunciated in that quotation are especially applicable to the treatment of the neuroses.

It may be considered by some present that I have wandered too far into the realms of psychology in the treatment of epilepsy, but let me remind any that may think so that our present knowledge of this disease has been mainly acquired through the studies of the cerebral functions. The education of the nervous system is a subject of the greatest importance to every practising physician, and it is only by an attentive study of each individual case in all its details by the physician that a proper system of education can be carried out. It is the physician's duty to be able to direct the particular ways in which the education of his patient should be carried on.

For every case requires its own particular training. "One of the great advances in modern education is that it recognizes that all are not cast in the same mould, and therefore cannot rationally be subjected to exactly the same kind of training; that what is suited to one is utterly unfitted to another, and that it is more than folly to submit all to one rigid, fixed mode of treatment."*

By following these lines of treatment more cases than at present will be prevented from drifting into hopeless chronicity and incurability.

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* Campbell: Flushing and Morbid Blushing, p. 217.