

produced by a beginning aneurismal dilatation, before it is large enough to press upon surrounding parts. By their involvement one would explain the acute pain produced by the lodgement of an embolus in a peripheral artery.

PHLEBO-SCLEROSIS.

Of late years evidence has accumulated which goes to show that a similar lesion exists in the veins. A paper, by Sack⁴⁴ is instructive as showing its relation to the lesion in the arteries. It is shown that the two sets of vessels are likely to be simultaneously affected in the same individual, and the lesion in the veins probably does not differ in etiology from the arterio-sclerosis.

According to Sack, phlebo-sclerosis occurs most commonly in the lower extremities, a fact which is also true of the arterial lesion. He ascribes its frequency in this location to the sluggishness of the circulation here and to variations in blood-pressure. Not only in general, from their dependent position, is the circulation less active in the lower extremities than elsewhere, but there are likely to occur local causes of congestion in them, such as ascites, pregnancy, heart disease, etc.

The nodose form does not exist in the veins. When disseminated patches are found in them, it is likely that they represent the beginning of the diffuse form.

Pokrovsky⁴⁵ has also published an elaborate paper on phlebo-sclerosis. He claims that there is a nodose form of the affection, and that many consecutive changes and degenerative processes follow and accompany it. Spillmann,⁴⁶ of Nancy, has written lately on the same subject. He quotes extensively from a thesis by Henry Thiebaut,⁴⁷ who seems to have devoted much time and attention to this subject.

Although numerous writers concur as to its existence and distribution, no one seems yet to have been able to trace to it any grave consequences. Similar lesion has been also found in the capillaries. Here the disturbance of function produced manifests itself by an increased permeability of their walls, in consequence of which edema of the tissues result.

The process seems to be so widely spread throughout the vascular system as to justify the name angio-sclerosis which several observers have applied to it.

ON THE ETIOLOGY OF EPILEPSY, WITH SPECIAL REFERENCE TO THE CONNECTION BETWEEN EPILEPSY AND INFANTILE CONVULSIONS.¹

BY G. L. WALTON, M.D.,

Physician to the Neurological Department of the Massachusetts General Hospital,

AND C. F. CARTER, M.D.,

Assistant Physician to the Neurological Department of the Massachusetts General Hospital.

BEFORE considering the relation between infantile convulsions and epilepsy, it will be well to fix in our minds the true meaning of the various expressions which denote increased muscular action, as even the fundamental matter of definition furnishes confusing inconsistencies in the works of the most recent author-

ities, in which we not infrequently find the terms contraction and contracture, spasm and convulsion, used almost interchangeably.

Contraction, or drawing together, implies in itself nothing pathological, but is a general term applied to the shortening of any tissue, though most commonly used regarding muscles; this process may be either physiological or pathological.

Contracture is a more or less permanent contraction.

Spasm is an involuntary contraction of muscular tissue, and may be tonic or clonic.

Convulsion is a generalized spasm, either tonic, clonic, or both, and is likely to be accompanied by loss of consciousness and other manifestations, though not necessarily so.

All these terms are used to describe symptoms, whereas *epilepsy* is an idiopathic disease, that is, "a disease conceived by itself, which is distinguished from eccentric convulsion, from the spasms of poison, from the convulsions that result from organic changes in the cerebro-spinal centres, and from every other form of known and recognized disturbance. It is certainly true, that an irritation of the foot, in the digestive tract, or in the convolutions of the brain, may cause, not only simple convulsions but also epilepsy proper, still, the difference should be recognized. Convulsions imply a condition of increased irritability; in epilepsy this irritability has an existence of its own, depending upon a faulty nourishment which exists after the eccentric irritation has been removed." (Reynolds.)

The distinction so clearly enunciated many years ago seems to have been curiously overlooked by some of the most recent writers, whose efforts at classification lead the reader into numberless contradictions, and retard, rather than advance, our knowledge of the subject.

In illustration we will only quote Hamilton, who, in Wood's "Hand Book," after defining epilepsy as a disease in which sudden losses of consciousness are attended by more or less convulsive muscular action, proceeds to describe a case of defective mental development with malformation of the skull, as an epileptic, and to include under the causes of epilepsy meningeal or osseous thickening, exostoses, vascular dilatation and sclerosis-atrophy or hypertrophy, and the existence of tumors, all varieties of fracture and depression, and subdural cysts, also protracted hemorrhage and metallic poison; the convulsions following sunstroke, however, with curious inconsistency, he regards as hardly true epilepsy. This author even gives the reader to understand (page 706) that the convulsive seizures of general paralysis should be classed under epilepsy, with accompanying dementia. Thus, under the morbid anatomy and pathology of *epilepsy*, he states that "in cases where dementia or other mental troubles are found, and especially in those in which the mental disturbance takes the form of general paresis, we may expect to find a well-diffused cortical sclerosis."

Again, while he states that uræmic and alcoholic convulsions should be differentiated from epilepsy, he includes metallic poison under its etiology — a distinction hard to understand.

In clear-cut contradistinction to these confusing statements, Westphal has said, "Not every patient with mental or nervous trouble, who has epileptoid or epileptiform attacks, should be classed as an epileptic."

In epilepsy it may fairly be assumed that an exalted

¹ Read before the Boston Society for Medical Improvement, May 25, 1891.

⁴⁴ Virchow's Archiv, cxii, 1888, p. 403.

⁴⁵ St. Petersburg Inaugural Dissertation, 1890; and London Medical Recorder, 1890, p. 302.

⁴⁶ Gazette de Med. et de Chir., 1890, p. 480.

⁴⁷ Thèse de Nancy, 1890.

degree of irritability exists in certain motor centres. Whether those of a special convulsive area in the pons or medulla, as held for example by Küssmaul and Tennen, Brown-Sequard, Schiff and Nothnagel, or in the cerebral cortex, as held by Ferrier, Luciani, Bartholow, Unverricht, Munk, Bubnow, Heidenheim and Horsley,² the writers will not at present commit themselves to an opinion. This irritability renders liable a general convulsive discharge on slight provocation, or even, at intervals, spontaneously. It is this underlying condition which constitutes the *disease*, the *convulsion* being only a symptom, nor is the convulsion even an essential symptom—as is evidenced by petit mal—the loss of consciousness being far more constant.

Writers are practically unanimous in ascribing to heredity the chief place in the etiology of epilepsy, mentioning not only the neuroses and allied neurotic temperament, as well as epilepsy proper in the ancestry, but also apoplexy and other organic affections of the central nervous system, which depend in no degree on a neuropathic taint. It is hard to see how the latter can play a very conspicuous part in the causation of epilepsy, in the progeny of the sufferer, however liable they may be to produce convulsions in his own case; and even regarding the neuroses, the investigations of the writers would lead them to the belief that even this connection has been overestimated. Migraine, for example, has been somewhat extensively regarded as analogous to epilepsy, while in point of fact their only similarity lies in a spasmodic recurrence with intervals of freedom. That one can pass into the other, or beget a tendency to the other in the progeny, is a matter of great doubt.

Wilks,³ dissenting from the prevailing view on this point, states that he has only known of one case where the two affections co-existed, and has never known the one disease to pass or develop into the other. He alludes to the remarkable exemption of epileptics from headache, and dwells on the fact that remedies act far differently on the two, for example, antipyrine and the bromides.

Again, Lamoine⁴ in discussing the etiology of epilepsy, is inclined even to deny the inheritance of the disease, and allows a secondary part to hereditary neurotic predisposition.

The discussion of this point may be left to a future investigation, and it is only mentioned here in passing, to show that the long-established views on the simple question of heredity are not universally accepted.

Coming to the question with which we are chiefly concerned in this paper, namely, the connection between infantile convulsions and epilepsy, a study of the authorities shows an almost unanimous expression of opinion that the former predisposes to the latter, a position which the writers consider untenable.

In infancy we have physiologically to do with an irritability analogous, perhaps, to that already mentioned as characteristic of epilepsy, so that convulsions are brought about by sources of irritation either inactive in the adult, or producing a different train of nervous symptoms less explosive in character. This irritability is, however, during the period of early development, physiological in the majority of cases, not pathological, and we should be chary of pronouncing

the resulting convulsions epileptic. This does not, of course, preclude the possibility that we have to do with one of the exceptional cases where convulsions mean epilepsy, beginning in childhood, a question which can only be determined by their continuance and nature. We have no more right to associate with epilepsy infantile convulsions arising upon intestinal or other irritation, through the agency of this physiological irritability, than we have to regard the regurgitation of food at this age as due to a pathological state of the vomiting centre. Indeed, the feverish condition which usually precedes the convulsions of infancy is in marked contrast to the condition preceding a true epileptic attack.

Such a course of reasoning as this will lead us to view the cases, as they present themselves, free from bias, and should prevent our acceptance of isolated cases of coincidence as proofs of an underlying principle, an error into which we are liable to fall if we accept blindly the dictum of tradition.

This subject should not be regarded as one of simply scientific interest, for we are forced in practice to express definite views upon it, and the realization that our answer may remove or deepen, as the case may be, a warrantable anxiety on the part of parents and friends, will lead us to consider carefully all evidence bearing upon the point at issue.

Let us consider, first, the age of commencement of true epilepsy. Statistics will naturally vary somewhat, but our observations agree in a general way with those previously published, at least to the extent of showing that true epilepsy *may begin* in infancy, and become continuous, a fact which will prevent our going to the opposite extreme and assuring the parent that there is no liability that the convulsions will continue.

Starr⁵ has formulated the following table.

AGE OF ONSET.	
Birth to 5 years	29 cases.
5 to 10 years	22 "
10 to 15 "	30 "
15 to 20 "	16 "
20 to 30 "	30 "
30 to 40 "	9 "
40 to 50 "	8 "
50 to 60 "	3 "
Age unknown	18 "
Total	165

Moreau's tabulation,⁶ of 995 cases is as follows:

At birth	87 cases.
Infancy	25 "
2 to 10 years	281 "
10 to 20 "	361 "
20 to 30 "	111 "
30 to 40 "	59 "
40 to 50 "	51 "
50 to 60 "	13 "
60 to 70 "	4 "
Total	995

According to Starr, therefore, 59 per cent. began before the age of 20 and according to Moreau 76 per cent.

Our own tabulation of 133 cases, treated during the last three years at the Massachusetts General Hospital, is given in the next column.

Percentage up to the age of 20=58. A comparison of our percentages with those of Starr shows a marked similarity.

The 133 cases mentioned by the writers, occurred

² For theories regarding pathology, with bibliography, see Prize Essay of Hare. (F. A. Davis, 1890.)
³ Lancet, August 11, 1888. ⁴ Le Progrès Médical, April 23, 1888.

⁵ Familiar Forms of Nervous Disease, New York, 1890, p. 256.
⁶ Mem. de l'Acad. de Méd., Paris, 1853.

amongst 2,539 cases of all varieties applying for treatment at the Neurological Department of the hospital, making a percentage of $5\frac{1}{4}$, a proportion slightly lower than the estimate of Reynolds (about 7 per cent. of all cases of nervous diseases), also than that of Putzel (a little over $6\frac{3}{4}$ per cent.).

The lower percentages found by the writers, unless possibly accidental, are probably due to care in limiting the diagnosis.

AGE OF ONSET.			
Birth to 5 years	.	.	20 cases.
5 to 10 years	.	.	18 "
10 to 15 "	.	.	20 "
15 to 20 "	.	.	19 "
20 to 30 "	.	.	35 "
30 to 40 "	.	.	12 "
40 to 50 "	.	.	3 "
50 to 65 "	.	.	4 "
Unknown	.	.	2 "
Total			133
		Starr's.	Writers'.
Birth to 5 years	.	17.67%	15.00%
5 to 10 years	.	13.33	13.50
10 to 15 "	.	18.17	15.00
15 to 20 "	.	9.75	14.33
20 to 30 "	.	18.17	26.33
30 to 40 "	.	5.33	9.00
40 to 50 "	.	4.83	2.25
50 to 65 "	.	1.83	3.00
Unknown	.	10.92	1.50

On reviewing the literature concerning infantile convulsions as a predisposing factor in epilepsy, one finds a prevailing, though not unanimous, affirmative opinion.

Neimeyer says: "It (epilepsy) never rarely, in the first years or months of life; one finds, also, sometimes, that patients in whom the disease first declared itself later, suffered already in early childhood from convulsions."

Webber:⁷ "Severe and prolonged convulsions in infancy are very frequently followed by epilepsy later in life."

Starr⁸ states that "infantile convulsions are the most common of all predisposing causes of this disease."

Nothnagel:⁹ "In hereditary epilepsy the attacks are apt to begin before the twentieth year. Such patients suffered from eclampsia often in first dentition and remain apparently healthy until the outbreak of epilepsy."

Althaus:¹⁰ "Where epilepsy is hereditary, the infant is liable to attacks of eclampsia, and true epilepsy is developed about, or at any time previous to puberty."

Among other authors expressing the same opinion, may be mentioned Rosenthal,¹¹ Hamilton,¹² Bastian,¹³ Brown-Sequard,¹⁴ Seguin and Ranney.¹⁵

A certain degree of dissent from the prevailing view on this point has been already expressed.

Putzel¹⁶ says, for example: "We not infrequently notice that epileptics have suffered from eclamptic attacks during the period of dentition, and some authors believe that frequently recurring eclamptic convulsions may produce an epileptic habit in the brain, and thus give rise to the independent existence of epilepsy. But the eclampsia of childhood is so overwhelmingly more frequent than epilepsy that we are very sceptical

with regard to its efficiency as a cause of the latter affection."

Hammond:¹⁷ "They (infantile convulsions) may pass into epilepsy; but if they do not, I have never been able to find a single instance in my experience in which epilepsy ensuing in adult life has been preceded by the ordinary infantile convulsions." This experience would point to the fact that infantile convulsions once discontinued, no fear need be expressed that epilepsy will ensue later, a view which coincides with our own, though our experience is not identical with that of Dr. Hammond, that *no* case of infantile convulsions has been found to precede epilepsy, our observations showing, however, no more such cases than can be explained by coincidence, as our tables will show.

Eustace Smith¹⁸ says: "As a rule, single fits, or convulsions occurring without other signs of nerve-lesion in a healthy child, are purely reflex, and have no gravity whatever."

Flint:¹⁹ "A paroxysm of convulsions in an infant or child often, with good reason, occasions anxiety lest it may prove the beginning of epilepsy. It is not always practicable to determine the point at once. There is more reason to apprehend epilepsy in proportion as the convulsions are not otherwise to be accounted for. Their non-recurrence affords the only positive proof that they are not epileptic."

From an *a priori* point of view it might be assumed that the children most liable to have convulsions on a given irritation, possessed a greater convulsive tendency than others, and might therefore be expected to offer a somewhat more probable basis than others, for future attacks. When we come to consider, however, the vast and vital step from simple reflex convulsions to true epilepsy, this argument will not weigh very heavily, against facts. It is to these facts, as far as come under our experience, to which your attention is called.

We have carefully analyzed 70 successive cases of pure epilepsy in hospital and private practice with reference to the question of previous infantile convulsions, the opportunity being also taken to find out whether the brothers and sisters have been subject to them. The first point was to discover the relative tendency for the two affections to fall together or separately in the same family. The number could have been made much larger, but care has been taken to exclude cases in which trauma or organic brain disease was likely to have played a part, as well as those in which uncertainty existed regarding the early history. We are almost of necessity restricted in this investigation to those cases whose parents we are able to question, as few persons, well or otherwise, can be depended upon to state whether they themselves or their brothers and sisters had convulsions in infancy. Of these 70 cases, nine commenced in infancy and became continuous; 56 had no history of convulsions in childhood, five only had a history of infantile convulsions followed by a period of immunity.

Of these five cases the history was as follows:

CASE I. Typical epileptic. An infantile convulsion occurred at the age of one year, nothing further appearing until the age of sixteen, when true epilepsy began.

¹⁷ Diseases of the Nervous System, 1881, p. 702.

¹⁸ Quain's Dictionary of Medicine, p. 695.

¹⁹ Practice of Medicine, Philadelphia, 1873, p. 754.

⁷ Nervous Diseases, New York, 1885, p. 348.

⁸ Loc. cit., p. 256.

⁹ Ziemssen.

¹⁰ Dictionary of Nervous System, New York, 1878, p. 247.

¹¹ Loc. cit., 1879, Trans., New York, p. 339.

¹² Wood's Hand-book of the Medical Sciences.

¹³ Quain's Dictionary of Medicine, p. 303.

¹⁴ Loc. cit., p. 450.

¹⁵ Lecture on Nervous Disease, 1888, p. 473.

¹⁶ Functional Nervous Disease, New York, 1880, p. 68.

CASE II. Typical epileptic. Had a single convulsion at the age of two. Epilepsy commenced at the age of twelve.

CASE III. Petit mal. Had four infantile convulsions between the ages of one and four. Present trouble began at eleven.

CASE IV. Typical epileptic. Had infantile convulsions. Epilepsy appeared at twenty-seven.

CASE V. Typical epileptic. Had three infantile convulsions in one year. Epilepsy began at fourteen.

Apart from these five cases there were none, among the seventy cases investigated, in which infantile convulsions had occurred, to disappear and be replaced by epilepsy, though, as stated, in nine the epilepsy came on in infancy and remained constant. These nine cases alone would show the possibility of epilepsy beginning at an early age, a fact already established, and should put us on our guard against assuring the parent of a child with its first infantile convulsion, that epilepsy is not to be considered at all. Such convulsions having, however, once disappeared, it would seem that we may reassure them with confidence, for the proportion of five in seventy (one in fourteen), is too small to be regarded as any more than a coincidence.

Curiously enough, the investigation of the *other children* (grown-up) in the families of epileptics has shown a much larger proportion of infantile convulsions among them than among the epileptics themselves, a fact which may possibly show an abnormal convulsive tendency in such families, but illustrates in a striking manner the absence of interdependence between the two affections. J. Lewis Smith, in support of the same view, even states that he has been struck by the *immunity* from infantile convulsions among epileptics, though we hardly imagine that the word immunity was meant to be taken too literally.

Various estimates of the percentage of epileptics in a community range around six in one thousand, no estimate being much higher than this. No statistics previously existing as to how many cases of infantile convulsions occur in a community, the writers have collected data on this point with the following result: Out of one thousand children taken consecutively at random from all classes of society, 111 have been found to have a history of infantile convulsions. Now, taking the percentage we find of infantile convulsions among epileptics (one in fourteen), we have the two affections occurring coincidentally once in two thousand individuals. When we consider how many cases, therefore, of infantile convulsions occur in every two thousand individuals by our statistics, (approximately 220,) it is at once evident how rarely the one condition is followed by the other. This would reduce the chances of a given child with infantile convulsions becoming an epileptic later in life, to something like one in 220, certainly hardly sufficient to warrant alarm, even allowing a balance for difference in statistics, for one in 220 is certainly no more than the average chance of ordinary individuals if six in one thousand is the usual rate. It would certainly seem that the proportion of one in fourteen as compared to one in ten, would place us on the safe side in saying that epileptics are at least no more liable than others to have had infantile convulsions, and conversely, that a child suffering from infantile convulsions is no more likely than any other to suffer from epilepsy later in life, after a period of immunity has removed the case from the

class of epileptics beginning in infancy and becoming continuous.²⁰

CONCLUSIONS.

(1) Epilepsy may begin in infancy and become continuous.

(2) Where infantile convulsions have ceased for a sufficient time to remove the case from the class mentioned under Conclusion I, the child is no more likely to become an epileptic than any other individual.

A CASE OF INTESTINAL ANASTOMOSIS, BY MEANS OF SENN'S PLATES, FOR THE RELIEF OF COMPLETE CHRONIC OBSTRUCTION CAUSED BY INVAGINATION AND SLOUGHING OF THE UPPER PART OF THE ILEUM. RECOVERY.¹

BY JOHN HOMANS, M.D.,

Surgeon to the Massachusetts General Hospital, Boston, Mass.

ABOUT the first of December, 1890, Dr. F. W. Chapin, of Springfield, Mass., wrote me in regard to operating on a case of intestinal obstruction. A day or two later he wrote that consultation had decided that the child was too weak to bear any manipulation. On December 21st, he wrote the accompanying letter:

SPRINGFIELD, MASS., December 21, 1890.

MY DEAR DOCTOR:—The patient, a girl ten years old, about whom I sent you a note the other day is alive and perhaps in as good condition as when I wrote. The history of the case is somewhat peculiar. Last July she was suddenly taken with severe vomiting and symptoms of collapse. A sausage-like swelling appeared in the left iliac region, which could not be felt by the rectum. The vomiting was not fecal nor of fecal odor. I finally concluded that there was impaction, gave a dose of calomel, and she immediately recovered. At times during the summer, however, she would have pain in the bowels, and on one occasion vomited a good deal. In September, or early in October, she was taken down again in about the same way as in July; a dose of calomel was given and partial relief was obtained, but the next day excruciating pain came in the bowels, a tumor appeared lying across the epigastric region, or a little below, and complete obstruction followed. About the first of November she passed a piece of intestine, and was better for a few days, one or two small fecal discharges taking place. Obstruction returned, however; and finally, about three weeks ago, a movement (small and flattened) was obtained by means of calomel. We have not been able to get anything through her since then. She has not vomited much, has taken a little nourishment by the mouth, but has been chiefly fed by the rectum. She is greatly emaciated, and her pulse is small and feeble generally, though sometimes of considerable volume under the influence of morphine, of which she takes about one-eighth of a grain every four to six hours hypodermically. She has always been a rather delicate child, and has had several attacks of bronchitis. I have thought that there may be tubercular trouble of the intestines in addition to the purely mechanical trouble. I had three local physicians see her with me about the time I wrote you, and they all

¹ Read before the Boston Society for Medical Improvement, May 25, 1891.

²⁰ It may be objected to our statistics, that the parents' word was taken regarding infantile convulsions. This is, however, the ground upon which the statement in the text-books has been based, in which epileptics are said to have had infantile convulsions; in fact such statements are the only data upon which we can proceed. Those statements may err, it is true, but the errors will tend to counteract each other after all, for whereas some parents may mistake something else for a convulsion, others will overlook or conceal them. We have taken the greatest care to exclude as far as possible convulsions arising from organic disease, and in one family where each of six children is stated to have had convulsions during scarlet fever, the entire family was excluded.