SACHS (B) & PETERSON (F.)

A STUDY OF CEREBRAL PALSIES OF EARLY LIFE, BASED UPON AN ANALYSIS OF ONE HUNDRED AND FORTY CASES.

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A STUDY OF CEREBRAL PALSIES OF EARLY LIFE, BASED UPON AN ANALYSIS OF ONE HUNDRED AND FORTY CASES.*

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FEW diseases are better known or more thoroughly understood than infantile spinal paralysis. Its clinical symptoms and its pathology have been definitely determined, so that poliomyelitis anterior acuta scarcely needs further study. The very opposite is true of infantile *cerebral* palsy. While there is but a single form of disease included under the term infantile spinal paralysis, we are forced to admit that there are several different forms of cerebral palsy. The attempt to fix upon certain cases of cerebral paralysis in children and to label them infantile cerebral palsy as a direct analogy of the better known spinal disease has led to great confusion, although we shall see that there is some justice in drawing this analogy. Nor is it entirely correct to speak of cerebral *spastic* palsies as distinguished from *atrophic* paralysis, for we shall report at least two cases in which the element of spasticity was entirely wanting, although the cases were undoubtedly of cerebral origin, the proof of which was furnished by the post-mortem examination in one of these two cases.

The large majority of the cases with which we are here concerned represent spastic forms of paralysis and as regards the distribution of the palsy may very properly be

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divided into cases of spastic hemiplegia, of double spastic hemiplegia or diplegia, and of spastic paraplegia. Since the clinical subdivisions are so easily made, it would seem to be a curious fact that these cases have been so poorly understood and so little studied. There were many reasons for this: first and foremost, the great difficulties in obtaining autopsies, the majority of these cases either living on to a very advanced age, or else dying in almshouses, where no interest was taken in them; then again, the condition was so frequently associated with idiocy; or the individuals were regarded as hopeless cripples that did not possesss sufficient interest to repay careful study. And lastly, the term infantile cerebral palsy proved to be a stumbling-block. A number of cases and some few autopsies were reported, disclosing a variety of lesions; there seemed to be little hope of bringing order out of chaos. The truth of the matter, that we had a number of different forms of disease and a variety of pathological processes to consider, was a long time forthcoming. We hope to show by this paper that much of the confusion that has surrounded this subject will be removed if we consider that a variety of morbid symptoms may give rise to any of the three forms of paralysis, and that the character of the paralysis will depend upon the site and extent of the morbid lesion. But for the difference in the areas of the brain affected and the degree of irritation or destruction of brain substance, the symptoms in all these cases would be very much the same.

The subject which we present has a live interest at this present time, and yet it is nearly fifty years ago since the first work in this field was done. In 1842, Prof. Henoch wrote his inaugural dissertation, "De Atrophia Cerebri," and gave an excellent account of infantile cerebral paralysis.[1] Heine[2] referred to these cases in a monograph on spinal paralysis of children, published in 1860. Little[3] was well acquainted with them. In 1868, Benedikt,[4] the neurologist, described them. The French schools soon took up the subject, and Cotard,[5] Wuillaumier,[6] Bourneville,[7] wrote important papers on spasmodic infantile paralysis, some of

them referring to the anatomical features of these diseases. As recently as 1883, several English authors (among them Hadden[8] and Ross[9]) published the clinical details of a small number of cases and the accounts of a few autopsies.

Two publications have stirred up the recent discussion on this question. The first was Kundrat's[10] monograph on porencephalus, in which this one morbid state was carefully studied. The second was Strümpell's[11] paper in 1884, in which he suggested that infantile cerebral hemiplegia was due to a polioencephalitis acuta. This single statement which was decidedly original, but had no post-mortem proof, has raised a great hue and cry which have not yet subsided. Whatever other good it may have accomplished, Strümpell's theory has at least imbued an old subject with new life. His article was quickly followed by a number of valuable contributions, among which those of Ranke,[12] Bernhardt,[13] Wallenberg,[14] Kast,[15] Jendrássik and Marie,[16] Gowers[17] and Hoven[18] are by far the most important of the European contributions. In America, able articles have been written by Dr. Sarah McNutt,[19] Drs. Sinkler,[20] J. Lewis Smith,[21] Knapp,[22] Lovett[23] and Gibney.[24] Chief and foremost of all is an exhaustive monograph by Prof. Osler,[25] in which 151 new cases were analyzed with a skill which characterizes all of Prof. Osler's work.

There would seem to be some need of an apology after this for the study of another series of cases. Our first reason for doing this is, that the work preparatory to this paper was begun several years ago, before the publication of Prof. Osler's monograph. Secondly, we had some views of our own to advance; and, lastly, the unusual number of cases at our disposal called for special elaboration.

The cases here reported upon were, with very few exceptions, examined by one of the authors of this paper. We are greatly indebted to Dr. Gibney and Dr. Townsend for referring the greater number of these cases to us from the Hospital for the Ruptured and Crippled. Dr. Gray was kind enough to allow us to use the notes on cases seen in his department. Ten of the cases were seen in private practice.

The physician is no longer content, or at least should not be, to make the diagnosis of apoplexy; of hemiplegia, or of paraplegia, in the adult. It is his aim to determine whether the special form of paralysis be due to hemorrhage, thrombosis, embolism, tumor, abscess, or what not. In short, he studies the symptoms of each case with special reference to pathology of the disease. And so with infantile palsies: it is not enough to recognize spastic hemiplegia, diplegia or paraplegia, but the attempt should be made to determine the special morbid condition underlying each form. The large number of cases made it imperative upon us to make a distinct effort in this direction.

In view of the small number of autopsies recorded in literature this effort may seem hazardous; but a single autopsy in a well-observed case is a guide in the study of dozens of others; and with the advances made in a knowledge of cerebral lesions, the neurologist is fortunate in being able to reason with a great degree of certainty from clinical symptoms to the morbid lesion which they indicate.

A thorough knowledge of the clinical symptoms is, however, the starting point of this study. In order not to weary you with the details of these cases, we give you the conclusions to be drawn from our entire series with reference to each point at issue. And we shall give but a few histories in extenso that you may recognize the chief types of which we treat. Before proceeding to these cases, we submit a table showing that of the 140 cases* there were 87 males and 53 females; there were 105 hemiplegias, 24 diplegias, and 11 paraplegias.

TABLE I.—Showing the sex and form of paralysis in 140 cases of infantile cerebral palsy.

Form of Paralysis.	*Males.*	*Females.*	*Total.*	
Right Hemiplegia	37	15	52	105
Left Hemiplegia	26	27	53	
Diplegia	15	9	24	
Paraplegia	9	2	11	
Total	87	53	140	

* Since the above was written we have seen at least fifteen additional cases, but we have concluded not to alter the tables in the body of this paper.

HEMIPLEGIAS.

CASE I.—(No. 48.) A. F., æt. four and a half years, male, first child, difficult labor and instrumental delivery. From very first day right hemiplegia. Slight athetosis and associated movements. Contracture at elbow, formerly pes equino-varus, improved by operation. All reflexes of right side lively. Mental condition fair.

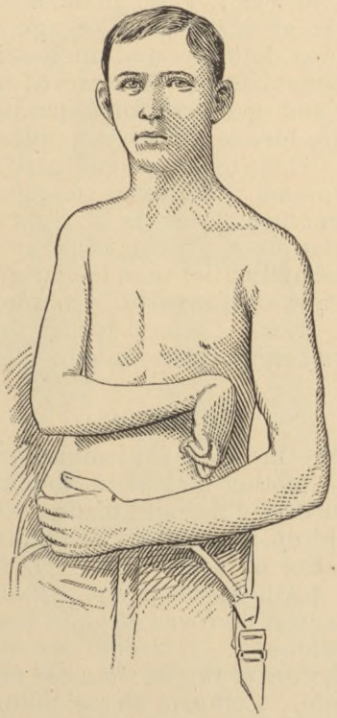


Fig. 1.—CASE IV., No. 138. Right hemiplegia with contracture and retarded growth of arm.

CASE II.—(No. 58.) L. B., female, æt. nine years. Congenital left hemiplegia. Labor normal; fourteenth child; movements of child ceased a few days before birth; expected to be still-born; weighed four pounds at birth. From age of two years epileptic attacks every two to four weeks. Fine associated movements of both sides. Arm, leg, and face of left side involved. Very great retardation of growth of arm and leg; some contractures of flexors of arm and wrist; imbecile; microcephalic.

CASE III.—(No. 44.) L. E., female, æt. eight years; acquired hemiplegia; onset at age of five years; convulsions for nine hours and coma; could not move, talk or walk for three months. Face and leg have recovered considerably; very great retardation of growth of arm. Marked associated movements. Contracture of right arm; reflexes of right side exaggerated.

CASE IV.—(No. 138.) J. K., male, æt. seventeen years. Right hemiplegia at eight years of age following typho-malarial fever; was delirious and unconscious during nineteen days; no convulsions. After recovering from coma, right arm, face, and leg were found paralyzed. Complete aphasia and entire loss of memory of everything occurring before typhoid. Had to be re-educated. Athetoid and associated movements. Reflexes exaggerated right side. Enormous contracture of flexors of right hand and fingers and great retardation of growth of right upper extremity. Right leg somewhat smaller than left; right talipes valgus; asymmetry of face. Electrical reactions and sensation entirely normal. Has recovered speech fully and is bright, but several years behind others in education. *Fig. I.*

DIPLEGIAS.

CASE V.—(No. 141.)* J. O., female, æt. sixteen. Congenital diplegia; mother kicked in abdomen by horse two months before birth of child and made unconscious thereby. Three other children, all healthy. Tedious labor, no instruments used, no fits or convulsions. Did not attempt to creep or walk; teeth at usual age. Patient has menstruated since tenth year and was weak in back, arms and leg from earliest childhood. Extreme spastic contracture of adductors and flexors of thighs; double talipes varus, equinus on right side. Left arm worse than right. Athetoid movements of left hand. Has frog walk. Intelligence good. *Fig. II.*

CASE VI.—(No. 31.) M. L., male, æt. three years. Congenital diplegia. Asphyxiated during labor. Mother had pneumonia, and died five days post partum. Rigidity of arms, legs and back. Hands did not unclinch for two years. Frequent convulsive seizures alternately of right and left

* This case, although under observation for a long time, was omitted from our list by mere accident. The history is sufficiently characteristic to deserve special mention.

side, including face. Cannot talk, walk or stand. Feeble minded; cross-legged position and all reflexes exaggerated.

Fig. II.— CASE V., No. 141. Diplegia; double talipes equinc-varus, athetosis of left hand.

PARAPLEGIA.

CASE VII.—(No. 50.)* C. F., male, æt. one year. Congenital paraplegia. First child, labor hard and dry for forty-eight hours. Asphyxiated. From first day up to age of six and a half months child had a rapid succession of tonic and clonic spasms affecting all the muscles of the body, causing rigidity of all extremities, opisthotones with extreme arching of back, enormous exaggeration of all reflexes, ankle and quadriceps clonus on slightest excita-

* While this article was passing through the press, this child died. An autopsy was obtained by Dr. L. E. Holt. A careful study of the pathological findings will be made and published in due time. But one other autopsy on infantile paraplegia has hitherto been published.

tion. Convergent strabismus ; crying continually, Mental condition probably imbecile. Epileptic spasms controlled slightly by bromide treatment. *Fig. III.*

Fig. III.—CASE VII., No. 50. Paraplegia. Photographed in epileptiform convulsion.

CHIEFLY MONOPLEGIA.

CASE VIII.—(No. 47.) H. K., male, æt. twenty-two months. Acquired right hemiplegia ; onset at six months following convulsions during pertussis and pneumonia. Trace of weakness in right arm ; distinct spastic paralysis of right leg ; knee-jerk exaggerated ; feeble-minded. This case made at first the impression of a monoplegia.

FLACCID PARALYSIS.

CASE IX.—(No. 61.) M., female, æt. two years at time of death. First-born of healthy parents. During the fifth month of pregnancy mother was thrown from a carriage without sustaining any serious injuries. The child born at full term apparently normal in all respects. At age of two to three months a general listlessness and nystagmus were observed. During its entire life child was unable voluntarily to move any muscle of its body. All muscles extremely flaccid, but all reacted perfectly to electrical currents. There was not the first symptom of any mental awakening. During the first year of its life child noticed light, but later on absolute blindness set in. There was a developmental defect of the optic nerves, which was reported upon by Dr. Knapp, a similar condition of the nerves having been observed in only two other cases. Hearing seemed to be acute. There was unusual hyperexcitability of auditory and tactile impressions. The child never had convulsions, not even during dentition ; no rigidities. All reflexes lively.

Speech was, of course, entirely wanting. The child died of pneumonia following bronchitis. The autopsy will be referred to later on. This case was made the subject of a special paper by one of us[26] (S.), and entitled "On Arrested Cerebral Development, with Special Reference to Cortical Pathology." This paper was a study of some of the cortical changes giving rise to idiocy, but we have since come to learn that the report of the case and the autopsy have a wider significance than was attributed to them at the time.

CASE X.—(No. 80.) H. M., male, æt. eleven and three-quarter years. The second of three children; an uncle said to have been similarly affected. Asphyxiated at birth; instrumental delivery. Began to teethe late; teeth have rotted away. Made imperfect attempts to walk at fourteen months; crept around on his buttocks; both feet turned inward, right more than left; learned to talk, but mind has always been very feeble. Has had frequent epileptic attacks, grand and petit mal. Both upper extremities excessively weak, but no rigidities. Lower extremities poorly developed. All muscles respond to faradic current, but some of them so feebly that very strong currents are needed. Knee-jerks weak but present; boy's father is a teacher who has done much with his defective mind.

From the records herewith presented to you, you will infer that much as the cases differ from one another, they also have much in common; they yield a distinct composite portrait. The child is either born with, or in its early life develops some form of paralysis; a hemiplegia, a diplegia, or a paraplegia. In the congenital cases there has been some disturbance during pregnancy, or labor has been tedious and difficult or definite cause cannot be given. In the acquired cases we have seen that the onset of paralysis may occur after acute infectious diseases, during convulsions, or from causes that cannot be fathomed. In the majority of cases there is marked spasticity and extreme contractures; in two cases there is a flaccid form of paralysis; in the last case reported, the knee-jerks and other reflexes were weak; in all other cases the reflexes were exaggerated, at least on the side or sides paralyzed. Some show peculiar associated athetoid or other post-hemiplegic movements; in all, there was more or less retardation of growth, and all stages of mental impairment were found from weak-mindedness to

complete idiocy; a few, however, are of good mental development. No changes in sensibility were observed, and the electrical reactions were never markedly altered.

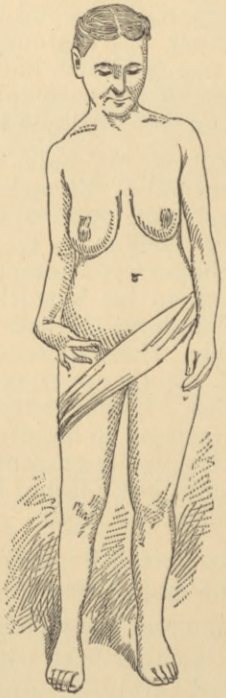


Fig. IV.—Right hemiplegia, from age of 9 months, in a woman 36 years. Contracture and retarded development of paralyzed side.

These cases and some of the symptoms they exhibit are referred to by the poet, historian, and the painter. The Duchess of Gloucester, according to Sir Thomas Moore, had much ado in her travail. Her son, "Richard III., came into this world feet foremost. Shakespeare makes "Richard" say of himself:

> "Deformed, unfinished, sent before my time
> Into this breathing world, scarce half made up,
> And that so *lamely* and unfashionable
> That dogs bark at me as I *halt* by them."

In Raphael's Transfiguration, the demoniac boy has characteristic athetoid position of one hand.

It is now in order to see whether our cases shed any new light on the understanding of these palsies, and what relation the facts hold to the commonly received classification into three distinct subdivisions.

TABLE II.—Showing the age at onset.

Age at Onset.	*Hemiplegia.*	*Diplegia.*	*Paraplegia.*	*Total.*
Congenital	22	20	7	49
During 1st year	27	1	2	30
" 2d "	17	1	—	18
" 3d "	13	2	1	19
" 4th "	4	—	—	4
" 5th "	4	—	—	4
" 6th "	2	—	—	2
" 7th "	1	—	—	1
" 8th "	5	—	—	5
" 9th "	—	—	—	—
" 10th "	—	—	—	—
Under 15 years	4	—	—	4
Unascertained	3	—	1	4
Total	105	24	11	140

From the above table we learn that of 105 cases of hemiplegia 22 are congenital; of 24 cases of diplegia 20 are congenital, and so are 7 or possibly 8 of the 11 cases of paraplegia. Diplegias and paraplegias are more likely to be of congenital origin, hemiplegias are more apt to be acquired in the first three or four years after birth; but it is well worth noting that there are 22 congenital cases of hemiplegia (over 20 per cent. of all cases of hemiplegia); some of the cases noted as occurring in the first year may be congenital, and this would help to swell the percentage of congenital hemiplegias. It will not do, therefore, to make the broad distinction so frequently made on the basis of the acquired or congenital character of these palsies. Our table is in happy agreement with the one given by Osler as regards hemiplegias (15 of 120 were congenital). Sixty of our hemiplegic cases were developed before the close of the third year of life; then there is a distinct falling off up to the age of ten; between ten and fifteen we have a few more; in three cases the age could not be ascertained. Will you also note that some of the acquired

cases of paraplegia or diplegia have occurred after the age of three years, and one or the other of these cases may have been congenital.

TABLE III.--Showing age at examination.

Age.	*Hemiplegia.*	*Diplegia.*	*Paraplegia.*	*Total.*
Under 4 years	40	16	8	64
Between 4 and 10 years	26	6	2	34
" 10 and 20 "	14	2	1	17
" 20 and 30 "	18	—	—	18
Under 40 years	7	—	—	7
Total	105	24	11	140

This table records the ages at examination, from which it is apparent that diplegia and paraplegia are comparatively short-lived while hemiplegias often attain a very considerable age. Statistics of the exact ages at death would be more accurate; but inasmuch as our material includes cases from every kind of institution, even from pauper asylums, the inferences to be made are tolerably correct.

TABLE IV.--Showing causes given in 91 cases of acquired cerebral palsy.

Causes Given.	*Hemiplegia.*	*Diplegia.*	*Paraplegia.*	*Total.*
Convulsions	20	1	—	21
Pneumonia	6	—	—	6
Trauma to Head	6	—	—	6
Pertussis	4	—	—	4
Measles	2	2	—	4
Scarlatina	3	—	—	3
Onset with fever	2	—	1	3
Hereditary Syphilis	2	—	—	2
Cerebro-spinal Meningitis	2	1	—	3
Onset with fever and convulsions only	2	—	—	2
Fright	2	—	—	2
Hyprocephalus	—	—	2	2
Vaccinia	1	—	—	1
Typho-malarial Fever	1	—	—	1
Small-pox	1	—	—	1
Tonsillitis	1	—	—	1
Epileptic Seizure	1	—	—	1
Gastro-enteritis	1	—	—	1
Unascertained	26	—	1	27
Total	83	4	4	91

TABLE V.—Showing ascertainable causes in 49 cases of congenital cerebral palsy.

Causes.	*Hemiplegia.*	*Diplegia.*	*Paraplegia.*	*Total.*
Instrumental delivery, tedious labor	4	1	1	6
Ante-partum trauma to mother	2	3	—	5
Premature birth	1	2	1	4
Asphyxia at birth	—	3	—	3
Asphyxia in twin birth	—	1	1	2
Tedious labor, breech presentation	2	—	—	2
Primipara, dry birth, tedious labor	—	—	1	1
7 mos. child, dry birth (48 hours)	—	1	—	1
Primipara (æt. 45), tedious labor	—	1	—	1
Maternal fright (ante-partum)	1	—	—	1
Uræmia of mother	1	—	—	1
Pneumonia of mother (died 5 days post-partum), child asphyxiated	—	1	—	1
Convulsions of mother during pregnancy, difficult la or	—	1	—	1
Mother in fever for 10 weeks, ante-partum	1	—	—	1
Unascertained	10	6	3	19
Total	22	20	7	49

In these tables we have analyzed, as far as possible, the causes in 91 acquired cases and 49 congenital cases of cerebral palsies.

Among the acquired hemiplegias the acute infectious diseases, including pertussis and pneumonia, play a very important role; a strikingly large number have come on during convulsions; in these cases the convulsions are not the initial convulsions of acute infectious diseases; the latter are considered separately in Table VI. The cases that have come on with fever and convulsions are noted in addition; but not wishing statistics to prove more than

TABLE VI.—Showing the relation of convulsions to the onset of the palsies.

Cases in which convulsions apparently preceded or were associated with the palsies occurring in —

Pertussis	3
Pneumonia	2
Scarlatina	1
Dentition	1
Vaccinia	1
Baptism	1
Fright	1
With beginning of menstruation	1
Fall on head	1
Gastro-enteritis	1
Total	13

Apparently immediate symptom of a focal lesion—in 2 cases.
Idiopathic and apparently only cause of palsy—in 20 cases.
Palsy occurring in ordinary epileptic seizure—in 1 case.

they should, it is but fair to add that in many of these cases fever may have been present, and that from among these 20 cases we may allow that several, if not all, show the onset Strümpell claims for his cases of polioencephalitis. We ask you also to note that in 6 cases of hemiplegia there was a distinct history of traumatism, that 2 hemiplegias and 1 diplegia were the result of cerebro-spinal meningitis; 2 cases of diplegia came on after measles, a fact of some importance. In 26 cases of hemiplegia we were not satisfied with the statements elicited, and have therefore marked them as "causes unascertained." The table of causes in the congenital cases points a moral. In 16 cases of the 49, say in 33.3 per cent., there was some difficulty in labor, simple delay or instrumental delivery. The older writers, Little, Gaudard,[27] and others refer to this cause, but have tolerably favorable statistics, Little mentioning but 4 cases; Wallenberg gives 6 of 160 cases, and Osler 9 of 97 cases. The authors mentioned referred to hemiplegia only, and speak of forceps delivery as the element of danger. Our percentage is higher, because we include all forms of cerebral paralysis, and tedious labor as well as instrumental delivery. The moral is, that the forceps should be applied, if necessary, or delivery hastened by other means if protracted labor can be averted. A child's brain and skull have a wonderful power of resistance, but do not credit them with greater virtue in this respect than they really possess. The mother's life is by far the more important, but it is well to reflect that other things being equal she prefers a child that is neither paralyzed or idiotic.

As regards the mode of onset, of the congenital cases, it would appear paradoxical to say anything; but in several cases which were distinctly congenital, attention was first drawn to the disease by the appearance of convulsions at an early day. In these cases the convulsions are due to the same lesion or process which is responsible for the palsy. In the acquired cases convulsions preceded the onset of the other symptoms in 36 of 83 cases of hemiplegia, and in one case of acquired diplegia; loss of consciousness generally accompanies the convulsions; in 6 cases there was a dis-

tinct onset without loss of consciousness or convulsions; this occurred in 4 cases of left hemiplegia and in 2 of right hemiplegia; 2 of these left hemiplegias were distinctly syphilitic.

TABLE VII.—Analysis of six cases in which there was onset without loss of consciousness or convulsions.

Hemiplegia.	*Age.*	*Cause.*
Right with aphasia	2 years	Tonsillitis.
Right	10 mos	Unknown.
Left	2½ years	Syphilis.
Left	8 "	Unknown.
Left	1½ "	Syphilis.
Left	2¼ "	Fall on head.

Strümpell has made the onset with convulsions and fever a distinctive feature of his cases. The 6 cases referred to in our list answer to his description of infantile cerebral palsy as regards the hemiplegic form of paralysis. It is more natural to infer that they are not cases of polio-encephalitis than that they are anomalous cases of that class.

On this subject of initial convulsions and loss of consciousness a word should be added, even at the risk of anticipating some inferences regarding the pathology of these palsies. Initial convulsions and loss of consciousness are distinctly cortical symptoms. They indicate very considerable cortical disturbance either by direct injury or by severe or sudden injury to any part of the brain, which would also imply disturbance of the cortex as of every other part of the brain. Relatively small injuries to the cortex, hemorrhages or cortical encephalitis, will bring about loss of consciousness and convulsions; relatively large injuries to the interior need not exhibit these symptoms. The exception is in cases of embolism or sudden hemorrhages in which the suddenness of the shock disturbs the entire brain. This argument was urged by one of us[28] (S.) in a paper published some years ago in which a typical case of capsular hemorrhage was diagnosticated mainly on the line of argument just referred to; and in examining statistics of Wallenberg and others with reference to this point, we find that convulsions occurred only in cases of

embolism anywhere, and in all cortical affections, however slight these may have proved to be. If all cases of infantile hemiplegia were cases of polioencephalitis corticalis, convulsions would invariably be present, but such is not the case.

The *form* of paralysis has been frequently referred to. Hemiplegia, double hemiplegia or diplegia, and paraplegia speak for themselves. Table I. gives the relative number of cases of each form in our list. Monoplegias are not included, although other writers had reported some such cases. We have seen but one case in which we were tempted to make a diagnosis of monoplegia, and this case gave distinct evidence on closer examination that the arm as well as the leg had been involved in the earlier course of the disease. The march of the disease resembles adult hemiplegia in this that the leg recovers very much more quickly than the arm for reasons that need not be given here. Under the heading of diplegia we have classed all cases in which both upper and lower halves of the body were involved; in some of these one leg or one arm had so far recovered that the cases might have been interpreted as hemiplegia with an additional involvement of the other arm or leg; but here again on closer scrutiny we became convinced that at one time all four extremities had been affected.

The involvement of the face is a matter of some doubt. The majority of the cases were seen at a time long after the recovery of the face. We can vouch, however, for the following statements. The face was affected in 11 cases of right hemiplegia, in 9 of left hemiplegia, and in 2 cases of diplegia. In 2 cases (1 of right and 1 of left hemiplegia) the leg was worse than the arm; in all other hemiplegics the arm was the part more affected. In all diplegics the legs were more affected than the arms. Trunk and neck muscles were distinctly involved in two cases of diplegia. Strabismus occurred in one of right hemiplegia, in four of left hemiplegia, and in three cases of diplegia.

It has been doubted whether aphasia followed the same laws in the infantile hemiplegia as in the adult form, and

Bernhardt[13] has seen fit to write at length on this special subject. Aphasia can only be said to be present if the hemiplegia comes on in an individual who had already acquired articulate speech, hence the 49 cases of hemiplegia and the 21 of diplegia which occurred before the age of two years are excluded from this consideration and of the remaining 56 cases of hemiplegia the fewest dispensary patients or their parents could give satisfactory statements. We have records of 17 cases of hemiplegia with undoubted aphasia. Of these 17, 10 were cases of right hemiplegia and 7 cases of left hemiplegia. Eight of these 17 cases were observed by one of us (S.) in private practice. Of these 8, 5 had been distinctly aphasic, and three of the 5 were cases of left hemiplegia. This relatively large proportion of aphasia in cases of left hemiplegia is rather striking by contrast with the adult cases. As we grow older we appear to become more and more left-brained. In earlier years both hemispheres are equally entrusted, so it seems, with this highest faculty of speech. Bernhardt also comes to the conclusion that aphasia in children accompanies right as well as left hemiplegia. Prof. Osler notes aphasia in 13 out of 120 cases of hemiplegia and only one of these with left hemiplegia. Wallenberg's statistics give 45 cases in 94 right hemiplegics and 17 in 66 left hemiplegics, but he includes all sorts of speech disturbances, and the statistics cannot well be utilized with regard to true aphasia. Defective speech was noted three times in our cases of right hemiplegia, twice in diplegia, and in most of our 80 cases of idiocy.

The reflexes are either lively or exaggerated in the large majority of cases. The exceptions are noted in Table VIII.

TABLE VIII.—Showing the condition of the deep reflexes (knee-, elbow- and wrist-jerks) in 11 cases in which they were not exaggerated.

Form.	*Normal.*	*Diminished.*	*Absent.*	*Total.*
Hemiplegia	4	1	3	8
Diplegia	1	1	1	3
Total	5	2	4	11

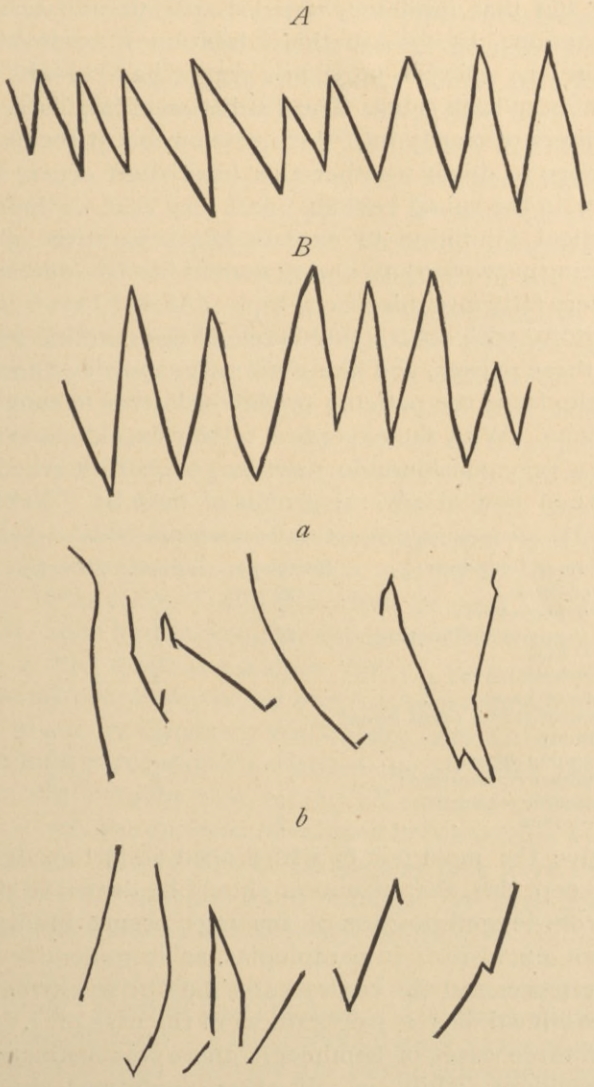
In all other cases the deep reflexes were exaggerated.

The points to be noted are these: The exaggeration of reflexes occurs in the parts paralyzed or paretic. The great excitability of the reflex is often the best proof to be had of the former involvement of the leg or arm, although bilateral exaggeration of the reflexes in cases of unilateral paralysis must be taken into account; owing to the extreme contractures of the opposing sets of muscles, the reflexes can frequently not be obtained. This is true particularly of the ankle clonus and triceps reflexes. We must note, however, that in several cases the knee-jerks and other reflexes were subnormal and in some cases absent. In one case (No. 50) the quadriceps clonus could be obtained.

Disturbances of motion, such as choreiform, athetoid associated movements are observed with unusual frequency after hemiplegia, and as we have learned also after diplegias in children.

In 105 cases of hemiplegia,			choreiform movements		occurred in	6
"	"	"	athetoid	"	"	21
"	"	"	associated	"	"	15
"	"	"	rhythmical	"	"	1
"	"	"	ataxia		"	1
"	"	"	tetanoid contractions		"	1
In 24 cases of diplegia,			choreiform movements		occurred in	1
"	"	"	ataxia		"	1
"	"	"	athetoid movements		"	1
"	"	"	nystagmus		"	2

These disturbances of motion after single and double hemiplegias have an unusual interest; they all point to some interference with the proper transmission of motor impulses either of the voluntary or of the inhibitory kind. The changes in the pyramidal tract are no doubt largely responsible for these peculiar movements, and that disturbances anywhere in the motor tract may bring about such aberrant movements is probable from the fact that these movements occur from the most diverse lesions, from lesions in the cortex as well as from lesions of the crus, as shown in an unpublished case of our own. Of associated movements we have seen a number of happy examples. In one instance the movements of the paretic arm and hand



A and B—Movements of the normal hand.
a and b—Movements of paretic hand.

following the movement of the opposite side were so considerable that we were able to take tracings exemplifying this fact. Westphal[28] has offered the theory that in health there is a tendency for both upper extremities to act con-

jointly, but that inhibitory impulses permit unilateral and single action. In disease this inhibition is removed, and therefore the effort to move one arm or hand results in the movement of both sides. The irradiation of impulses, therefore, enters primarily into this question, but it seems to us to be open to doubt whether this irradiation occurs in the brain or in the spinal centres. In every case we must call in cortical inhibition to explain the separation of these irradiating nerve currents during normal life. Greidenberg has gone very fully into this special topic. (Arch. f. Psych. vol. 17).

Rigidity with contracture is one of the cardinal features of all these palsies, and the deformity resulting therefrom, as a rule, leads the patients or their relatives to seek medical advice. With the exception of the flaccid cases before noted, a very considerable number go on to marked contracture of one or several groups of muscles. Table IX.

TABLE IX.—Showing the forms of contracture in the various cerebral palsies.

Form of Contracture.	*Hemiplegia.*	*Diplegia.*	*Paraplegia.*	*Total.*
Flexors of elbow	29	—	—	29
" " both elbows	—	3	—	3
" " carpus and fingers	23	1	—	24
" " knee	5	—	—	5
" " both knees	—	2	—	2
Ext nsors of knee	1	—	—	1
Adductors of thighs (cross-legged position)	—	13	2	15
Talipes equino-varus	17	—	—	17
Double talipes equino-varus	—	5	2	7
Talipes equino-valgus	3	—	—	3
Talipes equinus	2	—	—	2

will give the main points, which need no further explanation except this, that attention should be drawn to the fact that cross-legged position of the legs occurs in diplegias and not exclusively in paraplegias, as is generally stated. Furthermore, that the contractures may be so extreme that the individual has a frog gait, as in the case of J. O., and that in three cases of hemiplegia there was distinct talipes equino-valgus, while in all other conditions there was either simple varus or equino-varus position.

Why flexor and not extensor muscles are the seat of contracture, adductors and not abductors, is a puzzling question. It is probably due to the mechanical principles involved in the construction of joints.

A word in passing with regard to trophic disturbances. In many of these cases the circulation in the skin of the paralyzed limbs is as poor as in infantile spinal paralysis. There is often the same livid hue of the palsied limb. In all cases occurring early in life, and particularly in those of congenital origin, there is apt to be a very marked retardation of growth. This was most distinct in nine cases of hemiplegia, three of diplegia, and three of paraplegia. Atrophy of the thenar eminence was observed in one case of diplegia, coming on after a cerebro-spinal meningitis.

The association of epilepsy with infantile cerebral palsies is perhaps the gravest feature of these diseases. In our experience it is the one danger to be feared, and should be considered most carefully in any case of cerebral palsy in a child. In our list, 62 out 140 cases were afflicted with epilepsy, or 44.3 per cent. of all cases. There were among the hemiplegic cases 41 cases of general epilepsy, 9 of the Jacksonian type, and one case of petit mal (in all about 50 per cent.). In 24 cases of diplegia 7 had general epilepsy (29 per cent.), one had Jacksonian epilepsy. In 11 cases of paraplegia 4 had general epilepsy (about 36 per cent.). The percentage of epilepsy in our own list differs but slightly from that given by Gaudard, Wallenberg, and Osler. In view of the high percentages conceded on all sides, there can be but little doubt that taking all cases of ordinary epilepsy, a very fair proportion developed in connection with infantile palsies. Every sign of early palsy may have disappeared, while the epileptic taint remains. A case seen in private practice brings this out very forcibly:

E. B. (No. 81), girl seventeen years of age; menstruated at age of eleven; third of four children. All others died of acute infectious diseases. Father was fifty-six years of age at time of birth of child. Mother was thirty years younger. The child has had epileptic attacks every three to four months for some years. No convulsions during childhood. Had been treated for ordinary epilepsy by many physicians; brominized for years. Closer inquiry revealed the fact that about four years ago the girl had an apoplectic attack, and on examination we found distinct evidence of left hemiplegia. Very marked weakness of left side; greatly

increased reflexes ; slight mental enfeeblement and marked anæmia. Bromides were discontinued, the patient put on tonics, and is doing well, at least as well as under bromides, which had evidently deepened the mental apathy.

It will be seen that the percentage of epilepsy was greatest in hemiplegia, but that it was a distinctive feature of all forms of paralysis here considered.

Taking into account the cortical o.igin of a large number of these cases of epilepsy, it was natural to expect a higher percentage of Jacksonian epilepsies, but in most of these cases the original focal lesion has disappeared, and a general atrophy and sclerosis have been established. General and not localized epilepsy is the natural result of this change.

Together with the occurrence of epilepsy we should note the large number of cases exhibiting some form of mental enfeeblement. The conclusions on this point are presented in Tables X and XI, where we have attempted

TABLE X.—Showing the relation of mental defect to the age of onset alsy.

Age of Onset of Paralysis.	*Feeble-mindedness.*	*Imbecility.*	*Idiocy*	*Total.*
Congenital	6	15	14	35
Under 3 years	10	18	6	34
3–5 years	2	3	—	5
5–10 "	2	3	–	5
Over 10 years	1	—	—	1
Total	21	39	20	80

TABLE XI.—Showing the relation of mental defect to the form of palsy.

Form of Mental Defect.	*Hemiplegia.*	*Diplegia.*	*Paraplegia.*	*Total.*
Feeble-mindedness	16	2	3	21
Imbecility	31	7	1	39
Idiocy	7	8	5	20
Insanity (epileptic)	1	—	—	1
Total	55	17	9	81

to distinguish between feblee-mindedness and imbecility and idiocy. Eighty of 140 cases exhibited some form of mental impairment ; 69 were either in congenital cases or in those acquired in the first three years of life ; 52 per cent. of the hemiplegic patients, 71 per cent. of the diplegic, and about 82 per cent. of the paraplegics were thus afflicted.

The worst form (idiocy) was most marked in the paraplegias (45 per cent.), and least marked in hemiplegia, being found in only 6.75 per cent. of all cases. Here again mental impairment can be said to be in proper relation to the extent of the cerebral lesion which is presumably less extensive in cases of hemiplegia than in the other forms of cerebral palsies; though we must allow that the later development of many of the hemiplegias may have something to do with these results.

As a point of special interest we wish to add that Little in 19 cases of paraplegias in which there was some impairment of mental condition found 13 feeble-minded or idiotic (68 per cent.) and 6 (32 per cent.) of good intelligence.

TABLE XII.—Analysis of *stigmata degenerationis* (exclusive of contractures) present in 57 cases of cerebral palsy in children.

	Hemiplegia.	*Diplegia.*	*Paraplegia.*	*Total.*
Microcephalus	21	4	2	27
Leptocephalus	19	1	—	20
Macrocephalus	4	—	1	5
Marked cranial asymmetry	25	—	2	27
Marked facial asymmetry	19	—	—	19
Cranium proganæum	5	2	—	7
"Gothic" palate	9	1	—	10
Imperfectly developed teeth	10	1	1	12
Supernumerary teeth	—	1	—	1
Hirsuteness	—	1	—	1
Neuropathic ear	1	1	—	2
Strabismus	5	3	—	8

As for cranial defects,[29] the table appended will give all the information we have, though it is well to note that in most cases of hemiplegia there is a flattening of the skull on the side of the lesion, and in almost all cases of cerebral palsy most of the cranial diameters are below the normal averages.

In this review of clinical symptoms we have shown that excepting the fact of paralysis, there are no symptoms peculiar to infantile hemiplegia that are not also found in diplegia and paraplegia. While the symptoms vary somewhat in degree in these different forms, all forms have all symptoms in common, excepting those of the onset. It is a difference of degree, not of kind. It remains for us to prove whether or not a study of pathological conditions

compels us to draw a distinction between these three forms, and whether or not hemiplegia, diplegia, and paraplegia respectively represent distinct morbid entities. We shall see that a variety of morbid lesions is to be found underlying these conditions, and that the same lesion or condition may in the one case be responsible for a hemiplegia, in the other for a diplegia, and so on. And furthermore, if definite forms of disease are to be diagnosticated, the diagnosis must rest upon other symptoms rather than the mere form of the paralysis. This branch of our inquiry is beset with great difficulties. In the scarcity of autopsies we have shared the fate of other writers. In spite of our very large clinical experience, we have but two autopsies of our own.* But we claim this one advantage that in both these cases the post-mortem findings were of very recent date—a great advantage, if we reflect that in most of the cases recorded in literature the conditions found were the final result of pathological processes which had continued for years, and which shed no light whatever upon the initial morbid lesion, and yet this is the salient point of the entire controversy. With the information gained by our macroscopical and microscopical studies we have analyzed the records of 105 autopsies including our own. This list could have been enlarged if we had had access to the publications of Richardiere[30] and some others. Our list, however, in-

TABLE XIII.—Analysis of pathological findings in hemiplegia, diplegia and paraplegia, based upon the most recent autopsies, including Wallenberg's and Osler's cases, but not those of Richardière or Audry.

Lesions.	*Hemiplegia.*	*Diplegia.*	*Paraplegia.*
Atrophy, sclerosis and cysts	40	19	—
Porencephalus	2	4	—
Hemorrhage	23	—	—
Embolism	7	—	—
Thrombosis	5	—	—
Agenesis	1	1	—
Tubercle	1	—	—
General cortical sclerosis	—	—	1
Total	79	24	1

Not including 64 cases of hemiplegia with porencephalus, and 32 cases of bilateral porencephalus collected by Audry.

*A third autopsy has been added since the above was written (cf. note p. 301).

cludes all the recent cases,* and the report of these will atone for the omission of those described by the older writers.

The first and the most conspicuous feature of this table is the prominence given to atrophy, sclerosis, and cysts. All these are terminal conditions and are almost useless for the determination of the initial lesion. Cysts are no doubt frequently due to hemorrhages, and if this could be statistically shown, the number of cases due to hemorrhage would have been materially increased. Zacher has noted one case in which cystic formations occurred together with an osteoma, but the patient died thirty-three years after onset of lesion. Wallenberg has recorded an interesting case of cyst in the left peduncle.

Porencephalus is a secondary condition, and although much has been written on this subject, we know little of its origin. In some cases it is probably the result of arrested development, due to interference with fœtal circulation (Kundrat); in others it may have been the result of early encephalitis or even intra-uterine cerebral hemorrhage. A number of different lesions may bring about a condition of porencephalus; moreover, the term has proved to be exceedingly elastic, and what one author has termed atrophy, another has called porencephalus. Audry,[37] for instance, has collected 64 cases of this condition, some of which in the other lists are labeled atrophy or atrophy and sclerosis. We are forced, therefore, to rely upon cases in which the initial lesion has not disappeared; and here the first fact that is brought out with great distinctness is that hemorrhage, thrombosis, and embolism, the conditions which give rise to adult apoplexy, are also found to be a frequent cause of the cerebral palsies of early life. There is mention in the table of a case of tubercle reported by Seeligmüller.[38] It is hardly fair to include such a case, for it was one of general tuberculosis in the course of which a hemiplegia appeared. While no other similar cases have been published, it is evidence of faulty logic that Seeligmüller should

* Two cases of Kast, one by Hoven, Wallenberg, Salgo,[31] Langenbeck,[32] Fürbringer,[34] Zacher,[35] Hirt,[36] Peterson [cf. page 323 of this article], and Sachs.[26]

have inferred from this one case that latent tuberculosis is almost the sole cause of infantile hemiplegia. His case simply shows that hemiplegia may appear together with other symptoms, but there can generally be little doubt as to the nature of the process. It is for this reason that we have excluded from our list a case of our own in which the autopsy revealed a tubercle of the quadrigeminal region which had given rise to a hemiplegia in the course of the disease.

It may be surprising to find that polioencephalitis of Strümpell is not referred to. It is time that we should define our position with regard to this question. First of all, in order to distinguish this from two other lesions which have been termed polioencephalitis inferior (progressive bulbar paralysis) and polioencephalitis superior (nuclear ophthalmoplegia), let us speak of this as polioencephalitis corticalis. What proof have we that there is such a condition? Anatomical proof, none; we are willing to concede, however, that some of the many cases of atrophy and sclerosis may have been due to this polioencephalitis, but it is unfortunate for Strümpell's theory that all of the autopsies made soon after the onset of the disease have shown other conditions, and not a polioencephalitis. But let us be charitable or just, and say that even these autopsies were not made in cases sufficiently recent. We must add, however, that cases which correspond very closely to the cases which Strümpell considered typical of polioencephalitis corticalis showed hemorrhage, embolism, etc., of recent origin. Strümpell says, however, that not all cases of infantile hemiplegia need be due to this cause, and that all authors have misinterpreted his views. Is there no probability, then, that a few or any of the cases of infantile hemiplegia are cases of polioencephalitis corticalis? There is some circumstantial evidence showing that there is a brain-lesion which would seem to be analogous to spinal palsy of children (poliomyelitis anterior). Möbius[39] gives the history of two children of one family, aged one and one-half and three years respectively, who were stricken down with fever, loss of appetite, and somnolence. One developed

a typical poliomyelitis of the upper extremity; the other, spasmodic hemiplegia without aphasia. This is striking clinical evidence, though some might claim it to have been merely a coincidence. Another proof: Strümpell has but very recently reported two cases of adult apoplexy in which every one would have made the diagnosis—and indeed he made it—of embolic softening, but the post-mortem examination revealed a condition of encephalitis hæmorrhagica of the gray as well as of the white matter. Marie,[16] who is inclined to support Strümpell, expressed the opinion in 1885 that the encephalitis would attack the white as well as the gray matter, and thinks that this would not destroy the analogy with poliomyelitis, for in that condition the white fibres are sometimes involved. Jendrassik and Marie favor the perivascular (inflammatory) origin of the condition of lobar sclerosis, which they have carefully described. In view of all this, we venture the opinion that polioencephalitis corticalis may be the cause of some of the cases of infantile palsies; but, we add, not of the hemiplegia alone, for we have seen several cases, including one seen by the courtesy of Dr. Holt, in which all the symptoms were those of Strümpell's disease, but there was a diplegic and not a hemiplegic form of palsy. In these cases the cerebral character of the symptoms was so distinct that a confusion with poliomyelitis was out of the question.

We insist that, until further pathological proof shall be forthcoming, polioencephalitis corticalis shall be diagnosticated last, not first. In a short paper, published some years ago in the JOURNAL OF NERVOUS AND MENTAL DISEASE, one of us insisted that there was strong reason to think, by analogy with adult apoplexy, that the lesion might be the same in the infantile form. A short abstract of that history will show the analogy:

M. M. (Case No. 75), boy, two and one-half years old; one and one-half years previously pneumonia; tonsillitis with fever up to 102°. Four days after this, typical right hemiplegia and aphasia, positively without coma or convulsions. The onset was as typical as in the ordinary mild hemorrhage into the internal capsule in the adult, and the

progress of the disease and the mode of recovery (which became complete) were quite like what we see in adult cases.

In 1887 the evidence on this point was not so strong as it is now, and this leads again to the subject of thrombosis, hemorrhage, and embolism. These vascular troubles were generally considered to be peculiar to advanced age, with the exception of embolism, which every one is ready to concede may occur at any age in which heart-disease occurs. Thrombosis was thought frequent enough, from the occurrence of syphilitic endarteritis in children. Hemorrhage in adults is attributed to the existence of miliary aneurisms. In the child, miliary aneurisms have been found, and Prof. Osler[25d] has described a large aneurism of the anterior cerebral artery occurring in a boy six years of age. If atheromatous degeneration of the arteries be less frequent than in adults, another condition is found to which Recklinghausen refers in his book on the "Pathology of Circulation and Nutrition," page 84. This is a fatty degeneration in the wall of the cerebral blood-vessels. Little notice has been taken of this, as indeed of all else that pertains to the vascular pathology of children.

Meningeal and cortical hemorrhages are shown to be more frequent in children, while all other cerebral hemorrhages are more frequent in adults. This will explain the more serious character of the symptoms in the young, and the more frequent occurrence of epilepsy and mental impairment in children. As the prognosis would be materially affected according to the cortical or cerebral character of the lesion, we have sought for the point of differential diagnosis between lesions so situated. To this end the occurrence of convulsions is specially to be considered. In the paper referred to it was stated: "Loss of consciousness is an extremely variable symptom; it seems to depend rather upon the quantity of blood effused than upon the area involved. Not so with convulsions. A convulsion, if it is anything, is a cortical affair, the result of cortical irritation." To which should be added that the irritation may be direct or indirect; the sudden shock imparted to the

cortex by the occurrence of embolism anywhere in the brain is apt to cause convulsions, and a lesion in any part of the gray matter of the central nervous system may excite convulsions; but hemorrhage over the cortex, even if slight, is apt to be accompanied by convulsions. Hemorrhage into the interior of the brain, unless very large, and the condition of thrombosis—slow occlusion of a cerebral vessel—are more apt to be marked by absence of convulsions and possibly also by preservation of consciousness. In other words, if you can exclude embolism and sudden and very large hemorrhage, and lesion of the lower gray centres, the absence of convulsions at the onset of an apoplectic attack is in favor of a lesion in the interior of the brain rather than in or upon the cortex. Of this I am more certain still, that whatever may be the symptoms accompanying the onset of an apoplectic attack, if the convulsions reappear after the initial symptoms, the lesion is cortical, or else an additional insult has occurred. A possible exception might be made in favor of thalamus lesion. These statements are corroborated by the results of recent autopsies, though only a small number of cases refer to all the facts needed. A case observed and examined post-mortem by one of us (P.) is in point:

A. W., male, aged fifteen, bright at school, expert swimmer, at age of eight or nine years was in the habit of diving a distance of twenty to thirty feet from a railroad-bridge. Shortly after this began to suffer from intense headaches, which gradually grew worse, until he was twelve years old, when mental changes began to be apparent. Gradually loss of memory, confusion of mind, and steady progress toward dementia. At the same time moroseness, melancholia, morbid fear, and coprolalia. One month after admission into the Poughkeepsie Asylum had a severe epileptic fit. Two days later right hemiplegia with constant right-sided hemi-epilepsy; became comatose. The clonic spasms of the right side continued at intervals of several days, but gradually became limited to the right leg alone. Four days later, death. Autopsy showed the outer dural surface to be normal; on the right side the subdural space presented a limited pachymeningitis hæmorrhagica interna, but merely a thin organized detachable stratum of long

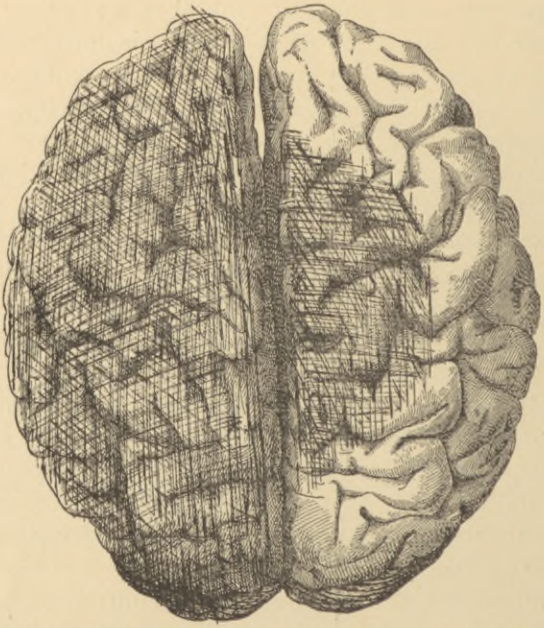


Fig. VI.—Showing extent of pachymeningitis hæmorrhagica over superior surfaces of hemispheres.

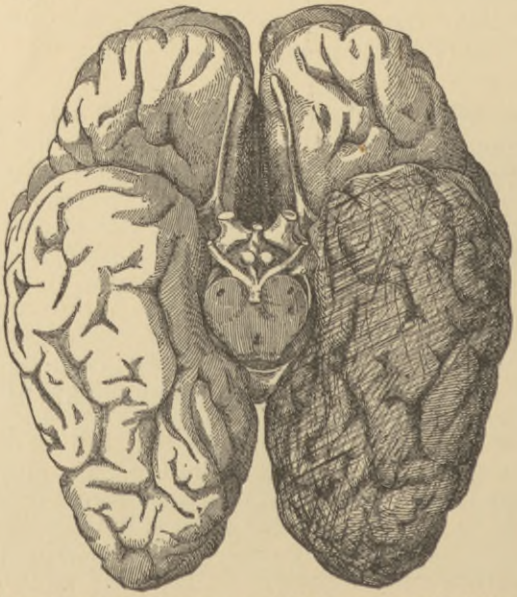


Fig. VII.—Showing extent of process on inferior surfaces of left hemisphere.

standing. Over the left side, however, the pachymeningitis was exceedingly widespread, extending over the whole surface of the hemisphere, both above and below, except in the anterior fossa of the skull. Over the convexity of the hemisphere the hæmatoma was very thick, consisting of strata of various ages, some of them undoubtedly dating from the beginning of his symptoms. Between two and three ounces of fresh coagula were spread on the convexity, especially in the Rolandic region. The extent of the pachymeningitis and hemorrhage is shown in the accompanying illustration. The left hemisphere was greatly compressed; the brain-substance itself seemed normal. The brain weighed 35¼ ounces. The ventricles were widened and distended with clear serum. There was no disease of or injury to the cranial bones. There was no lesion of any kind in substance of hemispheres, ganglia, pons, or medulla. *Figs. VI. and VII.*

Thrombosis occurs in a comparatively small number of cases. It is due either to the fatty change in the walls of the blood-vessels, or to syphilitic endarteritis, of which we have seen several cases, and Seibert[40] has described one, and is probably found in marantic cases. Gowers lays particular stress upon the thrombosis and occlusion of smaller cerebral veins, and thinks this the most important factor in the causation of infantile hemiplegia. All that we can say is that he must prove this to be true; the autopsies analyzed give no evidence whatever of this condition.

Thus far we have had reference chiefly to the pathological lesions of infantile hemiplegia. In diplegia nothing is said in our table of hemorrhage, thrombosis, and embolism, though it is more than likely that some of the cysts referred to were originally due to clots, and the occurrence of meningeal hemorrhages over both hemispheres giving rise to diplegias is well known. But these cases either die early before the form of paralysis is well established, or if they live for many years the initial hemorrhage has disappeared and the secondary conditions only remain. You will please observe, however, that what is properly termed agenesis corticalis occurs in a case of hemiplegia reported by Kast, and in the case of diplegia reported by one of us (S.) The

microscopical changes appear to have been very similar in both cases—in the one case unilateral, in the other bilateral, which accounts for the hemiplegia in the one case, for diplegia in the other.*

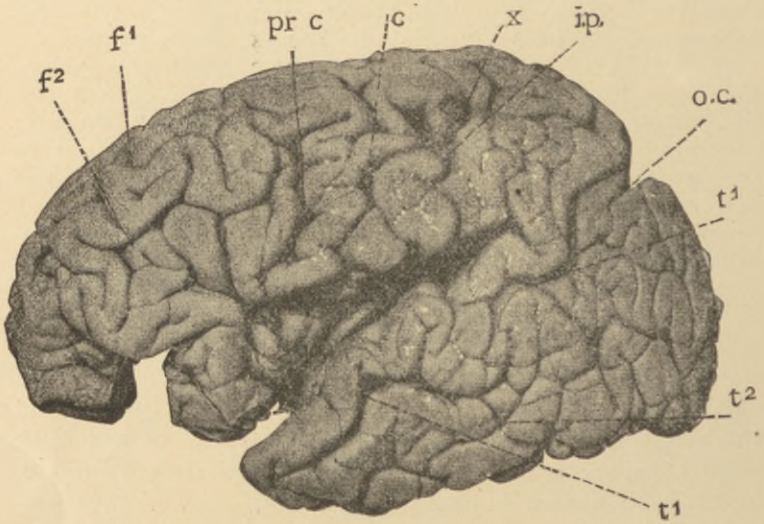
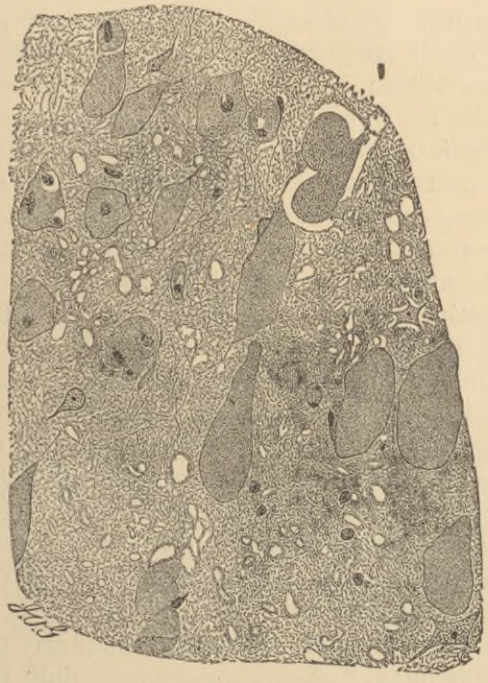


Fig. VIII.—Outer aspect of surface of left hemisphere, showing exposure of the island of Reil. (X) Region from which first block of tissue had been removed. (Cf. this journal, vol. x-14).

In the cases of Kast and Sachs there was no evidence of the agenesis having been due to early intra-uterine inflammation; in both cases there was a mere arrest of development and nothing more. The changes found in the case reported by one of us were limited to the large cells, in the cortex of which there was not a normal specimen to be found throughout the entire brain. There was no evidence of perivascular inflammation and only very slight thickening of neuroglia-tissue, with considerable retardation of development of the white fibres entering the gray matter of the cortex. The changes in the cells and the atrophies of the surface will be seen on inspection of the two accompanying figures. *Figs. VIII and IX.*

* A similar condition has been found by Jensen[41] in a case of idiocy.

The pathology of paraplegia has not been determined. We have but one case with autopsy, that of Foerster,[42] in which case Birch-Hirschfeld found a general cortical sclerosis with probable descending degeneration.* Ross was induced to state, only a few years ago, that he thought the



× 500 diameters.

Fig. IX.—Section of first temporal convolution. Distortion of pyramidal cells. Smaller cells and neuroglia cells distinctly nucleated.

changes of the spinal lateral columns due to traction at birth might be responsible for spastic paraplegia. This is a bare possibility; but the occurrence of idiocy and mental enfeeblement in 9 out of 11 cases of our own and in most of the reported cases, and the frequent occurrence of convulsions, make a cerebral origin much more probable.

The question arises whether it is possible to determine the morbid lesion in any given case, and we submit the

* See note page 301.

following as the result of our clinical and pathological studies:

The form of paralysis is not the most important factor in diagnosis.

Acquired cases of hemiplegia and diplegia, but particularly the former, are apt to be due to the same causes that prevail in adult apoplexies; namely, hemorrhage, thrombosis, or embolism. Meningeal and cortical lesions are more frequent. Absence of convulsions at onset probably points to an intra-cerebral lesion. Occurrence of convulsions at onset points generally to cortical lesions or to embolism anywhere in the brain. In a few cases affections of gray matter lower down in the central nervous system may also be attended by convulsions.

Frequently repeated convulsions after onset point with great certainty to cortical lesion.

Cases coming on after acute infectious diseases have been proved to be due to vascular derangement, particularly to hemorrhage and embolism, but some cases may be due to a polioencephalitis corticalis; the onset with very high fever and convulsions makes the latter more probable.

Traumatism is an important factor in the causation of meningeal hemorrhage during early life and particularly during the act of labor.

Congenital cases of diplegia and hemiplegia may be due to early meningeal hemorrhage and possibly to an early encephalitis.

In acquired and congenital cases of hemiplegia and diplegia we may have either a condition of porencephalus or simple arrest of development. If acquired, there would seem to be the history of slow development of the symptoms without predisposing cause. If congenital, there may be a history of traumatism to mother.

In later life hemiplegia or diplegia associated with large amount of mental impairment and with contractures point to general atrophy, sclerosis, with secondary degenerations, or to a condition of porencephalus, the origin of which it is often difficult to determine.

In conclusion, a word regarding treatment. So far as prophylactic measures are concerned, we wish to repeat our warning to the obstetrician: Hasten protracted labor, for the skillful use of forceps and careful manipulation are less apt to do injury than the prolonged compression of skull and brain in the pelvic canal. In the acute cases the same principles which obtain in the treatment of adult cases should be applied.

Medical advice is generally sought for the relief of the secondary conditions—epilepsy, idiocy, and contractures. In the treatment of the epilepsy the usual remedies may be employed: above all, the bromides and chloral. But these should be administered with the utmost discretion, for in many cases the epileptic attacks are so infrequent that they do far less harm to the patient than the drugs, which increase mental stupor and are very apt to cause anæmia and malnutrition. The proper use of electricity and massage in the early stages will prevent to some extent the formation of extreme contractures. If such contractures exist, we place our reliance upon the orthopedic surgeon, who finds in these cases a very wide field for the exercise of his surgical and mechanical skill. In cases in which the hand is so deformed as to be of less use than an artificial hand would be, amputation would be justified, provided there were no marked rigidity at the elbow. This has been thought of in the case of J. K., whose portrait has been shown. In one case, in this city, with extreme athetosis, the arm was amputated and the patient rendered more comfortable. This should be done only in the severest forms.

The question of surgical interference in cases of hemi-epilepsy deserves passing notice. In very recent cases, in which there is no prospect of spontaneous recovery and in which the character and site of the lesion can be accurately inferred, the idea of operation might be entertained, but it is wholly inadvisable in cases of long standing in which secondary conditions have been permanently established. The result in two cases of Bullard[43] and Mitchell[44] has not been encouraging.

Trepannation for the relief of severe headaches and local epilepsy may be attempted.

As for the condition of mental enfeeblement, much can be done by careful manual and mental training. This should be begun at an early day, and the results will be as satisfactory as in Bourneville's department of the Bicêtre, where the improvement is evident from the change in the facial expression of patients on admission and after they have been under treatment and instruction for some time. In only two cases that we have seen of infantile palsies can we conscientiously state that there has been complete recovery.

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