Thesis for M.D. Degree

by

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INFANTILE HEMIPLEGIA

with

ILLUSTRATIVE CASES.

DEFINITION. An acute cerebral affection of childhood, resulting in incomplete paralysis of one side of the body. This is intended to exclude (1) paralysis of chronic onset, such as is due to cerebral tumour, (2) infantile spinal paralysis, (3) spastic diplegia (Little's disease).

INTRODUCTION. This interesting though not uncommon affection was first brought to my notice some years ago while acting as clinical clerk at Gartloch Asylum. I had at that time the good fortune to be present at the autopsy on a case at which an example of the condition of porencephaly was discovered. I was greatly impressed by the appearance of this brain, and on meeting a case in general practice with similar clinical features, I was led to investigate the subject. After going in to the literature, I was particularly

particularly struck by the paucity of the observations made in the country. In this, as in many other points in connection with pediatrics, the Americans seem to be ahead of us. It is difficult to believe that this is due to the greater rarity of the disease on this side, and I would suggest that if it were more widely known as a distinct affection, much might be done towards a solution of the mystery that overshadows its etiology and pathology.

The history of the study of this affection dates from the beginning of last century. Reil (1812), Caranveilh (1827) and Billard (1821), were among the first to observe alterations in the infant brain, and Breschet (1831), Lallemand (1834), Rokitansky (1835) and Cruveilhier (1849) have referred to losses of cerebral substance in children, but till the time of Little (1853) and Turner (1856), the connection of these with paralysis was not recognised. Heschl in 1859 introduced the term "porencephaly" to express a localised absence of cerebral matter, and Cotard (1868) and Kundrat (Die Porencephalie 1882) have described the condition more fully. Strümpell's famous lecture in 1884 has formed a

a basis for much subsequent contention. In later years, Gowers in this country, and Osler, Sachs and Peterson in the United States have taken up the subject very fully, and their contributions must always remain of considerable value.

As regards the frequency of the disease, it may be confidently stated that though it is not by any means a common affection, many cases of it pass unrecognised, especially outside of hospital practice. It is too much the custom to class all the paralyses of children under the name of "infantile paralysis," and to presume that this in all cases is due to a spinal lesion. The recognition of the possibility of a cerebral paralysis would lead to the differentiation of this from the spinal cases, and a more accurate collection of data. This is forcibly substantiated by the statistics of Peterson[5], who states that in his hospital practice the cerebral infantile paralyses stand to the spinal in the relation of 1 to 2. Granting that cerebral cases are more likely to be sent to hospital, the proportion is still remarkable. In a few years Peterson collected

collected 451 cerebral cases, 332 of which were hemiplegic. Sachs[2] had previously published 225 cerebral cases, of which 156 were one sided paralysis, and Osler[1] has an analysis of 120 cases of infantile hemiplegia. Gowers[4] also quotes from a considerable experience. Those numbers, representing as they do merely the after effects of a previous acute affection, are not quite comparable with the frequency of other diseases, but they at least show that it is not uncommon for a motor cerebral lesion to occur in children. The following cases, collected in a comparatively short period, are fair examples of hemiplegia dating from infancy. The first case I have given in full detail, as it shows all the typical features. In the others all the unimportant negative points are omitted, and the facts stated as briefly as possible.

Case I.

CASE I. Thomas K., age $2\frac{2}{12}$ years, Pollok Street.

Reported 26th May, 1903.

History obtained from mother. When four months old the child had convulsions which lasted for a week. These are described as twitchings, beginning in the mouth and extending to the arm and leg, and confined entirely to the right side. During this week he was unconscious most of the time, but it is not known whether he was feverish. He regained his usual health, and a few weeks afterwards it was noticed that the right arm hung loosely at the side. In a short time it began to be quite stiff and the hand was firmly clenched. At this time also, it was thought that he did not display as much intelligence as he ought to, for his age. When eight or ten months old, the twitchings started again, and were repeated at intervals till six months ago, when he suddenly became generally convulsed; this lasted two or three hours, during which he got "black in the face, and worked all over". He was under medical treatment

treatment for this, and has not had any fits since. In February, he was admitted to the Children's Hospital, where an operation was performed. Since his discharge he has been failing in health, and "spasms" of the body and limbs have been noticed. The mother says that he displays no intelligence whatever, except when food is offered to him.

He was born at full time, there was no trouble during pregnancy, and the labour was remarkably short and easy for a first confinement. He was healthy in every way till the age of four months, and the mother can think of no cause whatever for the disease. A second child has been born since, which is healthy in every respect.

Present Condition. The child is of good size, but his muscles are rather flabby. His abdomen is distended and his face pale and spotty. The appearance shows great mental deficiency. He sits in his nurse's arms, sucking his left thumb, which is eczematous from the effects, and frequently emitting a sound more resembling the roar of a wild

wild animal than a human being. His eyes, head, and left arm and leg are continually in motion, and the movements of the right arm and leg - when these occur - have somewhat of a rhythmic character (mobile spasm). It is impossible to fix his attention, or get him to grasp anything. The face shows a slight flattening on the right side, there being an obvious droop at the corner of the mouth. The tongue is occasionally protruded to the right side. The pupils are equal and react normally to light. There is no squint or nystagmus. The right arm is held extended and rigid by the side, the wrist being flexed and the hand clasped on the thumb. There is no apparent atrophy of the arm compared with the left side. The legs when at rest show no difference, but it may be observed that the right is hardly ever moved, while the other is thrown about freely. The knee jerk and plantar reflex are more easily obtained on the right side, but there is no rigidity of the leg. The heart sounds are normal and there is no evidence

evidence of intrathoracic disease. Ophthalmoscopic examination reveals a slight pallor of the disc on the right side.

On enquiry at the Children's Hospital, Mr Parry tells me that at the operation he raised a horse-shoe flap, and exposed the left motor region. This was found to be occupied by a cyst, which was incised. On further examination a large defect in the brain was discovered, which Mr Parry considers to be a porencephalic condition. The wound was stitched and healed without any trouble.

Remarks. This is a very typical case, and there can be no question about the diagnosis. There is no circumstance which would lead one to think the condition originated before the fourth month.

CASE II.

CASE II. James B. Age 10 years.

Reported in Children's Hospital 12th Sept., 1903.

History from mother. There was no trouble during pregnancy or at birth. Child was perfectly healthy up till five years of age, when he had general convulsions. Immediately afterwards it was observed that he could not use his right hand. This soon became drawn up, and he walked strangely. Convulsions were repeated frequently. He had pneumonia and pleurisy last year, and was apparently worse after it. On enquiry, mother says that he sometimes was sick and complained of headache.

Present Condition. Right hemiplegia. No facial paralysis, but want of expression on right side. Tongue tends to be protruded to the left. Hand and arm held flexed across chest. Slight rigidity about the elbow, but most marked in flexors of hand, which is clasped over the thumb. Slight power of flexion, but power of extension of hand and wrist is gone. There is no permanent contracture in the arm.

Choreiform

Choreiform movements of right hand and arm, especially when boy is excited. Talipes equinus of right foot, which can be overcome with continuous pressure, but no further than a right angle. Knee jerks both exaggerated, but response is quicker on right side. Slight ankle clonus on the left side. Superficial reflexes all exaggerated on the right side, and Babinski's sign is present. The fundi oculi are normal except for a slight paleness of the right optic disc. The boy is of fair average intellect.

Measurements.	Right.	Left.	
Head (measured from glabella to occiput)	10 3/8	10	inches.
Humerus	9	9¼	"
Ulna	7	7½	"
Girth of Forearm	6 3/8	7	"
Leg	24	24¾	"
Girth of Calf	9½	10	

Remarks.

Remarks. In this case there was a question of cerebral tumour, but the symptoms of headache and sickness were absent during his residences in hospital, and there was no marked alteration in the optic discs. The other points are much in favour of an acute hemiplegia.

CASE III. Ann H. 17 years. Gartloch Asylum.

Reported 3rd June, 1903.

History from Mother. There was no trouble during pregnancy or at birth. Child was quite healthy and normal till three years of age, when, while playing one day, she suddenly became generally convulsed, with twitching all over right side and unconsciousness.

Fit

Fit lasted 13 hours. Gradually recovered in eight weeks and paralysis was noticed then. She also was blind in the right eye (? hemianopsia) and her speech was thick and difficult. Sight was recovered in twelve months. After this her intellect was very poor, and she would sit constantly at the fire, her left hand working in strange contortions. At eight years of age she began to have epileptic fits and she has had them frequently since. She is an illegitimate child, born, before her mother's second marriage, of an unknown father. There is no history of syphilis on the mother's side.

Present Condition. Mentally she is an imbecile, and talks childishly. There is no facial paralysis. Right arm is held flexed over chest with thumb turned in. Marked rigidity in all the joints of this limb and permanent contracture of biceps. Right leg also rigid, and extreme talipes equinus which cannot be overcome. Knee jerk markedly increased on right side though both are exaggerated. Ankle clonus on left side. Not much atrophy of muscles on right side.

Right

Right hand and foot colder and cyanosed. Internal organs normal. No involuntary movements present.

Measurements.

Head	$\frac{5}{8}$	inch less on left side.
Ulna	$\frac{1}{2}$	" " right side.
Leg	$1\frac{1}{2}$	" " "

Remarks. The history was easily obtained from the mother, who was very definite about the onset and other details.

CASE IV.

CASE IV. William C. 30 years. Gartloch Asylum.

Reported 10th June, 1903.

History from mother. There was no trouble during pregnancy or at birth. Healthy till five months old when he had severe bronchitis. About third or fourth day of this, convulsions started - twitching all over right side, with unconsciousness. Lasted for four days with intervals, and for weeks after, he was comatose, occasionally brightening up. Paralysis noticed at this period. Occasional general fits, becoming much worse at five years of age. Always dull in intellect.

Present Condition. Slight droop on right side of mouth which disappears on smiling. Hand usually kept over chest, but

but he can perform all movements, though very feeble. Very little resistance of extension, but slight permanent contracture of biceps. Hand is small and the skin thin and transparent. No rigidity about the leg. Extreme talipes equinovarus. Knee jerk easily obtained on both sides, but markedly exaggerated on right. No ankle clonus. Mentally he is very weak-minded. He is epileptic, but only one fit has been observed in the Asylum and this was general. No choreic movements have ever been seen.

Measurements.

	Right.	Left.	
Head	12	11½	inches.
Ulna	9½	10	"
Humerus	12¼	13¼	"
Girth of Forearm	7½	8	"
Tibia	13	14½	"

Remarks. The chief points about this case are the absence of any spastic condition of the muscles, and the extreme difference in the length of the limbs. Epilepsy, which seems to have been severe when he was younger, has disappeared almost entirely.

CASE V.

CASE V. Martha M. 16 years. Gartloch Asylum.

Reported 8th September, 1903.

History from Eldest Sister (30)

Prolonged labour with forceps. No illness during infancy, and no convulsions ever observed. A few weeks after birth left hand observed to be weak and stiff, and some time after that, the leg condition was also observed. Fits started at seven years of age and were always general. Attended school and intellect was always good till lately when she became rather stupid and bad-tempered. Fits are more frequent now and she has at times four or five in the day.

Present Condition. Anaemic, but well nourished girl. No facial paralysis. Left hand and arm kept rigidly flexed over

over chest and there is some resistance to movement. There is voluntary power of movement of all the joints though this is very much diminished comparatively, and especially so at the wrist. Slight rigidity in the leg. The foot is in a condition of talipes equinus, but can be forcibly flexed to a slight degree. Hand and foot colder and blue. No involuntary movements. Knee jerk markedly exaggerated on left side, but no ankle clonus. Her intellect is somewhat dull, but she speaks sensibly.

Measurements.

Head	fully ½	inch greater on left.
Ulna	1	inch less " "
Girth of Forearm	½	" " " "
Tibia	1	" " " "
Girth of Calf	1	" " " "

Remarks. There may be some dubiety about the sister's story, but the probability is that this case is a congenital or birth case. It is worthy of remark that this is a left-sided hemiplegia.

CASE VI.

CASE VI. Robert T. (33 years). Gartloch Asylum.

Reported June, 1903.

Mother dead. Father cannot be found.

History from Journal. Soon after birth he had convulsions lasting a week. No fits since. Paralysis noticed at the age of one year.

Present Condition. Slight facial paralysis more noticeable when face is at rest. Right hand held voluntarily over chest, but no rigidity about the arm. He has very little power in the hand which has a "griffin" deformity, being flexed and adducted at the wrist, hyper-extended at the metacarpo-phalangeal joint and flexed at the fingers. There is no rigidity in the leg, but an extreme talipes

talipes equinus exists and there is no movement at the ankle. The right knee jerk is exaggerated. There are no choreic movements, but the right hand attempts to imitate the movements of the left. Mentally he is a high grade imbecile and very docile.

Measurements.

Head	¼ inch less on left side.
Arm bones	equal.
Tibia	1½ inch less " "
Girth of Calf	3 inches " " "

Remarks. This case probably dates from the first year of life. Epilepsy is absent and the mental defect is not very extreme.

CASE VII.

CASE VII. Graham S. 28 years. Gartloch Asylum.

Reported June, 1903.

No relatives known, and history cannot be obtained, except that he has been imbecile and paralysed since infancy.

Present Condition. Arm is held in typical position, but there is only a slight paresis and no rigidity. Facial paralysis is more marked in this than the other cases. There is a talipes equinus partly spastic in character. The right knee jerk is markedly exaggerated. His movements are erratic, but not choreiform. Mentally he is quite idiotic and resistive to examination. He cannot speak and hardly understands anything that is said to him. He is not epileptic.

Measurements.

Measurements.

Head	¼ inch	less	on	left side.
Girth of Forearm	¾	"	"	right "
Leg	½	"	"	" "
Girth of Calf	2 inches	"	"	"

CASE VIII. Harry D. 13 years. Middlesbrough.

Reported Feb. 1903 and later.

History from Mother. During pregnancy mother had a fall downstairs, but went on to full term. Child was born feet first, but the labour was easy. The hands were stiff for some days after. When five or six weeks old he suffered from bronchitis and this continued till he was five months old, when convulsions of the right side started, and he was unconscious for some hours. Convulsions repeated when one year old, but none since. Paralysis of right side noticed immediately after first attack. Recovered power in arm, but

but has always been left-handed even in writing. Late in walking (over 3 years) and talking (between 4 and 5).

Present Condition. Hemiparesis of right side. Slight facial asymmetry in certain positions. No rigidity in arm and all movements perfect though weaker comparatively. Slight rigidity in knee, and talipes equinas which can be overcome to a right angle. Knee jerk slightly increased on right side. No ankle clonus. Babinski's sign is present. No involuntary movements. Mentally he is quite normal and can read and write well.

Measurements.

Head	½	inch	less	on	left side.
Ulna	¼	"	"	right	"
Girth of arm	½	"	"	"	"
Tibia	½	"	"	"	"
Girth of thigh	⅜	"	"	"	"

Remarks. The fact of the hand being least involved is not typical of a cerebral affection, but the history and symptoms conform to the usual characters.

CASE IX.

CASE IX. Minnie M. (14 years) Middlesbrough.

Reported 31st August, 1903.

History from Father. Healthy when born. About one or two months old, general convulsions started, with unconsciousness, fever and vomiting. Repeated at intervals till twelve months old. On attempting to walk at two years old, right leg was dragged behind. No weakness or stiffness of hand or arm noticed. Walked with crutch till lately. Until five years ago had no sensation in right leg. Always intelligent.

Present Condition. No facial paresis. Right hand and arm slightly weaker and thinner. Right leg much wasted - talipes equino-varus, quite flaccid except for permanent contracture of calf muscles. No voluntary movement except extension of toes. ~~Right~~ Knee jerk completely absent on right side and diminished on left. Leg cold and blue.

Measurements.	Right.	Left.
Head	Equal.	
Ulna	9¼	9⅜ inches.
Girth		

	Right.	Left.
Girth of Forearm	7¼	7 5/8 inches.
Tibia	13	15 "
Girth of Calf	7¾	10 "

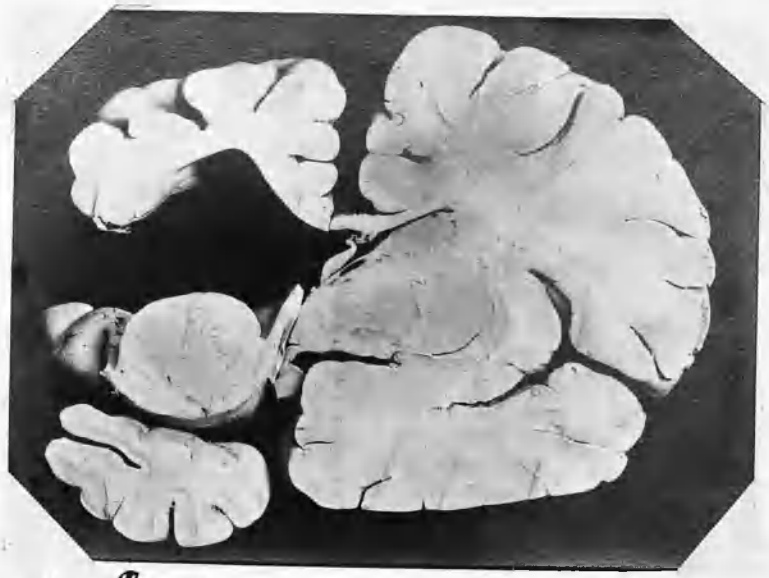
Remarks. This is an undoubted anterior poliomyelitis and I have included it to show the resemblances and also the differences between it and the cerebral cases. The next two cases are reported for the sake of the pathological lesions present.

CASE X. William H. 35 years. Gartloch Asylum.

Died October 1899.

History. Little is known except that he was imbecile and epileptic from infancy. He used to say that his sister had let him fall when a baby, and that was the cause of the paralysis.

Clinical Features. As I remember him he had hemiplegia of right side with contractures. He had occasional fits and was imbecile. He died of double pneumonia.



Coronal Section through both Hemispheres, showing relation of cavity to lateral Ventricle and atrophied corpus striatum (left)

Lateral Aspect of Left Hemisphere showing porencephalic area and atrophied convolutions

At the post-mortem, there was found a cyst in the left side of the brain, the outer wall of which was formed of thickened membranes. On opening this, clear fluid escaped, and a cavity was found extending inwards and communicating with the lateral ventricle about its anterior end. Ependyma was continuous with cavity. The extent of the deficiency may be seen from the photograph. The third frontal, Broca's, and lower parts of ascending parietal and frontal convolutions are absent and atrophy extends over first temporal and third parietal and the Rolandic gyri. Island of Reil is not to be seen and caudate nucleus is absent though lenticular nucleus was found on cutting sections. The atrophied convolutions are hard and rough on the surface, and depressed below the level of the others. Atrophy may be traced through the left crus and pyramid to the lateral tracts of the cord. The cerebellum is equal on both sides.

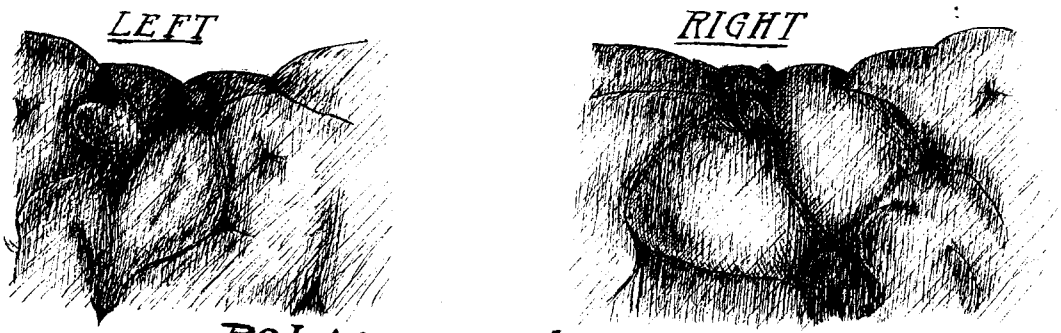
Remarks. The convolutions of the anterior margin have a radiate arrangement which, according to Kundrat, is a sign of foetal origin. It is unfortunate that the history and clinical

clinical features are deficient, but the association of the lesion with infantile hemiplegia is undoubted.

CASE XI. Agnes Paterson. (52 years) Gartloch.

Died April, 1900.

Clinical Summary. I did not see this case during life. She had been insane for 26 years. There was deficient development of both hands, but especially the left. The left leg was also defective, but less so than the arm.



ROLANDIC AREAS

The brain shows an atrophy in both motor areas, but more marked in the right. The affected gyri are shrunken and hard, and depressed beneath the surface. These were the only regions affected.

Remarks.

Remarks. This is not a true infantile hemiplegia, but it shows the localized atrophy and sclerosis which is a common lesion in that affection.

I shall now pass to the general consideration of the disease.

ETIOLOGY.

(1) Age. It is extremely probable that a good many cases of hemiplegia are congenital in origin, though it is difficult to say exactly how many. It is said that paralysis in an infant may exist without being noticed by the mother till it is at least a year old, but I am inclined to doubt this. I have found mothers very observant of physical defects in their children. It is also stated that paralysis from an intra uterine cause may not develop till the child is some months old, and this point will be discussed later. Osler says that only five out of his 120 cases were truly congenital, while Sachs and Peterson, from

from 140 cases considered 49 congenital. From my own cases I can draw no conclusion, except that in Cases V, VIII and X the question of intrauterine origin has to be considered. After birth the frequency is greatest in the first year and diminishes up to the sixth year after which it is comparatively rare till mature age. Thus, Osler gives 45 in the first, 22 in the second, and 28 between the third and tenth years. Gowers says three-fifths of the cases begin in the first three years, and Sachs finds two-thirds in the same period. Of my seven cases with authentic records, four began in the first year, one at three years and one at five years.

Immediate Causes must be indefinite, as there is a certain amount of fallacy in attributing any causative action to the common affections of childhood. There are, however, a great many precedent conditions which are said to be possible primary elements. Accident or fright to the mother during pregnancy, Osler has found in two cases, and difficult

difficult or instrumental labour in nine. Two of my cases would come under this category. Injury to the infant he has elicited in three cases. Pulmonary and infectious diseases are accountable for 16 cases, and infantile convulsions for five. Sachs gives pneumonia in six, trauma in six, infectious diseases in 25, and convulsions in 20. Endocarditis is said to be very rare and syphilis is quite negligible. Primary disease I have found in only two cases, and this was bronchitis. According to Gowers, in the majority of cases the attack is not distinctly secondary, and I think we may take it that, as a rule, it is not a sequel to any special affection.

SYMPTOMS.

SYMPTOMS. If there is much variation in the facts bearing on the etiology of the disease, there is still a great similarity in the symptoms of the onset, and although not absolutely pathognomonic of this affection these certainly point in most cases to an acute cerebral lesion. The child may be lying in its mother's arms, or playing about in perfect health, when it is suddenly seized with convulsions and becomes unconscious. The convulsions are in most instances clonic, and one sided, involving face arm and leg, though some have been reported in which the convulsions were general and of the ordinary type of infantile eclampsia. The unconsciousness may vary from a deep coma lasting for hours, to a slight hebetude. There is often some fever and vomiting lasting for a day or two, but owing to imperfect histories, writers are naturally not in agreement as to the frequency of these symptoms. Thus Osler lays no stress on the temperature, while Sachs and Peterson say that a slight fever is the rule. Whether these symptoms

symptoms are the evidence of a general toxaemia or the result of the cerebral disturbance is a point to be considered afterwards. The coma and convulsions may continue with lucid intervals for a variable period, usually some days. The date at which the paralysis is observed depends on the intelligence of the mother, and on the age of the child. In most cases it is noticed soon after consciousness has returned, but in very young babies some weeks may elapse before it is discovered. There are many exceptions to this form of onset. In a few there is no definite onset, the paralysis being observed soon after birth, - as in Case V.- or it may be the first indication of anything wrong with a previously healthy child. Taking the cases as a whole, it is exceptional for death to occur during the acute period. The convulsions cease, consciousness returns, and the child is left with a one sided paralysis. The chronic stage which then sets in, due to degenerative changes in the nervous system, is best described by taking the symptoms separately.

Paralysis.

Paralysis. The degree of paralysis varies very much. It is difficult to get data of recent cases, but I think the rule is for the face, arm, leg to be affected at the beginning. In a few cases perhaps, the face may escape, but when it is involved it is always on the same side as the other parts. Broadbent's law seems to apply to infantile as well as to adult hemiplegia viz., that those movements which are performed in association with the other side, are least affected and soonest restored. That is to say, the hand and arm suffer most, the leg next and the face least of all. In all cases there seems to be no further advancement of the palsy. As in the adult form, recovery takes place to a certain degree. In some the hemiplegia passes off altogether, and Gowers is of opinion, that in these cases the lesion is not in the motor tract, but in some other region of the cerebrum; the paralysis being the result of the general disturbance at the onset; this seems extremely probable. The return of a certain amount of power to the affected limbs, is most likely due to the property

property that one hemisphere has of taking up the function of the other. It has been proved by Muratoff, that excision of one motor cortex results in some secondary degeneration in the tracts in the same side of the cord. Proof of this is found in some of my cases which have exaggerated reflexes on the unaffected side. In a child, before specialisation of movement has been learned, this non-decussating tract may be more highly developed, and to some extent compensate for the loss of the other.

In the majority of cases, however, there is a certain residue of paralysis and it is from these cases that most of the statistics have been gathered. This hemiparesis results in a characteristic attitude and gait. The useless arm, kept flexed across the chest, and the deformed foot dragged behind or scraping the ground when walking, mark the case at once as an old hemiplegia. As regards the side which is affected, observers are agreed that the right and left are nearly equal in frequency. Thus

Sachs.....	R.H. 81	L.H. 75
Osler.....	R.H. 68	L.H. 52
Diller[13].....	R.H. 3	L.H. 3

The

The proportion in my cases is R.H. 8 and L.H. 1, but in the face of the fuller statistics, no conclusion may be drawn from this.

Atrophy of the Limbs. I can find no published statistics of this but it seems to be generally recognised. Taylor[7] calls special attention to it. I have found it very marked in all cases, and I should consider it an important feature in distinguishing a case beginning in early life. The difference in size is most marked in those dating from the first year. It is, of course, not a true atrophy, but a defective development of the limbs from disuse, and is easily distinguished from the rapid wasting in acute anterior poliomyelitis. The bones are shorter and thinner, and the muscles are likewise diminished in bulk, although when a spastic condition prevails, they may be well developed or even hypertrophied (as in Case III). The hand suffers most of all. It is smaller and the bones project beneath the thin clear skin.

Asymmetry

Symptoms contd.

Asymmetry of the Skull has been found by Peterson and Fisher[5] in the majority of cases. My measurements show this to be remarkably distinct. In every instance the circumference on the side opposite to the paralysis was less than the other by between $\frac{1}{4}$ and $\frac{5}{8}$ inch. It will vary according to the extent of the cerebral lesion and the age at onset.

Contractures and Deformities. I have mentioned previously the characteristic attitude of the paralysed hand and arm. The upper arm is kept close to the side, the elbow flexed, the forearm crossing the front of the chest and the hand pronated and clasped on the thumb. In one of my cases, and in a case reported by Shirres, there was a "main en griffe" due to a greater involvement of certain sets of muscles. This condition, however, is not altogether due to involuntary contracture or spastic rigidity of the muscles. In some cases the patient can extend all the joints freely. In others there is a certain amount of voluntary movement, but when one attempts to aid this by external force, there is some resistance. With steady pressure this can be overcome till a point is reached when movement is completely arrested. This marks the permanent shortening of the muscle. The rigidity which is felt on passive movement is

is due, of course, to loss of function in the pyramidal tracts of the cord. The motor cells of the anterior cornua, being free from the controlling influence of the higher centres emit an excess of tonic energy to the muscles, an energy which is probably derived from the sensory tracts and completes a reflex arc. This spastic condition is by no means pathognomonic of infantile hemiplegia, although it has received the name of spastic paralysis in contradistinction to the flaccid condition in anterior poliomyelitis. Contractures owing to paresis, are usually said to be due to the greater strength of the flexors over the extensors. It is difficult to disprove this, but I think that a more probable explanation is, that the attitude is at first one of convenience, to keep the useless hand out of the way. The flexors in this position have a mechanical advantage over the extensors, and with the increased tonicity, the flexion is maintained involuntarily. In time also, there will

will be an actual shortening of the muscles resulting in a permanent contracture.

In the lower limb the constant deformity is talipes equinus, and in some cases a certain amount of varus is added. This again, I maintain, is not due primarily to spasm of the calf muscles. To begin with, in a healthy infant the natural position of the feet at rest is one of equino-varus. When one leg is paralysed this position is maintained, and in this gravity will play an important part. Later, when rigidity sets in, it becomes impossible to flex the ankle fully, and in many cases there is no movement whatever. When the child begins to walk, the extension becomes even more extreme to compensate for the shortening of the leg. In some of the severe cases I have noted, the metatarsus is in a vertical line with the tibia, adding as much length to the leg as possible. Contractures of the knee and hip joints are not at all common.

The following table of contractures of the joints in hemiplegia

hemiplegia is given by Sachs.

Flexion of Elbow.....29

" " Carpus and Fingers....23

" " Knee.....5

Extension of Knee.....1

Talipes Equino Varus.....17

" " Valgus.....3

" Equinus.....2

Post-Hemiplegic Movements: Certain involuntary movements of the affected limbs are often observed, and they are almost characteristic of this affection. The most common are of three classes.

(1) Athetosis. This is defined as a regular rhythmic movement of the hands and fingers.

(2) Chorea is distinguished from the former by being more jerky and irregular.

(3) Tremor - of the character of paralysis agitans.

Osler has observed athetosis in 6 cases, chorea in 24, and

and tremor in one. Griffiths[13] has described two cases of unilateral tremor in children, one of which was post-hemiplegic, and Diller[13] notes 3 cases of athetosis. I have observed choreiform movements in two cases (Cases I and II) and there is a story of contortions of the hand in another (Case III). Sachs mentions ataxia and associated movements (i.e. the paretic hand imitating the voluntary movements of the other) but I have not seen this with any distinctiveness except in one case (Case VI):

Reflexes. The superficial and deep reflexes on the affected side are usually increased, and this is especially the case with the knee-jerk. Ankle clonus is usually out of the question owing to the contracture, and the triceps and wrist reflexes are likewise not to be obtained.

Babinski's sign I have observed in two cases. I have already mentioned that the reflexes on the sound side are increased in some instances. This point I have not seen mentioned

mentioned in the books.

Electrical Reactions. I have not been in a position to examine but they are said to be normal in all cases.

Sensation is also usually perfect.

Coldness and Lividity of the affected limbs I have noted in four cases and it is mentioned by Osler and others. It is said to be a vasomotor disturbance, but as it is not transitory, it is difficult to see how this can be. In all the cases there was a spastic condition in the muscles, and it might be that these by pressure on the deep vessels, caused a stagnation in the superficial veins. It is difficult to explain otherwise.

Aphasia has been seen in 17 cases by Sachs and 13 cases by Osler. An interesting point is that it may occur independently of the side affected. This shows that in children the speech centre has not become localised to one side of the brain, as it is found later. Aphasia is a temporary affection and will only be observed in those children who have already learned to speak. I have not seem it in any of

of my cases but there is a story of difficulty of speech in Case III.

The foregoing symptoms are the result of a localised lesion in the motor region of a cerebral hemisphere, and its secondary effects on the motor tracts, but unfortunately the lesion seems to gradually to affect the whole hemisphere with disastrous results for in a majority of cases the patients become the victims of epilepsy and mental degeneration.

Epilepsy. Although this is not the true primary epilepsy, the convulsions are in all respects identical. As a rule they are general. In a few exceptional cases however, they may begin on the affected side, or there may be an aura referred to that side. The attacks may start soon after the hemiplegia, or it may be years after, and when the patient gets to adult age they may cease altogether. They vary much in frequency and severity. In some of my cases there were only one or two general convulsions altogether, while in others they were as frequent as 14 in a day sometimes. Six

Six of the nine cases here recorded suffered from these seizures. Sachs gives the proportion as 50% of hemiplegic children and Osler and Gowers state that it is very common. Mental Defects are a fairly common sequela of infantile hemiplegia, and there is certain to be a large number of cases under institution treatment. In one asylum alone I was able to get five cases resident at the same time. A large number of course, will be kept at home. In only one instance (Case VIII) did I find a patient with a fair intelligence, and Sachs says that 13% of all cases suffer from intellectual deficienty, though the proportion is doubtless much larger. The degree of weakness will vary very much. The patient may be rather dull or stupid (as in Cases II and V), especially when he has been having fits, or he may be childish and simple minded, and unable to fight life's battle for himself (Case VI) Many however, are still lower in the scale and lack the power to converse or understand or even the capability of being educated to attend to their natural wants. They never regain their intellectual faculties, and spend their useless lives as a burden to society, and objects of pity charitably to the more minded.

MORBID ANATOMY.

The presumption that these symptoms are produced by a cerebral lesion, is fully verified by post mortem results, but as to the character of this lesion there is little evidence and necessarily some diversity of opinion. The reason for this is not hard to seek. Only in a small minority of cases do the patients die during or shortly after the acute attack and when they do so , the hemiplegia has not shown itself. As it is not a chronic advancing condition, all that remains in after life is merely the deformity caused by the destruction of the cerebral tissue. Out of 90 autopsies Osler could only find 16 of recent origin and Sachs reports 35 recent cerebral lesions. Admitting a low mortality in the early period, it cannot be assumed - as many observers do assume - that the lesions found in the fatal cases are typical of the disease. Indeed, one might go so far as to say that there may be other lesions less fatal, producing the same symptoms, and the same permanent effect on the brain.

The

The majority of recent lesions have been found to be vascular, and include haemorrhage, thrombosis and embolism. Haemorrhage is the most common. Osler quotes 9 cases, and Sachs has found 23. All these however, are open to some criticism. Osler's cases, for example, are all, except one, taken from literature. Three are congenital and attributed to injury to the mother during pregnancy, the children being probably still-born. One is due to an aneurysm bursting into the longitudinal fissure (his own case), four have no details as regards situation etc, and one was a child of six months in which the blood was poured into the lateral ventricles. With the exception of the last case and the congenital ones, the patients were all over six years. Clearly these cannot be called typical cases.

Sachs in his book on Nervous Diseases of Children discusses the three forms of cerebral paralysis together, and it is not easy from his statistics to discriminate any one form from another. If we admit that spastic diplegia of infants is as a rule the result of meningeal haemorrhage at birth - and this has been well proved by Sarah M'Nutt and others,- it is necessary to discard some from the number that

that Sachs submits. In his article on Cerebral Haemorrhage, he gives 83 cases as due to the following causes:-

Convulsions	20
Pneumonia	6
Trauma	6
Infectious diseases etc	25
Unascertained	26

These however, are not post mortem results, and there is no note as to the ages of the patients or the clinical features. Gowers gives the following table showing the liability to haemorrhage at different ages:-

	1-10 yrs	11-20 yrs	21-30	31-40	41 and over
per 1000	1.8	3	6	12	19

This obviously does not correspond with the frequency of hemiplegia.

The fact of haemorrhage being a cause of infantile hemiplegia

hemiplegia cannot be gainsaid, though it cannot be proved that it is a constant lesion.

As regards the situation of the haemorrhage, Sachs makes it pretty clear that it is in most cases on the surface of the brain, and from a branch of the middle cerebral artery. In some cases it is not very certain that it is not the middle meningeal artery, as some authors use the term "meningeal" and others "cortical." In traumatic haemorrhage cases, the middle meningeal would be most liable to injury. The blood collects in the Sylvian fissure, and presses on the brain substance. It extends in a layer along the pia-arachnoid, and in later stages a cyst may be found containing a grumous fluid and lined by a false membrane. Cases are reported also of clots or blood cysts in the basal ganglia, internal capsule and pous, but these are isolated cases and probably rare.

Embolism has been found in some cases. Osler quotes 6, five of which occurred in patients suffering from endocarditis and all these were above six years. Sachs in one instance 3 gives

gives three cases (1 yr, 2½ and 6 yrs respectively) in which an embolus was found in Sylvian artery and in the other seven cases are mentioned.[2] Abercrombie[15] describes a case with embolism and cortical softening in a child of three years. These cases cannot be questioned, except that as some of them are a complication of endocarditis they approach more to the adult type.

Thrombosis. As Gowers points out, it is often impossible to tell at an autopsy whether an arterial obstruction is primarily embolic or thrombotic. Osler quotes one case of thrombosis and Sachs five. Gowers gives a detailed description of a case of thrombosis in a vein leading into the longitudinal sinus, and argues that this may be commoner than is suspected, as there is a tendency for the thrombus to slip into the sinus.

Amongst other recent post mortems, I have been able to find two which differ from the foregoing. The first is one of Batten's[14] in which a boy of nine died in three days from the onset of hemiplegia. The paralysis was the first

first symptoms and he gradually became comatose. At the autopsy the left motor cortex was found to be softened, and microscopically there were found thrombosis of the smaller arteries with perivascular exudations. No lesion of the larger vessels was found. The second is mentioned in Holt's book[6], and is that of an infant who died of pneumonia some days after developing hemiplegia with convulsions. No gross cerebral lesion was found at the autopsy. The abnormal appearances found in after life indicate a localised absence of cerebral substance, and are described as (1) atrophy, (2) porencephaly.

Atrophy. By this is meant a diminution in size of certain of the cerebral convolutions. It may affect a region of the hemisphere or a single convolution, or it may be distributed irregularly over the surface. The affected gyri are narrower and shallower, being depressed below the level of the others, and the membranes covering them are often thickened and adherent. The cerebral tissue is hard to the touch, and on making sections for microscopic examination, it is found that the grey matter is thinner than normal and that there is

is an excessive development of neuroglia. The layer of pyramidal cells is the structure that suffers most. It may be entirely absent or represented only by a few atrophied cells. There is a corresponding decrease in the fibres in the white matter. Osler describes a layer of minute cysts sometimes seen at the junction of white and grey matter, and states that haematoidin has been found in their walls.

The convolutions of the motor region are most frequently affected, but atrophy is also seen in the parietal and occipital lobes. I distinctly remember a case in which the convolutions at the base of the occipital lobe were much shrivelled, and the white matter was so much atrophied that the posterior horn of the lateral ventricle came almost to the surface.

Case XI shows atrophy of the Rolandic convolutions on both sides with a corresponding lack of development in the limbs. Osler has collected 50 cases of cerebral atrophy and Sachs has seen 40.

Porencephaly

Porencephaly. This name was first introduced by Heschl in 1859 to express a loss of cerebral substance, or literally a hole in the brain. It is not a rare condition by any means. Osler has collected 24 cases, 6 of which are congenital and 11 have a history of beginning in early life with symptoms of hemiplegia. Audrey collected 100 cases from literature and added 3 of his own. Of these 90 had full anatomical details. He found it bilateral in 32, left sided in 38 and right sided in 20. He attributes it to various causes usually acting during foetal life. I have been able to collect from recent literature, 7 cases described in detail. The clinical history however, in nearly all of them is defective. I have put the main points in tabular form:-

	8a Norman	9 Edgerley	8 Clinch	11 Wigglesworth	11 Wigglesworth	10 Barratt	12 Shirres	Case X	Case I
Age at Onset of Symptoms	Unknown	Unknown	11 months	Unknown	Unknown	11 mos.	Birth	Infancy	4 mos
Paralysis	Right	Right	Right	Right Paresis	Left	Right	Right	Right	Right
Epilepsy	-	Yes	Yes	Yes	Yes	Yes	No	Yes	Yes
Mental Defects	Yes	Yes	Imbecility	Slight	Idiocy	Imbecility	Slight	Imbecility	Idiocy
Part involved	Motor Region Corp. Striata	Motor Region Corp. Striata	3rd Frontal to Angular Gyrus	Lower part of Rolandic Fissure	Parietal Occipital and Temp. Lobes	Motor Region	2nd & 3rd Frontal Rolandic & 1st Temporal	-	Motor Region
Surrounding Atrophy	Yes	Yes	Island of Reil	Slight	Yes	No	Yes	Yes	-
Communication with Lateral Ventricle	Yes	Yes	No	No	Yes	No	No	Yes	-
Cerebellum atrophied	-	Right	-	Right	Left	No	Right	No	-
Radiation	Yes	No	Slight	Yes	No	No	No	Yes	-
Theory	Congenital	Arterial Obstruct.	Arterial Obstruct.	Foetal Disease	Arterial Obstruct. or Trauma	Embolism	Foetal Embolism	-	-

Taking all the cases in review, it is seen that there is no uniformity in the area of surface involved. The centre of the defect however, is always about the situation of the operculum, and the cavity may extend forwards, backwards, upwards or inwards in various degrees. In the fresh brain it is filled with a clear fluid and strands of tissue sometimes cross the gap. The membrane covering it is occasionally thickened and distended to appear like a cyst. It seems pretty obvious that the hole is formed in the following manner. Certain of the convolutions and a corresponding amount of white matter have been undeveloped destroyed or atrophied and their disappearance has opened up the fissure of Sylvius. The arachnoid space has then distended in cystic fashion to fill up the cavity. If the lesion has affected the Island of Reil and the basal ganglia the lateral ventricle has protruded itself in to the cavity which then becomes lined by ependyma, the atrophied Island of Reil being represented in the outer wall. Many of the surrounding convolutions are small and shrunken and in some there is a striking radiate arrangement of the unatrophied gyri

gyri round the opening. This was noted by Kundrat and he considered it characteristic of the congenital cases. The explanation seems to be that the cerebrum falls in on the space and the well formed gyri become folded on themselves in an undulating outline. This I should think would take place after, almost as easily as before development of the infant brain. I do not think there is anything about a porencephalic brain that will distinguish it as being of congenital origin.

The affected hemisphere is always much smaller than the other, and there is probably a slight amount of general sclerosis present. The different regions of the brain communicate so freely that there must be some secondary degeneration among all the tracts. Atrophy of the motor tracts has been traced in most of the cases, through the pons and pyramid to the lateral tracts of the cord on the opposite side. The basal ganglia are usually atrophied. In some cases the caudate and lenticular nuclei are nearly if not entirely absent.*

*(Case X)

(~~Case I~~) The optic thalamus suffers to a less extent. An interesting point is that in some cases the cerebellum is atrophied on the opposite side, while in others it is normal, but I can offer no explanation of this variation.

As regards the age at which porencephaly is found, I can find no cases reported younger than Case I - in which it was discovered at operation, - with the exception of a double porencephaly of the frontal regions in a case of Schultzes described by Sachs. It was said to be congenital and the child lived for five months.

Osler is of the opinion that porencephaly is to be considered as an extreme degree of the condition described under Atrophy, and that is probably due to a similar primary lesion. As a frequent occurrence in infantile hemiplegia it is undoubted although it cannot be diagnosed before death. Sachs, indeed, says that it is impossible to tell before the autopsy what the condition of the brain is, as regards the extent of the lesion.

PATHOLOGY

PATHOLOGY

The chief interest in the consideration of this affection centres round its pathology. Opportunities of examining the primary condition are rare, but there is no lack of theories to account for it, and this has led to much controversy.

The first question to be considered is whether it is a distinct disease, always due to the same cause and having the same pathology in all cases. After consideration of all the evidence we must unhesitatingly answer in the negative. It varies in its etiology, its clinical features and its anatomy, and we must expect it to vary in its pathology. As in the adult form of hemiplegia there are different lesions, so there must be in the infantile. We can only say that there is a destructive process in the motor region of the cerebrum, and we cannot say that this in all cases is identical. In certain rare cases, the pathological factor is easily recognised to be the same as in the adult types, as for example, embolism in endocarditis, but for what I might call the typical case, with

with an acute onset in early infancy, ending in recovery and passing on to epilepsy and idiocy, there is yet no adequate explanation.

The etiology does not help us much. An important point is that it is almost limited to the first three years of life, indeed I should be inclined to neglect those cases over six years, as they are rare and approaching to the adult type. We must expect therefore, some cause that is predisposed to by the ages of the patients. The normal infant brain, although in gross structure it is practically the same as the adult, is undergoing development of the minuter elements. The motor region is complete about a month before birth, but, according to Cunningham, for some time afterwards there is a specialization of function going on, which leads to changes in the region of the operculum. Naturally there will be a rapid reproduction of new cells and the vascular supply will be abundant, and it may be the small vessels will not be in a fully developed state. Young children, also, are specially liable to injury, and to general diseases, to which hemiplegia is said

said to be a sequel. I have already pointed out that this is apt to be fallacious, as children are so often ailing that the question of "post" and "propter" is not easy to decide.

The symptoms limit us to an acute and not very mortal lesion in one cerebral hemisphere, and exclude chronic inflammations and new growths.

Lastly, the morbid anatomy, leaving out of account the comparatively rare fatal cases, simply points to a destruction of cerebral tissue.

I shall now pass to a consideration of the various theories which are held regarding the pathology, while admitting that their authors do not seek to explain it by one origin alone. For brevity I shall entitle them

(1) Congenital Theory.

(2) Vascular Theory.

(3) Inflammatory Theory.

Congenital

Congenital Theory While in many cases the one sided paralysis is noticed from birth or shortly after, and is justly attributed to an intra uterine cause or to the accidents of labour, it is said that many more originate before birth and are either not observed by the mother or do not have a definite onset till the child is some months old. In explanation of the latter statement it is said that the fibres in the corona radiata are not medullated at birth and have no conducting power. The cortex has therefore no work to do, and it is only when it is called upon to start, that its deficiencies are evidenced. This may be true, but one is inclined to ask why some cases are noticed at birth, and especially those cases of diplegia from a meningeal haemorrhage where the cortex is undoubtedly affected. It is difficult to see how a child could be perfectly normal in every respect with a large porencephalic defect in its brain.—Granting that there are many congenital cases, some of those will have to be considered under the other headings as due to an acute process occurring during foetal life or at birth. The others are said to be the

the result of a lack of development in the cerebral tissue, an "agenesis corticalis" as it is termed. Defect in development is usually attributed to non-closure of two sides of the embryo, or adhesion of the amnion (Coats). Porencephaly however, is not a mesial defect like encephalocele, nor could it be explained by the influence of the amnion unless the external structures were involved as well, and there is usually no indication of this. The arrangement of the convolutions in the rest of the hemisphere does not support the theory of an agenesis. One would expect to find a simple undeveloped form in the surrounding gyri, but instead they are so far developed as to be folded up, or they are small and hard and show evidence of a true atrophy. There is, therefore very little in favour of a lack of development.

Vascular

Vascular Theory The proposition that hemiplegia may be due primarily to a lesion in the blood vessels of the brain has very many points in its favour. It brings infantile hemiplegia into line with the better known adult variety which in most cases is vascular, and seeks to explain away differences in character by difference in age. The first glance at a porencephalic brain makes one think at once of a molar necrosis the result of rupture or obstruction of a blood vessel, and, having regard to the situation of the lesion, of the middle cerebral artery in particular. We must however, consider the possibilities in detail.

Haemorrhage is supported by the etiology, which gives traumatism, at birth or in infancy, as a factor, and would make hemiplegia a complication of convulsions and whooping-cough, which are said to raise the blood pressure in the cranium. The sudden onset, with some symptoms of pressure on the brain agrees with this theory, and morbid anatomy gives us more cases of haemorrhage than of any other recent lesion. Even with external injury or an increase of internal

internal pressure, a cerebral artery will not rupture unless there is some predisposing cause such as weakness of the wall. This weakness Sachs has found as miliary aneurysm in some cases, and quotes Recklinghausen and others as having discovered a fatty degeneration in the arteries of young children. Osler reports a case of cerebral aneurysm in a child of six months. This evidence is as yet very limited but it must be taken in absence of better. In any case the rule is for the middle cerebral artery or one of its main branches to rupture, and not as in adults the smaller capsular branches. The amount of blood lost would necessarily be large, and it is difficult in such a case to see how a child could live. It would certainly be fatal in an adult. If the patient does not die, there are symptoms of cortical irritation lasting sometimes for days, and occasionally accompanied by slight fever and delirium, which is not the usual course of a cerebral haemorrhage in an adult. Another objection to this theory is that in a porencephalic

porencephalic cyst there is no discolouration of the wall and crystals of haematoidin have not been found. This would be a crucial point in favour of haemorrhage. I have also heard it said by a Glasgow surgeon that he has trephined the skull in two cases of children thought to be suffering from recent cerebral haemorrhage, and no trace of blood was found.

Haemorrhage from a vein or sinus, the more probable sequel of traumatism, would most certainly spread itself over one hemisphere or even over both, and remains of it would be seen in the thickened membranes in after life.

Embolism Sudden arterial obstruction would account for most of the symptoms, and in a few cases an embolus has been found post mortem. The conical form which is the usual appearance of a porencephaly, resembles in many respects an infarction of other organs such as the lung or kidney, but of course this appearance is really due to the absence of the converging motor tracts. The area involved however, corresponds in a general way to the distribution of the middle cerebral artery viz. the 2nd and 3rd frontal, ascending

ascending parietal and frontal, and the three parietal and temporal convolutions and also the corpus striatum. The fact of this being the region affected, and the slightly greater preponderance of involvement of the left side over the right, coincides with the pathology of cerebral embolism in the adult. Fagge reports an adult case of Charcot's in which the Sylvian artery was plugged in its main trunk and cerebral softening resulted.

On examining cases carefully however, it is seen that the region involved does not exactly correspond with the distribution of the artery. There is either too much or too little affected. In Case X the obstruction would require to have been in the main artery before its capsular branches were given off, as the corpus striatum is involved. But the 2nd frontal, the parietal and temporal, and much of the Rolandic convolutions, still remain. This of course, might be explained by an anastomosis with the other arteries in the pia, which might vary in different cases.

The greatest difficulty in accepting embolism as a cause, is to find the source of the embolus. This is usually

usually easy in adults, where heart disease is almost invariably present. In infants, however, endocarditis is rare and most hemiplegics give no evidence of it. A case of porencephaly reported by Barratt,[10] was attributed to embolism in the first year, because the patient (a man of 30) had an old valvular lesion. The Sylvian artery however, was found patent and perfect, so we must hesitate to accept his theory. In another case by Shirres,[12] the source of the embolus was said to be placental thrombosis, occurring ata threatened abortion. The fact of hemiplegia being sometimes secondary to bronchitis and pneumonia, makes it probable that thrombosis of the pulmonary veins is a source, and the changes which the circulatory system undergoes in early life might also account for thrombosis and embolism but there is no proof of this whatever, and it is unwise to speculate too much on that point. In the rare instances in which embolism does occur, the clot, being apparently too large for the lenticulo-striate branch, remains in the main trunk of the artery. The sudden damming of the blood stream

stream would certainly cause a severe cerebral disturbance, but with the help of the collateral circulation it is quite possible that the attack might not be fatal.

Thrombosis in a cerebral artery must be a rare condition in the absence of a predisposing cause such as disease of the arterial coats, and it is probable that in the reported cases there was originally an embolus. Coagulation in a vein is not so unlikely. As an ante mortem occurrence in the longitudinal sinus of marasmic infants, it is quite well recognised. The case which Gowers reports of thrombosis in a surface vein causing atrophy of a convolution, might explain a large number of those cases of localized atrophy. In Case XI for example, there may have been a double thrombosis in the veins from the Rolandic fissure. Gower's theory however, has not been generally accepted.

Inflammatory Theory

Inflammatory Theory. In 1884 Strumpell first promulgated his theory that there was an inflammation of the cerebral grey matter similar to that which is seen in the anterior cornua of the spinal cord, and this he called polioencephalitis, in contrast to poliomyelitis. Apparently he had little foundation for his theory beyond the homology of the two structures, but the view was accepted by many in opposition to Kundrat's vascular theory of hemiplegia. In support of Strümpell's idea, Mobius[3] published the cases of two children of the same family who were stricken with pyrexia, and somnolence. One of them developed cerebral paralysis, and the other spinal. Of late years the theory has been vigorously attacked by many authorities. Sachs is especially strong in his opposition, saying that there is absolutely no anatomical proof, and that the facts are opposed to it. He admits, however, that there may be a few cases, and in another work describes an encephalitis of the nuclei of the cranial nerves. In his article on Cerebral Palsies, Gray[3] makes the statement that Strümpell's was the "most flippant suggestion ever made in medicine". Yet

Yet he remarks (without any reference to his authority) that the vascular theory has been disproved, and that porencephaly is due to a "peculiar cellular degeneration, independent of the vessels"!

The objections of recent authors to poliencephalitis are briefly these (1) that post mortem evidence is against it (2) that it ought to be more fatal (3) that poliomyelitis is not secondary. One might take the first two points together, and say that if it were more fatal there would be more post mortem evidence. But why should it be more fatal? In the cord the process is limited to isolated sections, and usually to the anterior cornua of these sections, and if it is thus limited in the brain, there is no reason why it should cause death, any more than, or even so much as a vascular lesion. The third objection is only half true. Infantile paralysis often accompanies other affections, and, according to Gowers, hemiplegia is not distinctly a secondary disease.

Let us consider briefly the points in acute anterior poliomyelitis. It is a disease of early childhood. It

It starts suddenly, often with slight general disturbance, and occasionally with convulsions (see Case IX). It is never fatal, but when from any cause the child dies, changes are seen in the grey matter of the anterior portion of the cord. These changes are minute haemorrhages and thrombosis with perivascular exudations, leading secondarily to death of some of the ganglionic cells and increase of the interstitial tissue. The cornu is supplied by the anterior median artery, so that the changes are probably primary in the small terminal branches of that artery, and limited by its distribution. There is good ground for saying that the process is microbic or at least toxic in origin, but it is still held that it may be a vaso motor phenomenon.

The conditions in cerebral paralysis are markedly similar. It begins in foetal life or infancy at a time when, as I have pointed out, rapid changes are progressing in the region of the operculum, and both cells and vessels are in a state of imperfect development. The onset is marked by constitutional disturbance which cannot altogether be

be accounted for by the local lesion. The acute period lasts a variable but usually short time, and there is no progression in the resulting paralysis. In anatomy also there is some analogy. The cortical arteries are end-arteries like those of the cord, and the cellular elements are similar both in function and structure. I have pointed out that in localised cerebral atrophy, the pyramidal cells are much destroyed, and minute haemorrhagic cysts have been found. This has a striking resemblance to cornual atrophy. It might be objected that there is a much greater loss of substance in the brain - as in porencephaly. This loss, however, is white matter chiefly,— degenerated motor fibres,- which in the other case pass almost immediately into the nerves, so that there is little loss of bulk to the cord itself. Further, Gowers describes and pictures actual cysts in the cord in poliomyelitis.

Finally, there is post mortem evidence in favour of this view. In a recent number of the Lancet, Batten, without any reference to the controversy, or even to Strumpell's theory, describes cases of "polioencephalitis" of different

different regions. Among them is the case mentioned above (see p 48) which at the autopsy showed a lesion in all respects resembling that of acute anterior poliomyelitis. The case mentioned by Holt, might have shown similar changes, had the motor region been examined microscopically.

As I have said before, the exact pathology of poliomyelitis is still uncertain, and that of polioencephalitis will be much more so. The fact of its limitation is against its being a bacterial inflammation, and it may happen that it will be found to be a purely vascular phenomenon, with this difference, that the changes occur in the terminal cortical arteries and not in the large vessels. The experiments of Prevost and Cotard have shown that ligature of a cerebral artery does not produce serious disturbance in the

the nutrition, whereas injection of a fine powder plugs the arterioles, and these having no anastomoses, softening and necrosis of the grey matter is produced. If it were found to be due to organisms, the lesion might be the result of microbic emboli, which might be specific, or, in themselves non-pathogenic. On the other hand, the changes might be caused by a selective poison circulating in the blood in the course of one of the so called primary diseases, though in such a case one would expect it to have a longer and progressive course. These points have to be considered in the pathology of acute poliomyelitis, into which I do not intend to enter further. I have pointed out the marked resemblance and the probable identity in the pathology of the cerebral and spinal paralysis, and now leave the solution of the problem till further investigations into the character of the more common spinal variety have been made.

Conclusions

Conclusions as Regards Pathology. In the foregoing, I have attempted to show cause for the following beliefs:

(1) That a simple idiopathic lack of development as a cause of porencephaly cannot be accepted.

(2) That haemorrhage from the large cerebral vessels is present in most of the fatal cases, but the conditions are against its being the most frequent lesion.

(3) That embolism is possible, only when there is a source for the embolus, and that is only in a very few cases.

(4) That thrombosis of an artery or vein is very unlikely, and the cases reported are not sufficient evidence.

(5) That the lesion will be found in most cases to be in the small cortical arteries, and that its pathology is analogous to that of acute anterior poliomyelitis.

DIAGNOSIS

DIAGNOSIS. During the period of onset the diagnosis must present serious difficulties. Occurring sometimes in the course of an acute disease, pyogenic meningitis will be at once suspected, and it will only be with the recovery of the patient that the real affection is recognised. Indeed, one might say that those cases of reported recovery from meningitis might be explained by a pathology similar to this. Tubercular meningitis might be eliminated by the absence of a prodromal period, and the sudden onset, but, of course, these are not always characteristic. After recovery of the patient from the acute attack, the diagnosis should always be easy. The first point is to distinguish it from anterior poliomyelitis. The extent of the paresis,- affecting the whole of one side, - the exaggerated reflexes, the absence of the reaction of degeneration, and the rigidity of the muscles should make this clear.

Later on, I should also include facial asymmetry and diminished size of the opposite side of the cranium as points in favour of a cerebral affection. When epilepsy and

and mental defects set in, there is usually no doubt left. The only other difficulty would be to exclude hemiplegia due to chronic lesions, such as tumour. In this the history of the onset and partial recovery, the absence of Jacksonian epilepsy and of symptoms of intra cranial pressure, such as headache, vomiting, and optic atrophy, would be crucial points.

The diagnosis of the cause of the hemiplegia cannot be definite. Occurring in the course of endocarditis it will be attributed to embolism, and after a protracted labour or injury, it may be put down to meningeal haemorrhage. When the child dies, this probability will be more considerable.

PROGNOSIS.

PROGNOSIS. It is usually said that the prognosis as regards life is good, and that recovery is the rule, but as we do not know how many cases die before the diagnosis is made, this statement must be taken with caution. It is obvious that many children who die from "meningitis" or "convulsions" may have had a localised lesion in the cortex as a primary cause. However, it is certain that the majority do recover, and once the acute period is over, there is little anxiety about the child's life. The paralysis also will pass away to some extent, but the patient will always be under the cloud of prospective secondary degenerations in the way of epilepsy and idiocy.

TREATMENT.

TREATMENT. As in the case of most nervous diseases, medical treatment is purely expectant. During the acute period, a little might be done by the application of iced cloths to the head, and the administration of chloral and bromide, on the presumption that the brain is congested and irritable, and there is a possibility of haemorrhage. When the child recovers, the restoration of the muscular power should be assisted by massage and electrical treatment. If there is a tendency to epilepsy, this should be combated with the usual treatment, in the hope that no further general effects will result. The patient should be educated with care, every consideration being paid to the weak mental condition.

Surgery does not hold out much hope of good results at present. In the acute stage it is perhaps advisable that the skull should be trephined, and the motor region examined for blood clot, but as I have said, this was done in at least three cases, and no clot was found. In the later stages, nothing radical can be attempted. If the surgeon could transplant cerebral tissue, and form a new motor cortex, one would expect some good result, but until that can be done, there is little to be gained by operation.

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