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Acute Leptomeningitis in Children:

its

Prognosis and Sequelae.

by

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I N D E X .

	<u>Page.</u>
General Considerations	1 - 6
Historical	6
Anatomical & Physiological Points...	9
Tuberculosis Meningitis...	14
Serous Meningitis & Meningism	16
Aseptic Purulent Leptomeningitis	27
Meningococcal Meningitis	28
Sporadic form	31
Epidemic form	42
Completeness of Recovery	49
Pathology	52
Internal Hydrocephalus	54
Loss of Vision	71
Deafness	76
Deafmutism	80
Mental Deficiency	87
Paralytic Sequelae...	93
Persistent Convulsions	102
Persistent Ataxia	105
Pneumococcal Meningitis	107
With Typhoid Fever	111
With Influenza...	111
With Congenital Syphilis	112
Bibliography	113
(arranged with Authors in alphabetical order.)	

Ages thus:-

- 3. three years.
- . 3 three months.
- 3.3 three years,
 three months.

ACUTE LEPTOMENINGITIS IN CHILDREN;
its PROGNOSIS and SEQUELAE.

In selecting this subject for a Thesis, I was influenced by the fact that, although much work has been done recently on the Diagnosis and Treatment of the various forms of Leptomeningitis, considerably less attention has been paid to the after effects thereof; for this disease, if not immediately fatal, exhibits a marked tendency to leave a residue of ill effects in the form of various deformities, which may or may not be ultimately recovered from.

Complications during the acute stage of the illness only, i.e. Paralysis or spasm of muscles, Metastatic abscesses, Epistaxis, Irido-cyclitis, etc. are not dealt with, but only those which remain after convalescence from the original Meningeal Inflammation.

Naturally, such investigation must be to a large extent clinical, in that very few of the cases followed up and examined at intervals are available Post Mortem, in order to trace the seat and nature of the lesion causing the Sequela. On the other hand, examinations on fatal cases frequently shew lesions which might quite well be responsible for any given Sequela, and together shewing the latter to be

dependant, roughly, on three facts:-

- (1) Fibrous thickening and consequent scar contraction of a previously acutely inflamed Membrane, also, probably invariably, involving the subjacent brain tissue itself.
- (2) Compression of brain tissue by an acquired Internal Hydrocephalus.
- (3) That all cranial nerves and vessels are surrounded by lymph channels directly continuous with the Subarachnoid space, thus facilitating the spread of any infective process from this area along them.

I may mention here that I have been unable to find definite records of any sequela brought on experimentally in animals, the subject being either killed outright by the intra-spinal injection of organisms, or else being completely unaffected. Courtellement mentions a case of a dog which developed an acute, rapidly fatal, ascending paralysis four months after injection of this sort with Meningococci, although it shewed no sign of Meningitis at time of inoculation.

Most of statistics and cases noted were obtained from the Casebooks of the Royal Hospital for Sick Children, Edinburgh, for about the last 10 years.

The parents of 42 children discharged either "Cured" or "Relieved" were written to. Of these 29 answered, and 23 children were brought up for

examination, either once or repeatedly. In addition, particulars were obtained of the progress of 4 others, and also 12 cases which came up for treatment or advice during my term of House Physician are dealt with, including all those of Meningism described.

Cases removed to the City Fever Hospital were followed up in the Casebooks there.

I went over the records in the Royal Deaf and Dumb Institution, and also in Donaldson's Hospital for Deaf and Dumb, Edinburgh, and saw all the cases at present under tuition in both these Schools.

It will be seen that all the cases of the Tuberculous, Pneumococcal, and Septic varieties were fatal: all the cases described as suffering from Sequelae were discharged after Cerebrospinal Meningitis of Meningococcic origin, either Epidemic or Sporadic, and I shall give a brief resumé of the points on which the presumption is based that these are only slightly modified forms of the same disease.

Naturally in the earlier cases, before the present refinements of diagnosis arose, there may have existed a certain doubt as to the true nature of the case. I went over, and carefully annotated, the notes of each, and excluded all those in which there was not sufficient evidence, either Clinical or Bacteriological to bear out the Diagnosis.

The following Classification of Acute Leptomenin-
gitis was followed, (Osler) being roughly cast into
three groups:-

Tuberculous,, Meningococcic, Forms due to other
organisms.

Primary.	(1. Cerebro-Spinal Fever.	(a) Sporadic (b) Epidemic	{ Diplococcus { Intracellularis
	2. Pneumococcic	{ Meninges in- volved alone, or in a general { Pneumococcic { Infection.	{ Pneumococcus of { Fränkel
Acute Lepto-Meningitis. Secondary.	1. Tuberculous.		B. Tuberculosis.
	2. Pneumococcic	(a) Secondary to { Pneumonia, en- docarditis, etc. (b) Secondary to { Disease or in- jury of Cranium { or its Fossae.	{ Pneumococcus
	3. Pyogenic	(a) Following { local disease of cranium, or a { local infection (b) Terminal in- fection in va- rious chronic { maladies.	{ Various forms of { Staphylococci and { Streptococci.
	4. Miscellaneous Acute Infec- tions.	{ In Typhoid Fever, { Influenza, Diph- theria, Gonorr- hoea, Anthrax, { Actinomycosis { and other acute { Diseases.	{ Causative { Bacteria.

For certain purposes, the following is also useful.

(1) Forms where bacteria found in Cerebro-spinal Fluid,

- (a) Serous.
- (b) Purulent.

(2) Forms where Cerebro-spinal Fluid sterile,

- (a) Serous
- (b) Purulent (Widal)

The conditions of Serous Meningitis and Meningism have been described, and their Prognosis gone into.

I also append a Scheme of the Sequelae that have been described here, for purposes of reference.

- (1) Secondary Hydrocephalus.
- (2) Motor Derangements.
 - (a) Paralyzes (Local or Widespread) } Spastic
 - (b) Persistent Convulsions. } Flaccid
 - (c) Ankylosed joints.

- (3) Sensory Derangements.
 - (a) Headache.
 - (b) Shooting pains in Limbs.
 - (c) Ataxia.

- (4) Mental Derangements.
 - (a) Backwardness in Walking or Speaking; Persistent Nocturnal Enuresis.
 - (b) Mentally Defective.
 - (c) Idiocy.

- (5) Sudden Death.

Special Senses.

- (6) Affections of Eye.
 - (a) Squint.
 - (b) Corneal Opacities from Keratitis.
 - (c) Dimness of Sight } With no discoverable change in eye
 - (d) Complete amaurosis } itself.
 - (e) Permanent Blindness } (Following Thrombosis of Retinal veins, or Metastatic Iridocyclitis.
 - (f) Secondary Optic Atrophy.
 - (g) Homonymous Hemianopsia.

- (7) Affections of Hearing.
 - (a) Complete Bilateral Deafness, with
 - (b) Staggering gait.

(8) Deaf Mutism.

(9) Loss of Taste.

HISTORICAL.

Although Epidemic Cerebrospinal Meningitis was first described as a separate disease in 1805, it was not till about 1837 that Lespès reported some recoveries after an epidemic who "were deprived of the use of one or other sense." Shortly after this, the frequency of Post-meningitic Deafness was again pointed out, and Hilton was the first to draw attention to the obstructive nature of Acquired Hydrocephalus in 1868.

Since then, cases of Post-meningitic sequelae have been published at intervals, mostly on the Continent.

In 1894 Dupré first described the condition of Meningism, and this, together with the rapidly growing knowledge of Hysteria, and other nervous diseases, cast doubts on the possible curability of Meningitis proper. As Dupré said:-

"It was very difficult to honestly say that true "Meningitis was curable at all;" he could only barely admit the possibility of a chronic Meningitis, and still less an acute Ventricular Meningitis, resulting

in a chronic Hydrocrphalus." In later years, however, mainly owing to the researches of Quincke, Weichselbaum, Still and others, the various forms of Acute Leptomeningitis have been more accurately differentiated. Cures of true meningitis, with or without sequelae, are now certain - in Suppurative cases very seldom, in Tuberculous almost as rarely, but with modern serum therapy the Prognosis as regards the Meningococcic forms is very encouraging, both as regards the frequency and completeness of the cure.

Causation of Sequelae, considered Clinically.

Exactly why certain recoveries should be marred by some lasting deformity cannot be explained satisfactorily, as the most severe cases, even if prolonged may be completely cured. On the other hand, very slight attacks may leave permanent after-effects, e.g. Hydrocephalus or Deafness in young children. Moreover, it will be seen that the majority of my cases followed severe attacks, and were noted as exceptionally severe in the only two cases of Spastic Diplegia found. These latter cases also illustrate the fact that the sequelae may first appear either at the very onset of the acute illness, or some considerable time after, during convalescence.

Relapses are said to favour the onset of sequelae, but I have not been able to bear this out. None of the cases shewed any special tendency thus, and in

many the sequela first was noticed in the early days of the illness.

Courtellemont mentions the fact that Abortive forms of Meningitis are often followed by a Flaccid Paralysis. In these cases, however, the diagnosis from Poliomyelitis Anterior Acuta can only with difficulty be made, and the constitutional symptoms at the onset of the latter disease are notably slight.

Hildesheim urges necessity of ascribing all the after-effects to Acquired Hydrocephalus, either apparent or concealed - the latter if cranial ossification is complete, and only asserting itself by the commencement of symptoms due to cranial compression. This view is strengthened by the growing conviction that many cases of "Congenital" Hydrocephalus are really due to a Basal Meningitis either in utero, or very early in life and probably specific in origin, but is negatived by the pathological findings in many cases.

I have been unable to trace any symptom, train of symptoms, or defect in previous health which might render the patient more susceptible to the onset of sequelae, and can only state that the recovery is less likely to be complete:-

- (1) In very young children, Hydrocephalus being principal danger.
- (2) In very severe initial attacks, Deafness being the commonest and most hopeless relic.
- (3) In cases where the acute symptoms are very prolonged.

It is remarkable how great the tendency is for sequelae, apart from the two just mentioned, to clear up completely in time; this also is against the Hydrocephalus origin of all.

Pathological Findings.

These will be described later under separate headings.

Anatomical and Physiological Points.

The subarachnoid space of the brain and spinal cord with its various "wells" or "cisterns", has no anatomical connection with the Sub-dural space, and contains in its meshes most of the cerebro-spinal Fluid, normally only about 2 ounces in all. Its only communication with the Ventricular System of the brain is through 3 orifices, all at outlet of 4th Ventricle:-

- { The Foramen of Majendie.
- { The 2 Lateral Foramina of Luschka.

It sends distinct prolongations round the cranial nerves, notably the Optic and Auditory pairs, and of course extends between the layers of the Velum Interpositum.

Key and Retzius injected coloured fluids into the subarachnoid space of the Spinal Cord, and noted its spread into the Perilymphatic space of the Internal Ear, into the Aqueductus Cochleae which communicates with it through the Scala Tympani of the Cochlea, also

into the Perineural sheaths of the 7th and 8th nerves in the Internal Auditory Meatus as far as the Lamina Cribrosa. The fluid could never be traced into the Middle Ear, the Endolymphatic space, or the Aqueductus Vestibuli except in infants, where the non-closure of the Petro-squamosal Suture in the roof of the middle ear allowed direct communication between this cavity and the general Subarachnoid space.

In addition, these authors, with Jochmann more recently, were able to trace this injection right forward over the base of the brain, and if the pressure was maintained, shewed that it "filtered" through the Pacchionian Bodies into the Superior Longitudinal Sinus, thus reaching the blood stream direct.

The lymphatics of the Central Nervous System are peculiar in that they commence as ramifications round each nerve cell,--the Pericellular Sacs of Bevan Lewis, and finally ending in the Subarachnoid space, have their only communication with the general venous and lymphatic systems through the Pacchionian bodies. The lymphatics of the peripheral nerves are in the form of tubular spaces between the lamellae of the perineural sheaths, these channels opening into either the Subdural or Subarachnoid spaces. Continuations of this nature also surround the arteries down to their minutest ramifications.

The Cerebro-Spinal Fluid.

This is now practically accepted as being a secretion of the Choroid Plexuses by a process analogous to the selective action of the kidney, on the following grounds:-

- (1) These structures, present in each ventricle consist of a mass of vessels carrying arterial blood only separated from the ventricular cavity by a layer of highly differentiated cubical epithelial cells. Their bloodsupply is abundant, by the anterior and posterior choroidal arteries; the corresponding veins, - those of Galen, - are narrow, tortuous, and withdrawn deeply between the layers of the velum interpositum.

Barlow and Lees quote a case of non-progressive Hydrocephalus in an infant, where P.M. a cystic degeneration of these plexuses in the lateral ventricles was found.

- (2) The normal fluid differs from a lymphatic transudate (Halliburton) in that it is clear and limpid, is non-coagulable because it contains no albumin or fibrin ferment, but only a trace of proteid mostly in the form of globulins and albumoses, and contains a special Fehling-reducing body belonging to the Pyrocatechin group. Moreover, if the normal balance is disturbed experimentally, as by continuous

withdrawal of the fluid by Lumbar Puncture, it gradually becomes very closely to resemble in every way an ordinary transudation from the blood.

Leonard Hill demonstrated that the pressure of the Cerebro-spinal fluid must correspond to that in cerebral veins, and that this balance is mechanically maintained. Any slight increase in tension in the former is relieved by its removal through the Pacchionan bodies into blood stream and thus out of cranium with a sort of safety valve action. Naturally the intra-cranial arterial tension must always be greater than the venous, otherwise the cerebral circulation would cease.

Before going on to consider the various forms of Leptomeningitis, I have to express my great indebtedness to the Medical Staff of the Royal Hospital for Sick Children, Edinburgh, for allowing me to use their cases, and specially to Dr John Thomson, whose House Physician I then was.

Dr Claude Ker kindly permitted me to examine the records of cases admitted to the City Fever Hospital, and my thanks are also due to the Medical Officers of the various Deaf & Dumb Institutions in Edinburgh for their permission to visit the patients and look up Registers of cases under their care.

I do not intend to go into the differential diagnosis between the various forms of Acute Leptomeningitis, but merely append a Table shewing the distinctive characteristics of the Cerebro-spinal fluid in each, this being withdrawn by lumbar puncture and examined at once.

<u>Tuberculous.</u>	<u>Epidemic Cerebro-Spinal.</u>	<u>Sporadic Cerebro-Spinal.</u>	<u>Suppurative.</u>
<p>Clear, opalescent, gradually becoming turbid, and may be purulent in later stages.</p>	<p>Turbid, yellowish and purulent may be very thick.</p>	<p>As in the Epidemic form, but as a rule less marked. In infants the exudate tends to be drier and more "sticky."</p>	<p>Thick and purulent usually greenish yellow.</p>
<p>Pressure much increased.</p>	<p>Increased, or too thick to run.</p>	<p>...</p>	<p>ditto often very thick.</p>
<p>Fine fibrin network forms on standing.</p>	<p>Is coarser, and more abundant.</p>	<p>...</p>	<p>Very marked.</p>
<p>After withdrawing Fibrin-Albumen much increased, 5 - 10%</p>	<p>Moderately increased up to 5%</p>	<p>...</p>	<p>Moderately increased up to 5%.</p>
<p>Pyrocatechin reaction diminished or absent.</p>	<p>Absent (if reappears, good sign).</p>	<p>...</p>	<p>Absent.</p>
<p>Lymphocytosis.</p>	<p>Polymorph. Leucocytosis.</p>	<p>...</p>	<p>Polymorph, leucocytosis, much degeneration of cells.</p>
<p>Tubercle Bacillus can be isolated from fibrinous clot in 85-95% of cases.</p>	<p>Meningococcus Intra-cellularis of Weichselbaum.</p>	<p>Still's Diplococcus isolated. May precisely resemble Weichselbaum, but usually differs slightly i.e. shows greater vitality, will grow on broth freely. Agglutination & Opsonic Tests do not quite correspond.</p>	<p>Practically any pus-producing organism. In order of frequency: - Pneumococcus, Streptococcus, Staphylococcus (usually P. aureus)</p>
			<p>B. Pfeiffer. B. Typhosus. B. Coli Communis, etc.</p>

Tuberculous Meningitis.

The cases numbered 304.

Of these 247 died in Hospital, and 57 were sent home In Statu Quo, none of whom presented any reasonable chance of recovery.

The total deaths from this cause in 1907 as detailed in Registrar-General's Return, (England & Wales alone) were 5885, or 1.122% of the deaths from all causes. Of these:-

113 (1.9%)	were between	0 - 3 months.
337 (5.9%)	" "	3 - 6 "
1956 (33.2%)	" "	6 - 24 "
1481 (25.2%)	" "	2 - 5 years.
984 (16.7%)	" "	5 - 10 "
411 (6.6%)	" "	10 - 15 "
only 584 (9.9%)	" "	15 - 45 "

thus shewing the prevalence of the disease in young children, but the comparative immunity of babies under six months.

The disease is, as shown, almost universally fatal. Barlow says:-

"In rare cases of old standing Tuberculosis,
 "there may be found Post-Mortem a circumscribed area
 "on convexity of brain presenting fibrous circatrical
 "material associated with some small caseous deposit
 "and scanty granulations indicative of long-healed
 "Tubercular changes. These are most often found in
 "adults, andso in localised forms of Tuberculous

"Meningitis there may be a slight chance of recovery, but if generalised, none whatever."

Thiemich says:-

"Prognosis is almost always hopeless; none of reported cures have been kept under observation for years, and in many of them the diagnosis was not rendered complete by finding bacilli in the cerebro-spinal fluid."

Martin, however, has recently collected all cases reported as cured in the literature, carefully excluding all those where the diagnosis was not positive absolutely, the standard required being the discovery of Bacillus Tuberculosis in the cerebro-spinal fluid by microscope, culture, or experimental inoculation.

He found 7 cases of certain remissions, all in children, and ultimately fatal after an interval of from 4 months to $2\frac{1}{2}$ years, - one after $5\frac{1}{2}$ years.

He also describes 20 cases of "certain" complete recovery, 8 being noted as under the age of 12 years. Of them all, however, only 3 were watched over a period of a year, and the shortest time was 3 weeks, so naturally the difficulty is to say for certain whether the condition was not merely that of remission of the disease for a time.

In one case, however, a boy died of Phthisis aet. 17. He had had undoubted Tuberculous meningitis at 9 years, and at the Post-Mortem 8 years later the site

of the old Meningitis was found, the membranes over each fissure of Sylvius specially being thickened and glued together. There were no recent tubercles whatever on the brain.

Of effective Treatment, little is to be said; in none of those reported cured, was Tuberculin used, nor did any special line of treatment preponderate.

In 1905 Drs. Duret and Alexandri gave it as their opinion, after extensive trials in France from 1890-1900 that Surgical interference was of no value in generalised cases, and was now given up. In circumscribed cases, the possibilities were better, but diagnosis usually impossible, therefore but few cases had been attempted.

Stiles in 1908 reported 2 certain cases greatly improved by drainage of the subarachnoid and subdural spaces, in one the symptoms reappearing if the wound was closed. Otherwise the attempts to use operative treatment seem to have been very few in this country.

Serous Meningitis and Meningism.

These conditions are similar, the essential difference being one of degree, both pathologically and as regards clinical severity. That is to say, Meningism, as described by Dupré, is a train of meningeal symptoms with no pathological findings here but slight hyperaemia, whereas in Serous Meningitis the cerebro-spinal fluid is in addition increased in

amount and altered in character. Clinically therefore, the latter condition fills in the gap between tuberculous meningitis - essentially a serous inflammation at first - on the one hand, and meningism on the other, the prognosis improving vastly with each step. Also meningism, if not checked, will tend to assume the serous form.

It is necessary to recall the Classification:-

(1) Acute Leptomeningitis with organisms in Cerebro-Spinal Fluid.

- (a) Serous.
- (b) Purulent.

(2) Cerebrospinal fluid sterile.

- (a) Serous (albumen may be slightly + with little fibrinous clot and few lymphocytes)
- (b) Purulent (Widal)

(1) is not dealt with here, as the serous form is only an early form of the purulent unless it is at once fatal, due to a fulminant infection probably depending on an individual idiosyncrasy.

(2) a. and including Meningism.

Causation.

Never primary, but always secondary to some infection elsewhere. The actual causative factors are:-

1. The Toxins of any organism capable of producing inflammatory changes, specially:-

- (Pneumococcus.
- { B. Typhosus, Paratyphosus, or Coli, from Gastro-intestinal derangement. Osler only saw 5 cases of the serous form in 1500 of Typhoid, although slight Meningitic symptoms very common at outset.

(B. Pfeiffer (influenza)
 { B. Tuberculosis thought by some to be very
 { important. Armand Delille injected ex-
 { tracts of toxins obtained from this organism
 { into subarachnoid space of brain, and ob-
 { tained pathologically an acute inflammation
 { of Pia mater, with great proliferation of
 { Polymorph and Lymphocyte cells.

2. Reflex, as from Teething in infants. Here process does not usually extend beyond meningism and is easily relieved.
3. From Middle ear disease. This may be very difficult to distinguish clinically from acute primary meningitic inflammation, even although the latter is quite unaffected. Nevertheless, an acute serous meningitis often co-exists without any actual continuity of infection.
4. Vasomotor changes, as in Quinck's idiopathic internal Hydrocephalus (see later)

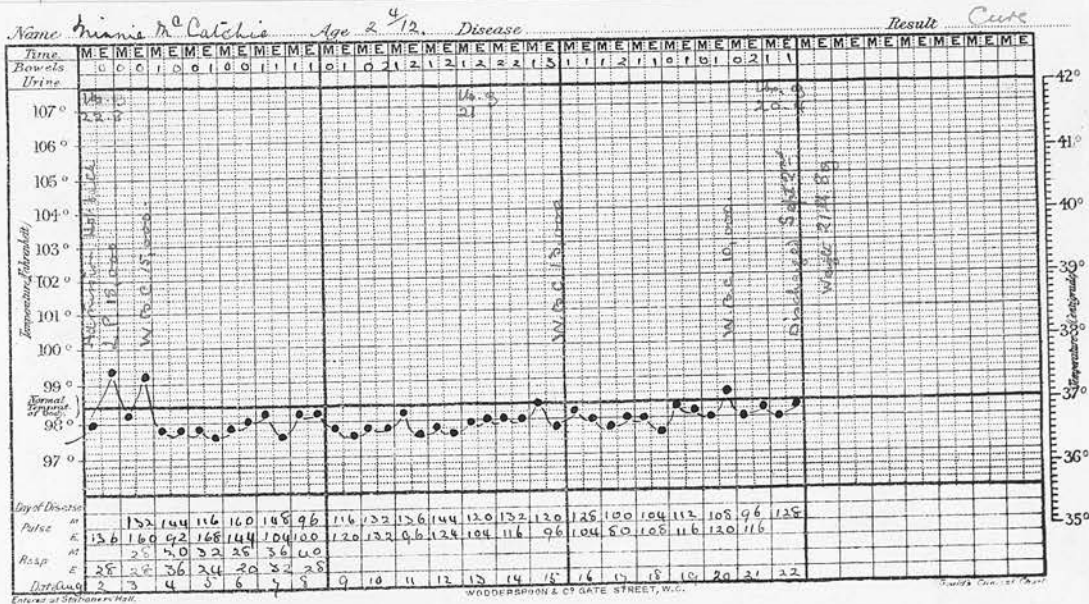
PROGNOSIS.

Being a secondary disease, very hopeful if the initial lesion can be cured without delay. Meningism usually passes off soon, and with no difficulty, eg. a brisk purge to a teething baby.

The serous form is also usually very amenable, and it is essential to begin treatment early, - Lumbar Puncture, depletive measures as leeches over Mastoid processes, and purgation -, together with immediate treatment of the causative lesion. If delayed, the excess of cerebrospinal fluid may get beyond the balancing power of the cerebral circulation, or the

ILLUSTRATIVE CASES.

CASE I.



Nothing in Family History or Previous Health, always "smart" healthy child.

Three weeks ago became tired, dull, and drowsy, with occasional interludes of normal brightness. Vomited at first, continued headache, neck held stiff, and has gone completely off feet.

For last week has had twitchings, specially of face, and arms shewed coarse jerky volitional tremor, and has at intervals had slight convergent squint. Very constipated and unconscious for last few days.

On admission was in dazed condition, very irritable if touched, and typical cerebral facies present.

Pulse 136, irregular and radial felt contracted.

Leucocytes 18000.

Pupils reacted sluggishly; fundi normal.

All tendon jerks ++, no Babinski or rigidity present.

Lumbar Puncture. 14 cc. clear fluid removed under considerable pressure; albumen distinctly +, fine fibrin clot formed, with few lymphocytes, but no tubercle bacilli could be isolated.

Heart, lungs, whole gastro-intestinal tract, urine and ears healthy as far as could be made out.

After 10 days began to improve, L.P. having been done twice, and 30 cc. more fluid, as before, removed.

Fortnight later was discharged perfectly well, except that was not walking, and that arms still shewed volitional jerkings to less degree.

Two months later was just beginning to walk, with very unsteady ataxic gait, but unsteadiness of upper extremities had quite passed off, and altogether child was very fit.

Tuberculous Meningitis was diagnosed at first, but later progress of case pointed to a simple serous form, although no primary lesion could be discovered. Encephalitis was rendered unlikely by the slow onset.

CASE II. With Influenza.

M.W. aet. 8 years.

Nothing in Previous Health or Family History, except recent epidemic of influenza.

Ten days ago suddenly began with severe frontal headache, vomiting of cerebral type, very dull, constipated, and irregularly fevered.

On admission, very drowsy, but irritable if touched. Marked cerebral facies, with general rigidity.

Pulse 120, irregular. Temperature 102.5° to 104°. Resp. 28. W.B.C. 16000.

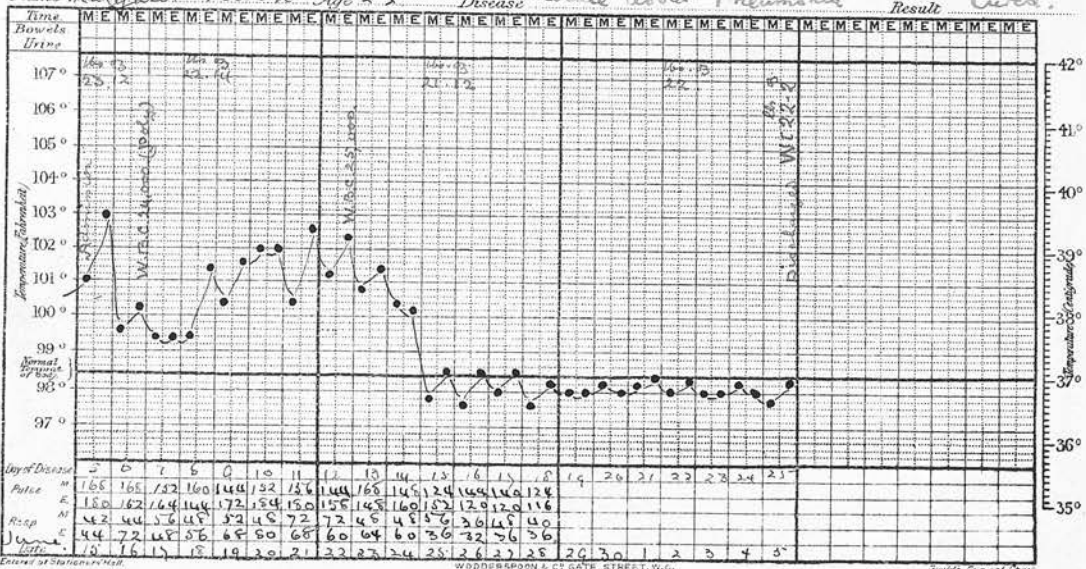
Lumbar Puncture. Clear fluid under pressure; few lymphocytes.

Abdomen, Lungs, ears, etc. appeared healthy.

After 1 week temperature fell by crisis, pulse slowed to 65, and child had a speedy convalescence.

CASE III. With Pneumonia.

Name *Margaret Wilson* Age *35* Disease *Acute Lobar Pneumonia* Result *Cured*



Admitted complaining of pains in head and abdomen, drowsiness, cough and fever.

Been dull and irritable for week or so, when suddenly 5 days ago became flushed, fevered, perspired freely, very drowsy, and generally rigid.

On admission was pale, with cerebral facies,

drowsy, but extremely irritable if touched, and Stocker's sign pronounced. Cry loud and sharp, no cough.

Pulse was irregular. Leucocytosis 24000 (Polymorph).

Reacted slightly to Von Pärquet (human).

Knee jerks +; distinct tache cerebrale.

Lungs, heart, abdomen, ears, etc. no abnormality found on repeated examination.

Urine shewed medium diminution of chlorides.

On 14th day of illness distinct physical signs of Right lower lobe Pneumonia developed, and from this date uninterrupted convalescence began.

I also had a case of a child aet.3, who exhibited all the symptoms and signs of a mild attack of cerebrospinal meningitis, except that by lumbar puncture the fluid was clear, no increase of pressure, but slight excess of lymphocytes present.

After fortnight's illness, began to recover and was discharged well, with diagnosis of Serous Meningitis. In some weeks, however, symptoms came on again as before and child died after illness of two months. P.M. typical findings of chronic cerebro-spinal meningitis with Diplococcus of Weichselbaum was found, and organism isolated.

SEROUS MENINGITIS OF QUINCKE.

(acute idiopathic internal Hydrocephalus.)

This disease is not common, and is probably an ependymitis of Ventricles of brain, caused either

- (1) by a process analogous to angioneurotic oedema, (Quincke)
- (2) by an exacerbation from some cause,—probably toxic or psychical, of a pre-existing serous meningitis, of type described elsewhere.

If acute, it is very apt to be confounded clinically with suppurative meningitis, while if more chronic with illdefined cerebral tumours; but the cerebrospinal fluid, although under increased tension, does not differ materially from the normal in either case.

Quincke reported several certain cures, and stated that long remissions of symptoms were frequently seen.

The only case seen which might be of this nature is as follows:-



CASE IV

K.C. aet 6⁷/12 years.

Very marked tubercle on mother's side, but always strong healthy child. When 6 years old suddenly began to complain of severe frontal headaches, with vomiting. Three months later noticed walking becoming bad, and gradually became paralysed, and suddenly became blind, with slight internal strabismus of left eye. Intelligence remained good.

When admitted, had well marked spastic paraplegia, with slight weakness of upper extremities and lumbar centres; double optic neuritis going on to Atrophy; slight lateral nystagmus with weakness of muscles; cerebrospinal fluid normal in every way, but under increased pressure;

Pulse 65·75, very irregular.

She gradually became much duller mentally, and was discharged in this condition after 2 months.

Unfortunately, she could not be found again later, so no further history is available.

The diagnosis at first was Tuberculous Cerebellar Tumour. Owing to the mode of onset, the gradual development of symptoms all well attributable to increasing pressure, together with the increased tension of the cerebrospinal fluid, it was later thought to be possibly a case of the nature under discussion.

ASEPTIC PURULENT LEPTOMENINGITIS.

Widal has recently described cases of this condition. The clinical course is either slow and latent, but more often begins with acute and typical Meningeal symptoms. It seems to occur occasionally as a complication secondary to many acute disorders, - gastrointestinal, pulmonary, etc. - as well as under unknown circumstances.

The cerebrospinal fluid although thick and purulent by lumbar puncture, is absolutely sterile. Widal quotes as a useful corroborative feature the fact that the polymorph. cells present show a perfectly distinct, unfragmental appearance of the nucleus and cell protoplasm generally, in contrast to the disintegrated and indistinct forms seen in ordinary suppurative meningitis.

PROGNOSIS.

Is excellent, most cases recovering completely.

MENINGOCOCCAL CEREBRO-SPINAL MENINGITIS.

Up till recently under this head were included 2 diseases.

- (1) Epidemic. (due to *Diplococcus intracellularis meningides* of Weichselbaum, discovered in 1887)
- (2) Post-basic Meningitis of infants. (first described by Gee & Barlow in 1878, and shewn

by Still in 1898 to be due to a diplococcus,
 very closely resembling that of Weichselbaum)

During the last few years, however, these have
 come to be looked upon merely as the

{Epidemic} forms of the
 {Sporadic}

same disease, the organism of Weichselbaum, being the
 causative agent in each, slightly modified in the
 latter condition in infants, and the sporadic usually
 being the milder form.

Koplik, in one outbreak of 30 cases during the
 great New York Epidemic of 1904-5, noted 6 cases in
 infants below 2 years, which precisely - both clini-
 cally and bacteriologically - corresponded to the "Post
 basic" disease, and conversely severe sporadic cases,
 in infants or not, may shew the severer symptoms, and
 also the bacteriological findings, of the Epidemic
 form. One thing is certain, and that is the special
 susceptibility to the sporadic form of infants below
 2 years, for which reasons will be given shortly.

The main clinical differences, and their gradual
 assumption of the epidemic type as age advances, were
 well shewn by Langnead, who had collected 10 cases of
 typical "Post-basic" in children over 3½ years.

	<u>Rash.</u>	<u>Temp.</u>	<u>Optic Neuritis</u>	<u>Amaurosis.</u>
"Post-basic" in infants.	Very rare.	Usually slight or no rise. Higher and irregular if fatal.	7% (Barlow & Lees)	At least 33 ¹ / ₃ %
"Post-basic" in older children, over 3½ years.	2 in 10 had Herpes.	8 in 10 irregularly intermittent.	5 in 10	1 in 10
Epidemic form.	6 in 30 Purpuric 5 in 30 had Herpes.	No fixed type, usually high, sometimes intermittent	about 15%	Very rarely.

The cultural differences are very slight; Still's organism usually

- { Grows more rapidly and strongly on agar,
- { Grows well on broth,
- { Has much greater vitality,

than that of Weichselbaum.

These distinctions, however, are being gradually minimised by careful study, notably by Netter. He has grown those of each form in an identical manner, and kept that of Weichselbaum actively growing for 4 years.

Their Agglutination and Opsonin Reactions differ, i.e. the serum from epidemic cases, though agglutinating the organisms for, epidemic cases in other parts of the country, will not do to those from Sporadic cases.

Ker sums up by saying "The differences are so slight that they represent a modification of characteristics rather than a distinction in kind."

A possible cause for these modifications is as follows:-

Fowler, with Stuart Macdonald, pointed to the likelihood of the organisms in the Epidemic form reaching the central nervous system through the gastro-intestinal tract, and not through the cribriform plate of ethmoid, basing their statement on the following facts:-

- (1) The frequency with which diplococci are isolated from the nasopharynx, both in actual cases and in "Carriers".
- (2) The pathological findings point to lesions of cord being older than those of brain.
- (3) The very early and constant abolition of the abdominal reflex.

These, together with the following:-

- (4) The eustachian tubes in infants are shorter, wider, and less straight than in elder patients
- (5) The remains of the Petrosquamosal suture may remain unossified for the first 2 years of life, thereby allowing direct communication between the middle ear and Meninges,

indicate the possibility that, in infants, the infection is through the middle ear direct to middle fossa

of base of skull, whereas the organisms in older patients become modified to a slight degree during their passage through the gastro-intestinal tract and thence to Spinal canal either by lymphatic or blood stream.

SPORADIC CEREBROSPINAL MENINGITIS.

Of this class, the cases between 1900-1909 numbered 81, mostly infants.

Of these:-

- (a) 9 were discharged apparently cured (11.2%)
- (b) 27 were discharged with some Pathological condition still present. (33.5%)
- (c) 45 Died in Hospital.

Of (a) 8 were seen again later:-

	<u>Case.</u>
1 was perfectly well after 4 years.	15
1 " " " " 2 "	6
2 were " " " 1 year	5 & 7
1 was " " " 10 months	8
1 " " " " 3 "	11
1 " " " " 2 "	9
1 " " " " 6 weeks	10

Of these, however, Cases 5, 6, 15 were over age of 3 years.

Of (b) 15 were seen again later:-

10 had Hydrocephalus, apparently progressive, except in	}	Cases
(1 Died suddenly 3 years later)		16 & 17
(1 " of Diphtheria 2 years later.)		later.)
2 became Deaf-mutes		21 & 25
2 Mentally Deficient		27 & 28
1 Spastic Diplegia		30

Of (c) As many Post-Mortem reports as possible were consulted, the most marked feature found being the almost universal occurrence of Hydrocephalus with blocking of the Cerebrospinal foramina below 4th Ventricle by basal adhesions. (Lees and Barlow found this distinctly present to greater or lesser extent in 38 out of 50 fatal cases.)

In addition to above, 6 cases came up to Out-patients for treatment of Sequelae, viz:-

	<u>Case.</u>
1 Spastic Diplegia	31
1 Mental Deficiency	29
1 Severe Headaches (died suddenly)	18
2 Deaf-mutism	20 & 26
1 Persistent Ataxia	34

Synopsis of Cases.

Age	Cure	Some Pathological condition present on Discharge.	Death
Below 1 year	3	18	36
Between 1-2 years	3	5 { 2 Hydrocephalus 1 Deaf-mute 1 Secondary Amentia 1 Sudden death	5
Older than 2 years.	3	4 { 1 Deaf-mute 2 Spastic Diplegia 1 Persistent Ataxia	4

This shews special preponderance (71.6%) of cases in infants below 1 year, with by far the highest percentage of deaths, and also discharged incapacitated

In this latter class we have { 2 Deaf-mutes
2 Mentally defective
1 Sudden death after
(apparent recovery.

the rest under this age being sent out either with progressive Hydrocephalus and emaciation, or else moribund.

Thus only 5.3% had any chance of complete recovery.

It is also seen how the prognosis improves in every way the older the child at the onset, especially if the difficulty of diagnosing a primary encephalitis before the use of Lumbar puncture be considered, as Case 30 in particular might well have been due to this disease alone.

Pathology.

This will be gone into under separate headings. Some authors, notably Hildesheim, ascribe all sequelae to an apparent or concealed hydrocephalus. The latter says - "The most frequent and fatal complication is "Hydrocephalus. In nearly every case which withstands "the first onslaught of the disease, even in children "apparently recovered, this is shewn by an increased "growth of the head or, if this be completely ossified, "by convulsions, insanity, paralysis, incontinence, or "low moral tone."

This seems an exaggerated view, in that 4 of my cases were seen 1-4 years after the initial meningitis, and none of them shewed any sign of this complication, and neither did the 4 children seen after lesser periods. In addition, one of them died from acute

nephritis a year later, but no hydrocephalus, etc. was found P.M.

Cohn also describes a severe case, completely cured, dying some time afterwards of another disease, no pathological findings whatever being found P.M. in the brain or its membranes.

Moreover, as an acute inflammation of meninges can hardly be imagined to exist without coexisting involvement of subjacent brain tissue, this latter condition, if marked, would naturally in process of healing bring about sclerotic changes, and therefore contraction of tissue, as well as adhesions between membranes.

One must remember, however, the possibility of a latent hydrocephalus, as in Case 18, where severe headache and sudden death followed after a period of 3 years of perfect health.

Prognosis and Treatment.

Until Lumbar Puncture was advocated in 1893, the methods of treatment were vague and unsatisfactory; hot baths, counterirritation to head, and drugs such as mercury having little or no effect on progress of disease.

From this time, however, a more rational line of treatment has come to be adopted, the first advance being the systematic withdrawal of the pathological

cerebrospinal fluid, at first only with symptoms of increased intra-cranial pressure, and then as a routine in every case.

Koplik reported 21 cases treated thus between 1899-1903, mortality being 38% all over, only 13% in cases above 2 years, while all 6 cases under 1 year died.

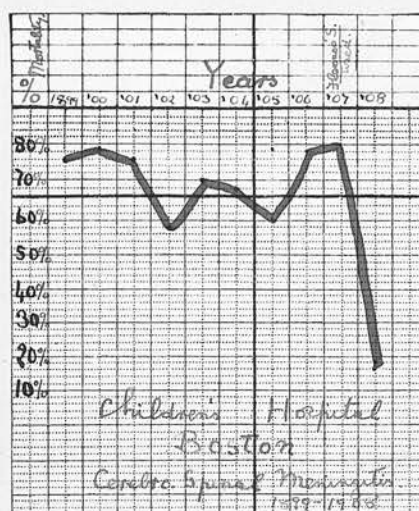
The next step was the use of various anti-meningococcal sera, such as Ruppell, Kollé and Wasserman, at first subcutaneously, and latterly directly into the spinal canal. Various isolated cases were reported, but Koplik again shewed 13 cases treated thus between 1903-5, ages being between 3 months and 11 years.

8 recovered completely, 2 were improving rapidly, 2 died, and 1 was discharged "improved."

In 1907 Flexner introduced his serum (see under Epidemic) and although the agglutination and opsonin reactions are not quite similar in the Epidemic and sporadic organisms, this antiserum is of great use in both forms. The chief points are early and free injection after withdrawal of an equal bulk of the infective cerebrospinal fluid, with frequent repetition if the symptoms do not abate.

Dunn's mortality is charted below; average was about 20 cases per year for the last 10 years. The

death-rate will be noted as dropping to 19%, after running from 58-80% in non-serum treated cases.



Koplik treated 13 cases with Flexner's serum, all sporadic, and 2 were very severe.

Age	Cases	Cure	Death
Below 1 year	3	1 aet. 10 months. Had chronic Hydrocephalus.	2
Between 1-2	3	3	0
Between 2-11	7	7	0

Again the mortality will be noted as very great below 1 year of age, but several cases of cure in infants have been reported, e.g.

Rosewarne of a baby aet. 5 months with typical attack clinically and bacteriologically.

40 cc. Flexner injected in 3 doses, and child recovered perfectly. No sign of Hydrocephalus etc. 2 months later.

Only 1 of the present cases was treated with Flexner, and that after hydrocephalus had shewn itself and with no benefit. Intra-ventricular injection of serum has been tried unsuccessfully in this type of case.

1 was cured after treatment with Ruppell's serum (Case 5)

CASES

of apparent Cure

CASE V

M.K. aet. 4- (see Fowler, Journal of Neurology & Psychistry. January 1909).

Typical case of severe sporadic form, clinically and bacteriologically, treated with Ruppell's serum intraspinally.

Seen 9 months after discharge.

Is perfectly healthy child, very excitable and frequent night terrors. Very bright. Head well formed, circumference 20" (average 20" - 20 $\frac{1}{4}$ ").

CASE VI

J.K. aet. 3.4.

Moderately severe attack, with complete blindness and bacteriologically verified. Became very

emaciated, but after 3 weeks chronic illness began to improve, and discharged month later well, walking and seeing perfectly, and putting on flesh. The actual date of return of sight was not noted.

Seen 2 years later.

Been very well. Went to school aet. 5, and is not backward in any way. Sees well, and fundi are normal. Head well formed, circumference $20\frac{1}{4}$ " (average 20" - $20\frac{1}{2}$ ").

CASE VII

F.M. aet. 4.

Illness commenced suddenly with convulsions, some fever and sudden screaming fits. Head retraction gradually became very marked, with tense fontanelle. $\frac{2}{3}$ ss. fluid removed by Lumbar Puncture.

After 3 weeks symptoms began to pass off, and after one remission for few days, was discharged well.

Died 1 year later of acute nephritis, having suffered no ill effect from the meningitis.

CASE VIII

J.B. aet. 1.3.

Ill for 3 weeks, vomiting, drowsiness, loss of weight, frequent screaming fits, twitching of face muscles, and champing of jaws. Fevered irregularly, neck stiff, and at times firmly retracted, and some

general rigidity. Completely blind, no sign of hydrocephalus, but cerebrospinal fluid under medium tension. Gradually symptoms passed off, and was discharged after 1 month hearing and seeing well, and no sign of hydrocephalus.

Seen 10 months later.

Doing very well, putting on weight, no abnormality present.

CASE IX

T.G. aet. 5.

Sudden onset, vomiting, head retraction, drowsy with screaming turns, limbs stiff and flexed.

Fontanelle bulging and child quite blind.

Gradually symptoms passed off completely, and was discharged after 2 months very well, gaining weight and certainly seeing.

CASE X

R.I. aet. 1.9.

Sudden onset, vomiting, constipation, drowsy but with screaming turns and irritability if touched. Head retraction developed, with rigidity of legs, and Kneejerks +. Pupils reacted sluggishly; distinct double optic neuritis. Had 1 fit, twitching over left side followed by limpness, and unaffected by Lumbar puncture at the time. Lost much weight, but symptoms gradually cleared up, including the optic neuritis.

Left hospital 6 weeks later, very well, and no sign whatever of hydrocephalus.

CASE XI

C.D. aet. '5.

Sudden onset during convalescence from cold; vomiting, little fever, drowsy, but very irritable if touched. Marked head retraction with opicthotonos, bulging fontanelle, and internal strabismus of left eye. Cerebrospinal fluid turbid, great increase in Polymorpho.

After 5 weeks began to improve, symptoms all passed off, and after 2 short remissions was discharged well.

Seen 2 months later.

Very well, no sign of hydrocephalus or other change.

CASE XII

J.S. aet. 3.3.

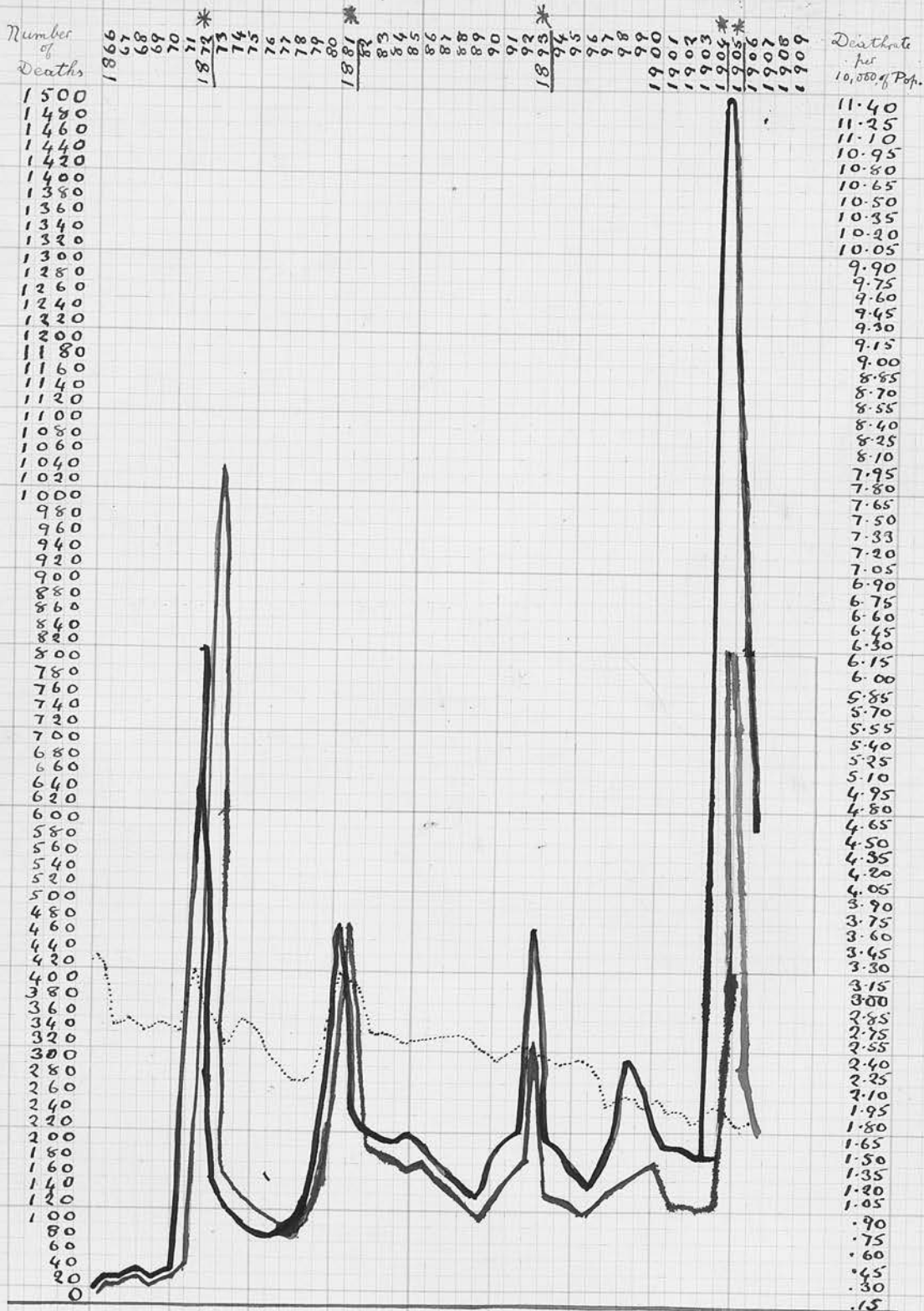
Sudden onset with vomiting, drowsiness, but great irritability, irregular fever. Head retraction developed, squint, diplopia at times, with marked Photophobia, and after 3 weeks was very emaciated, with general rigidity, and unconsciousness. After 7 weeks illness symptoms began to pass off, and finally left hospital very well.

Seen 4 years later.

Been very well, bright, and not backward in any way. Went to school at 5, and now, aet. 7.10, is head of "infants' class" (average for 1st. Standard 7-8).

No sign of hydrocephalus, and sees, hears, runs, etc. well, and is a strong healthy boy.

Old City of New York. 1866-1909



Number of Deaths from Cerebro Spinal Meningitis.
 General Death Rate
 Death Rate from C.S. Meningitis (per 10,000 of Pop.)

EPIDEMIC CEREBROSPINAL MENINGITIS.

The mortality in this disease has always been high, and also sequelae have been associated with it since 1837, when deafness of this causation was first described. The incidence is charted below, with death rates, in New York City since 1866.

The death rate in Edinburgh epidemic of 1907 was 78% over 112 bacteriologically proved cases:

60% of all these cases were under 10 years of age, and the mortality below 1 year was 91.3%

That in the great New York Epidemic of 1904 - 5 was 73.5%, and only 84% of recoveries were completely cured (Billings).

Over various German, French, Portuguese and Nigerian epidemics, and those in Glasgow and Belfast, the case mortality varied between 58 - 80%.

Koplik's experience was that 100% of children below 1 year died. He based this statement on 23 cases, 18 dying and 5 discharged with progressive Hydrocephalus and emaciation.

Holt never saw one under 1 year old recover, and he lost 83 in one epidemic.

During the 1907 epidemic, 61 cases were admitted to the Children's Hospital, but if not immediately fatal, were sent on to the City Fever Hospital for treatment there:—

Synopsis of Cases.

Age	Cases	Cured	Died
Under 1 year	22	0	22
Between 1-2 years	9	1 (complete & permanant after 2 years)	8
Between 2-12 years	30	13. Of these 9 were seen from 1-2 $\frac{1}{2}$ years afterwards. 6 were absolutely healthy and 3 were deaf, -2 of which were also mute, and the other having an ankylosed anklejoint.	17

In addition, 3 more were seen for treatment of the sequelae later:-

	Case
1 Deafmute	23
1 Persistent Convulsions	33
1 Paraplegia (passing off)	32

The treatment, as in Sporadic, was vague and unsatisfactory until the introduction of lumbar puncture, and later the use of various anti-meningococcic sera by Kollé, Wasserman, Ruppell, Merck, and others, with varying success until it was employed by intra-spinal injection, the subcutaneous method having been given up.

More recently Flexner, at the Rockefeller Institute, and Dopter at the Pasteur Institute, have produced Antisera apparently of much greater utility in this condition than any of their predecessors.

The former, first used in 1907, is obtained by utilising the horse as provider of serum, the process of immunisation being long and difficult, vaccines being first injected, and then live cultures with injection of an autolysate prepared from the diplococcus alternating with it. Injections are made thus about once a week.

The serum has a distinct bactericidal action, and must therefore of course also have an anti-endotoxic power; hence the necessity of injection directly into the seat of disease.

The main points in its application are:-

- (1) Early and sufficient doses (circa 30 cc) repeated at intervals of about 24 hours till the symptoms yield.
- (2) Withdrawal of about as much of the infective cerebrospinal fluid by the lumbar puncture through which the serum is injected later.
- (3) Similar treatment of relapses.

The Results may be summarised as follows:-

- (1) Fall in Temperature, usually following first injection.
- (2) Great improvement in general condition, and rapid amelioration of symptoms, rigidity being last to disappear.
- (3) Cuts disease short, within 11 days on an average, instead of some weeks.

- (4) Cerebrospinal fluid very soon loses turbidity; organisms are much diminished in number, become mostly intracellular, and of slight vitality. Exudate rapidly clears up.
- (5) Relapses are few, and yield easily.
- (6) Recovery is complete.

Actual Case Mortality.

In the early days of serum treatment, this was brought down to 35.45%. To shew how prognosis has improved more lately, I append Flexner's figures for all cases, 523 in number, treated with his serum up to January 1st. 1909 (Holt).

He issues serum free of all charge, only stipulating that a complete report of each case treated should be returned.

Age	Cases	Recovered	Died	Mortality
0- 2 years	59	34	25	42.4%
2- 5 "	88	63	25	28.4%
5-10 "	104	88	16	15.4%
10-15 "	70	54	16	22.9%
Total				
0-15 years	321	239	82	25.2%
15 and over	202	129	73	36.1%
All Ages	523	368	155	30.8%

Of cases under 1 year, 25 were treated; 13 (52%) recovered completely, 6 of these being injected during first week of illness, and only 1 injected so soon died. Of the fatal cases, 4 shewed signs of Hydrocephalus before treatment began.

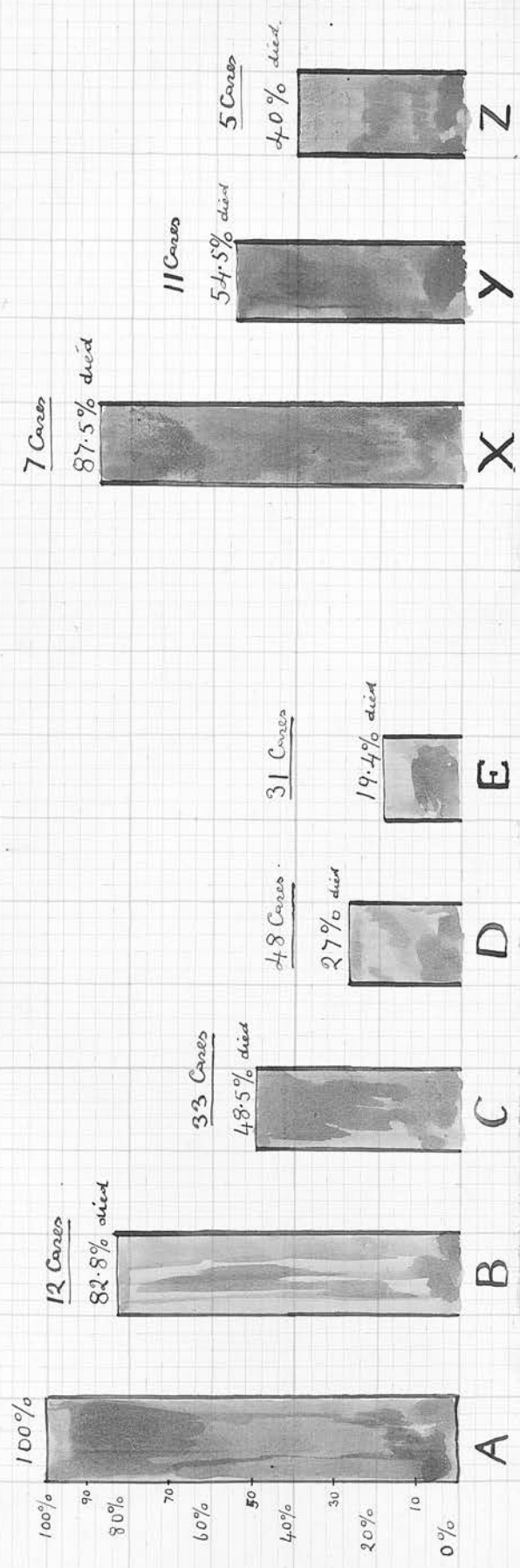
This, of course, includes all the earliest cases, and more recently, since the serum has been more easily available, and better understood, results have correspondingly been better.

Netter results are shown below, and the contrast between serum and nonserum treated cases is marked.

In addition, his last cases, 50 in number and all treated with Flexner, show a case-mortality of only 18%, or 8.89% if cases moribund when first seen, or dying from intercurrent disease are excluded. $\frac{1}{3}$ of these cases were under 2 years of age, and mortality was 15.4%

Mortality in Cerebro-Spinal Meningitis

Below 1 year of age



B Cases in 1909 that did not receive Serum.

C C.S.M (Weichselbaum) treated with Serum 1898 to 1908

D C.S.M. treated with the Serum of 1909 {Dopter, Kille, Wassermann, etc.}

E C.S.M. treated with Fleener's Serum, 1909

X Cases treated without Serum. 1899-1908

Y Cases treated with Serum in 1909.

Z Cases treated with Fleener's S 1909

Netter.
Paris.
91 Cases.
about 300 Cases in all obtained up to end of 1st 3 months of 1909.

Dopter also reports 196 cases treated during 1909 with his own Serum (nature not accurately known)

Mortality was only 15.8%, or only 10.3% if cases moribund when first seen, and those dying from inter-current disease are excluded.

COMPLETENESS OF RECOVERY.

Non-serum treated cases.

Many recoveries were marred by some lasting sequela, the frequency being given as

16% by Billings, in New York Epidemics.

23.5% by Netter in Paris.

The large number of deaf-mutes in Germany, Austria and surrounding parts where spotted Fever is so prevalent bear this out, also the fact that the Glasgow authorities found it necessary to start a new Deaf and Dumb School because the number of these children increased so markedly after the great Epidemic in 1906-7 in that town.

In Bart's Hospital Reports 1876, deafness is said to "usually follow".

Cohn at Posen collected 27 cases which recovered during their Epidemic of 1905-6-7; mostly treated with vaccines, and the case-mortality was 61%. He divides these 27 into 3 classes.

<u>Mild.</u>	<u>Moderate.</u>	<u>Very severe.</u>
8 Cases, all children, and all completely recovered, now being normal children.	15 Cases. 3 complained of weakness of limbs. 3 complained of Impaired Memory. 2 Diminished Intelligence. Several had Headache on exertion, stooping, etc., but in several paralysees completely disappeared in time. 1 adult shewed Hydrocephalus some months later, and 1 child shewed Progressive Hydrocephalus & Idiocy.	4 Cases. 3 were absolutely Deaf, and in 2 of these there was a co-existing Facial Paralysis. 1 of them died a year later of diphtheria, but no pathological findings (Naked Eye) The 4th case (a child) recovered completely.

From this he adduces that the milder the attack the better the prognosis, and also that the recuperative power is greater in children. Against this is the fact that in children only the mild cases survived the initial attack unharmed, with very few exceptions.

Serum-treated Cases.

There is no doubt that the risk of sequelae is remarkably lessened, the more so as the urgent necessity of early treatment is recognised, eg. Robb treated 90 cases, with a recovery rate of 71%. Of these, 4 were deaf, but 3 of them had shown it before any injection was given. The other shewed signs of Mental Impairment later.

Netter has reduced the figure from 23.5% to 2.85%

Vaillard remarks on "The rapid clearing up of the

disease, leaving no sequelae after it."

Holt, commenting on Flexner's last report, said "Sequelae are very infrequent, probably due to shortening of disease. When they have occurred, it has been in cases treated late, and often before serum was injected at all. In almost all other cases not fatal, recovery has been complete."

In Flexner's series of 307 Recoveries, there were 19 Cases of Sequelae:-

12 { all under age of 12 except one }	Deaf; one of these (aet. 11) also blind, and was in this condition before being treated first on 22nd day.
2 (aet. 12 and 17)	Sudden Death on 35th and 130th day respectively from concealed Hydrocephalus.
1 (aet. 9 years)	Blind of 1 eye from Iridocyclitis.
1 (aet. 9 years)	Severe Kyphosis.
1 (aet. 25 years)	Mental Impairment.
1 (aet. 24 years)	Severe Headaches.
1 (aet. 18 years)	Protracted Arthritis and Choroidites.

Netters series of 48 cases shewed only 2 with Sequelae.

1 (aet. 6 years)	Deaf.
1 (ditto.)	Paralysis of Lower Extremities, with Neuritis; gradually improving.

Thus we have 355 cases in all, and only in 5.4%
was there any sequela.

In only 3 of the cases was the time of onset of



sequela noted, - very difficult in children - and here deafness shewed itself first before the 3rd day. Many of the cases were not injected till late in disease, and 5 of them were noted as "Exceptionally Severe."

It must be remembered, as before, that these include the first cases treated, before the necessity of early injection was so emphasized.

I shall now proceed to deal with the different Sequelae separately and in more detail, also giving illustrative cases of each. First I have placed a résumé of the Pathological conditions usually found Postmortem, drawn from the works of various authors, notably Councilman, Mallory and Wright, who had a large number of cases in New York.

It will be seen that all the cases described followed Meningococcal Meningitis, either epidemic or sporadic, therefore it is the pathology of this form specially which is gone into.

PATHOLOGY.

The process is one of inflammation of the Pia Mater with a seropurulent, fibrinopurulent, or purulent exudate most marked over base, and with gradually increasing œdema and thickening of the membranes.

This process also invades

(1) Tissue of brain or spinal cord.

(2) Substance of cranial or spinal nerves, microscopically shewing the usual infiltration with cells and organisms, most marked immediately below meninges and round the dilated blood vessels.

There are often extensive areas of actual softening with haemorrhages, and the neuroglia shews acute proliferative change, very marked round ventricles. The surfaces of the latter are usually soft and mushy, with varying amount of purulent fluid which in more chronic cases, usually with adhesions about base of brain and great dilatation of ventricles, may be quite clear. The evolution of the latter condition was well described by Von Ziemssen.

The 2-6-7-8-Cranial Nerves are most affected, and the inflammation can definitely be traced outwards from the brain along their sheaths. In acute cases the 7th and 8th nerves may be imbedded in a mass of pus, the 8th being most disintegrated; its sheath being often softened and broken down, and the nerve fibres themselves infiltrated, the internal ear being filled with pus.

In chronic cases the exudate is gradually converted into dense cicatricial tissue, causing

further adhesions, and with final shrinking and drawing together of the parts. It may be, however, practically completely absorbed in time, so that no sign of inflammation is left.

In sporadic cases the processes are usually less acute, and in infants better limited to the subarachnoid space about base of brain. Many of the immediate symptoms - head retraction, amaurosis, vomiting and screaming fits - may be attributed to irritation of nerve tissue in this neighbourhood (Ferrier).

The accepted view now is that many cases of "congenital" hydrocephalus are really due to meningitis in very early life - either intra or extra-uterine. This is borne out by the fact that many cases of secondary hydrocephalus follow a very mild, but recognised, attack of true meningitis in later life.

ACQUIRED HYDROCEPHALUS.

The frequent presence of this condition is undoubted.

Of the 27 cases of sporadic cerebrospinal meningitis sent out I.S.Q., 22 showed it to a greater or less extent, and in many of these it was

rapidly progressive to a fatal termination.

Lees and Barlow, in 50 Post-mortems, shewed a great dilatation of the Ventricles of Brain in 29 cases, and in only 8 was the accumulation noted as slight or barely recognisable.

Although I did not see a case of it noted as occurring after the Epidemic form, cases have frequently been reported in the literature, notably by Joslin, Knox and Sladen, Cohn, Koplik, and others; in some the presence of this complication was not suspected till the condition was seen after death, so indefinite was any symptomatology which might have been present previous to this.

In 35 cases of Hydrocephalus, admitted or seen for this condition, the primary condition was noted as follows:-

"Congenital"	19
Secondary to C.S. Meningitis	7
Birth Injuries	3
With Congenital Syphilis	3
With Cerebral Tumour	2
Misplacement of Cerebellum	1

VARIETIES

(1) Apparent.

(2) Concealed.

Occurring where cranial ossification is complete

May assert itself during acute illness, during convalescence, only after some months, or may cause sudden death, following apparent good health.

Classical symptoms are:- Paroxysms of severe headache with vomiting, irregular fever, pain in back of neck and extremities, various paralyses, convulsions and coma.

CAUSATION.

3 Theories are given.

(1) Obstruction.

Here there is some impediment to the free outflow of cerebrospinal fluid from ventricles, usually by obliteration of foramina at outlet of 4th Ventricle. The normal nature of the cerebrospinal fluid in many cases (Lees & Barlow) bear this hypothesis out. Possibly there may also be obstruction to veins of Galen from spread of inflammation to velum interpositum, and hence interference with venous, and therefore cerebrospinal fluid, return. It is unlikely, however, that this last is of much importance, as these veins - return flow from Choroid Plexuses - are very small compared to their arteries, and are deeply withdrawn from the ventricles. It has been shown that the Pacchionian bodies are the

true passage-way for the cerebrospinal fluid into the blood-stream.

(2) Inflammatory.

In most Postmortems, the ependyma of ventricles and epithelium covering choroid plexuses are seen to be thickened and granular, indicative of inflammation. This, of course, means increased secretion of fluid, and this cause is borne out by the altered nature of the fluid-increased albumen etc - even in simple serous meningitis, where the tension, as estimated on lumbar puncture is also increased.

(3) Paretic Dilatation of Arteries to Choroid Plexuses.

These plexuses of the Lateral Ventricles have a double arterial supply viz:- anterior from Internal Carotid, and posterior from Basilar artery. If from inflammation about these trunks, a paretic dilatation of vessels was caused, naturally an increased secretion of cerebrospinal fluid would take place. This view was advanced to meet the fact that in Tuberculous Meningitis, where the inflammatory changes are marked in the anterior fossa of skull, hydrocephalus of some degree is a constant feature. It is unnecessary, as in this form of meningitis there is a very constant corresponding inflammation in the ventricles

themselves, as Cause (2) holds.

Probably the Inflammatory and Obstructive causes go together in most cases, viz:-

First we have the acute Hydrocephalus at very onset of the meningitis, and if the excess of fluid thus secreted is got rid of at once by either natural or artificial means (lumbar puncture), no harm may result. This happy termination will be seen to depend on absence of early basal adhesions, obliterating foramina from ventricular chain, although doubtless this may occur at very beginning of the acute illness.

Secondly, if the inflammation passes off with no obstruction so far, there is the later possibility of obliteration of these foramina by cicatrication and contraction of the affected parts, and consequently damming back of the cerebrospinal fluid.

We do not know what excess of fluid can be removed by natural means, if no obstruction is present. Once the distension of ventricles has attained a certain magnitude, there is additional blocking caused by squeezing of brain tissue, and consequently of the thin-walled veins and lymphatics, between the growing accumulation and the unyielding cranium. As there must be a disparity between the arterial (in choroid plexuses) and venous (therefore cerebrospinal fluid) pressures in order to maintain the cerebral circulation at all, the secretion of cerebrospinal fluid will

continue, with corresponding exaggeration of the hydrocephalus.

It is doubtful if any retrogressive change is possible if obstruction is present. Some cases (16 - 17 - 18) shewed apparent quiescence for 2 - 5 years, but 2 of them died very suddenly, and the other died of diptheria,-no Postmortem obtained.

The presumption is that in the chronic cases - in well-nourished and intelligent individuals with hydrocephalic heads - the brain has accommodated itself to the new conditions perfectly, and that no obstruction to the normal cerebral circulation is present. Ruffer notes the case of a very brilliant medical man aet. 33 with an enormous hydrocephalic head!

Lees & Barlow describe a case of apparently quiescent post-meningitic hydrocephalus dying months later of an intercurrent malady, and here a cystic and degenerated condition of the choroid plexuses was found.

The special tendency to the development of hydrocephalus seen in the sporadic form in infants may be explained as follows:-

- (1) The great chronicity of the disease - not at once fatal owing to its comparative mildness - together with the concentration of the disease about the base, and the special vulnerability

of the tissues.

- (2) The more abundant secretion of cerebrospinal fluid to supply the rapidly developing nerve tissue, together with the fact that the Pacchionian bodies are less developed early in life, would tend to the easier derangement of the normal cerebral circulation.

EFFECTS OF HYDROCEPHALUS.

After complete ossification of skull, this complication may assert itself weeks or months after apparent cure of the meningitis with symptoms previously noted, and will cause death after a more or less chronic illness.

Sudden death may follow after a similar period of absolute health, and no lesion being found P.M. except the ventricular accumulation (Case 18): the cause of death being either due to cutting off of blood supply to, or to direct pressure on, the vital centres in medulla. In the case mentioned death was preceded by severe headache for some weeks, and in any case of this nature the possibility of an exacerbation of the previous ventricular inflammation must be considered (see Serous Meningitis)

Before the cranial sutures are ossified, the

increasing tension can cause expansion of the vault, and therefore symptoms are usually less acute.

That all sequelae are due primarily to an acquired hydrocephalus is negatived by the following:-

- (1) The pathological findings in fatal cases.
- (2) The great tendency for sequelae to pass off, thus indicating a paralysis of function rather than a destruction of tissue, Deafness being the notable exception, but it appears very early in the meningitis, and here (1) is very definite.

Temporary amaurosis in infants, and not in adults surely depends on the more delicate and less fully developed visual centres in the former?.

Moreover, amongst others, 2 of the cases of mental deficiency (27-28) had definite blindness during the acute illness, but later saw perfectly, with no fundal change, and now, after nearly 10 years exhibit no sign of hydrocephalus whatever.

- (3) Special sense affections are not common in chronic hydrocephalus.
- (4) None of cases of sequelae here described exhibit any of the signs of increased intracranial pressure - convulsions, generalised paralyse, etc - together with the usual mental

condition of hydrocephalus.

Case 18 shewed P.M. a very marked hydrocephalus, the sac exercising considerable pressure on optic chiasma and tracts, and this child had never had any visual trouble whatever.

TREATMENT.

Operative means - the only rational form - have so far been very discouraging once the hydrocephalus is definitely established.

The importance of early, and, if necessary, repeated withdrawal of the excess of fluid by lumbar puncture in the acute meningitis has been emphasised.

Later on, if the ventricular system is cut off from the spinal canal by adhesions, no benefit could result from this; moreover no success has attended ventricular puncture direct. Little or no benefit has been demonstrated by ligation of the carotid arteries.

In 1898 Watson Cheyne and Sutherland, bearing in mind the principles of the cerebral circulation as laid down by Hill, first proposed drainage of the lateral ventricles directly into the subdural space. They shewed one case with marked improvement; after 6 months there was diminution in size of head, and no untoward symptoms whatever, and it is interesting

to note that each lateral ventricle had to be operated upon separately, owing to blocking of foramen of Monro.

Since then, several cases have been reported. Ballance had a cure by this means after hydrocephalus had been present for 6 years. The child, although deaf-mute, was very intelligent, and learning to speak rapidly.

Taylor notes 6 cases, 3 dying, and 3 shewing marked improvement.

The possible success of operation seems to depend on:-

- (1) Early operation, as anything interfering with the development of a child's brain during the first 3 years of life is very serious, the normal development within this time being enormous, including the medullation of pyramidal tracts, etc.
- (2) Must ensure slow, steady, and permanent drainage.
- (3) Minimum of brain manipulation.

ILLUSTRATIVE CASES. (Selected)

CASE XIII. Rapidly Progressive.

Boy aet. 7.

At 5 had moderately severe attack of sporadic

form, and week or two after onset was noticed to be blind.

Head now distinctly hydrocephalic, circ. $16\frac{3}{4}$ " (average 16") and very emaciated.

Died 5 months after onset, head gradually increasing in size, and he remained blind.

CASE XIV. More slowly progressive.

Boy aet. '5 with mild attack of sporadic form.

After 2 weeks noticed to be blind, no other ocular symptoms or signs. L.P. shewed clear fluid under tension, albumen +, and few lymphocytes.

Discharged after '3 with progressive hydrocephalus and quite blind.

Seen '2 later.

Sees. Head heavy and falls back, marked hydrocephalus, all sutures gaping, circ. $18\frac{1}{2}$ " (average $17\frac{1}{2}$ - 18").

Seen '3 later.

Still progressive. Circ. 19" and child is now very emaciated. Sees, and pupils react sluggishly.



CASE XIV^A Slowly Progressive.

Boy aet. '9.

At '3 had some fits, head retraction, and screaming turns. Very irritable if touched, and gradually became emaciated; symptoms slowly passed off, but head began to increase in size.

Now he shows distinct hydrocephalus; circumference of head $18\frac{1}{2}$ " (average 17")

Seen '7 later.

Head increased $1\frac{1}{4}$ " in circumference, sutures closed, but fontanelle wide open and tense. Sees and hears well, is just beginning to walk, and seems good tempered, intelligent baby

CASE XV. Progressive; apparently gradual blocking of foramina at base of brain.

Girl aet. '9.

2 months ago had mild attack of sporadic form; symptoms slowly passed off, but great emaciation; noticed head was growing larger, and now exhibits marked hydrocephalus; head circumference $18\frac{7}{8}$ " (average 17"). Soon after onset was noticed to be blind, and from beginning has been very restless, with frequent screaming turns, and cervical rigidity.

Eyes exhibit slight lateral oscillatory movements at times; pupils inactive; eye-grounds normal.

Lumbar puncture - 20 cc. under great tension removed. Albumen slightly +, no cells or organisms.

After 10 days.

Head measures $\frac{1}{2}$ " less, and probably could see.

After 10 days.

Head increasing in size again gradually; Lumbar puncture been done several times, but no tension of fluid, and no relief of cerebral tension.

Discharged after 2 months, slowly progressive.

Seen after 3 months.

Head increased $1\frac{3}{8}$ " Certainly sees, but does not recognise mother; lies constantly in relaxed, apathetic condition.

Pupils moderately dilated and react sluggishly.

Fundi normal.

CASE XVI. Sudden Death, after apparent quiescence.

Girl aet. '4.

Attack of medium severity and gradually recovered. '3 after onset noticed head growing larger, and that she had been blind since soon after onset. At this time head circumference was $17\frac{1}{2}$ " (average $16\frac{1}{2}$ ") and all sutures widely open. Quite blind; pupils dilated and inactive.

3 months later began to see, and a month later left hospital seeing well, and discs normal. She had lost tension of fontanelle, and was putting on weight rapidly.

After $2\frac{1}{2}$ years.

Just died very suddenly; no cause known. Had

grown well, with no further increase in size of head, and was happy and intelligent, with good sight.

CASE XVII. Death from intercurrent disease.

Apparent quiescence.

Girl aet. 2.9.

At 1.6 had moderately severe attack of sporadic form. After 2.3 weeks, symptoms gradually passed off, but head noticed to be growing too large. This has slowly increased, with great general weakness. Intelligence not altered, spoke as much as before illness, walked with assistance, and held head up well.

Now head circumference $20\frac{1}{2}$ " (average 19"). She is rather drowsy, and speech is becoming more uncertain. Gait steady, and lumbar puncture shewed no excess of fluid.

After 2 years.

Death reported from diphtheria.

Had been very well since discharge, head not increased in size, and fontanelle soon closed completely.

Was not backward at all for age (5).

CASE XVIII. Sudden death, after quiescence.

Boy aet. 5.3.

Complaint. Severe frontal headache, with vomiting

Duration. 2 weeks.

Had severe attack, of sporadic form, at 1.6, but

left hospital after 8 months completely recovered. Began to speak at 1.8, and no sign of disease whatever till 2 weeks ago.

Then headache began, no cause, persistent, evidently severer paroxysms, and relieved by pressure. Also vomiting of cerebral type, not severe, as child was never confined to bed. Has lost weight rapidly.

On admission.

Very drowsy and apathetic, intelligent if roused, and headache still present with slight cervical rigidity. No sign of hydrocephalus, and sight, hearing, gait, muscularity and sensation unimpaired. Pupils dilated, react well. Ophthalmoscopically; slight congestion of veins.

Continued to lose weight, temperature persistently sub-normal (97.6° to 98.2°) and pulse 102, regular, tension average.

Headaches completely relieved with antipyrin, mental condition distinctly brighter, but child died suddenly and unexpectedly 3 weeks after onset of headaches.

POSTMORTEM EXAMINATION.

Well developed child.

Brain (see stereoscopic photographs).

Marked general flattening of convolutions; no recent acute inflammation.

On removing the brain, the infundibulum, which projected as a thin bladder or cyst-like structure, burst, and about 10 ozs. of clear cerebrospinal fluid escaped from the ventricles.

There was distinct old fibrous thickening, especially over the under aspect of the Pons and neighbouring parts of brain, evidently the result of an old basal meningitis.

The cerebellum near the medulla was adherent to the latter, and formed a cone-like projection passing down into the foramen magnum.

The membranes were thickened over the various cisterns, and there was matting of the roots of the cranial nerves.

The distension of the 3rd. Ventricle and infundibulum had caused considerable pressure upon the optic chiasma, and to a less extent laterally on the tracts.

Other Organs.

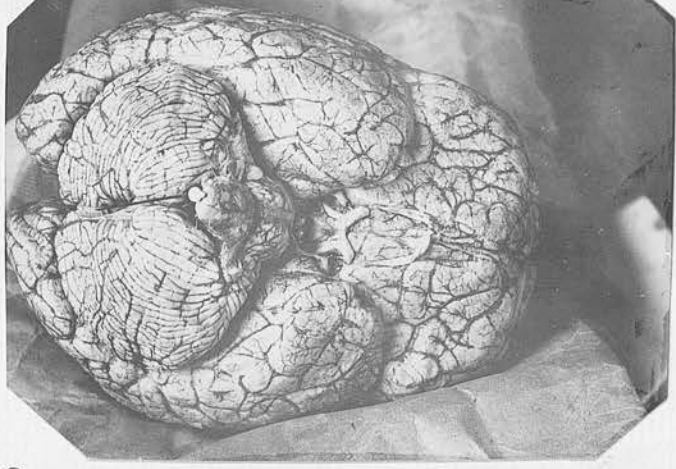
Only some congestion, specially of lungs.

Summary.

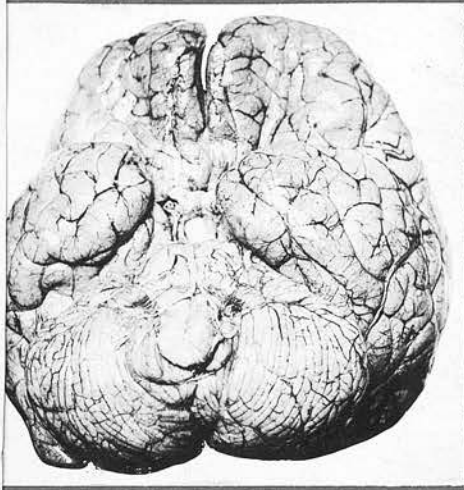
Chronic Hydrocephalus secondary to basal meningitis.

OVER.

Stereoscopic,
With
Case XVIII



Compare Normal Brain.



Base of brain. Infundibular 'Cyst' is ruptured.



Ditto. Section showing great dilatation of Ventricles.

PULL.

PULL.

LOSS of VISION



This ranges from Temporary Amaurosis, seen in infants, to Permanent Blindness from Secondary Optic Atrophy, usually seen in older patients.

The cause has been described earlier as being in most cases actual involvement of the nerve-paths by organismal or toxic action, and not having acquired hydrocephalus as the initial complication, presumably because the optic nerve trunks are stout, and can easily accommodate themselves to the conditions of increased intra cranial tension (vide Case 18)

Boerhaave described the case of a French beggar, who had for some reason been trephined, and used to solicit alms in the bit of his own skull! On pressing over the exposed dura mater, he used to put himself into a deep sleep, "with all symptoms of apoplexy", this being preceded by a phase of specks before eyes, and then complete blindness. On relaxing the pressure, the symptoms passed off in reverse order. This train of symptoms, however, is only characteristic of an acute cerebral anaemia.

Temporary Amaurosis.

This form, described as "a form of blindness where the patient can see nothing, nor can the Doctor," is frequently seen in infants,—the figure already given being 33% of all cases. It is not described as

a symptom in older people, and is not usually seen in a chronic hydrocephalic case.

The light reflex is sluggish, and the pupils dilated as a rule, the presence of the former shewing the integrity of Meynert's fibres, which pass from the optic nerve to the third nerve through the superior quadrigeminal body. The eye-grounds are quite normal (optic neuritis being exceedingly rare) and there is no photophobia.

The symptom usually comes on very early and may pass off in any time up to 7 months (Case 27), there may be no other sequel whatever, nor does the blindness tend to recur.

Of the 8 cases cured, 3 were undoubtedly blind during the acute illness (Cases 6, 8, 9)

Of 10 discharged with Hydrocephalus, 5 were blind during the acute illness. Of these:-

2 continued blind till death, in few months.

3 regained sight after varying periods, although hydrocephalus increased. One of them died suddenly $2\frac{1}{2}$ years later after apparent quiescence and had no relapse as regards vision in this time (Case 16)

Of the other cases, there were blind during the acute illness:-

2 Mental Deficiency (Cases 27, 28)

1 of Persistent ataxia (Case 34)

None of the cases of Deafness were noted as ever blind, and Flexner's returns only shew 1 case with these symptoms in conjunction, here very marked hydrocephalus being also present.

Seat and Nature of Lesion.

Four possibilities.

- (1) Actual inflammation and destructive changes in paths concerned.

This is rendered unlikely by the facts that the blindness comes on suddenly and completely with no photophobia, disappears entirely in a comparatively short time, and that neither clinically nor Postmortem is any pathological lesion found.

- (2) Thrombotic obstruction of vessels.

Unlikely for same reasons.

- (3) Acquired hydrocephalus and compression thereby.

This is negatived by the transient nature of the blindness, by the fact that definite cases of hydrocephalus are not usually associated with it, that in several of my cases the blindness disappeared although the hydrocephalus increased progressively, and that loss of vision need not be associated with any

other symptom of cerebral compression.

- (4) A paralysis of the Visual Centres by a toxæmia, the amaurosis being analogous to that seen in uræmic and other toxic conditions (where the origin of the poisoning material is less definite) and being clinically similar to that functional amaurosis sometimes seen after severe convulsive attacks which apparently is due to temporary exhaustion of these visual centres.

This is probably the true cause, and is strengthened by the nature of the onset, by the lack of pathological findings, and by the fact that homonymous hemianopsia has been described as occasionally preceding the complete blindness. Its prevalence in infants is due to the comparatively slow development of this special sense after birth, hence greater delicacy and less stability of the nerve cells responsible for it. Its temporary nature brings it into line with the other sequelae - paralysees etc. - in which the great tendency is to pass off completely in time.

Permanent Blindness.

This is due to Optic Atrophy secondary to Neuritis, or perhaps occasionally to thrombosis of the retinal artery.

It is not common, considering the frequency with which optic neuritis occurs in Epidemic Meningitis (about 15% of all cases), and may be uni- or bilateral.

Sneller, analysing the causation of 333 cases in a school for the blind, found 10.9% were due to optic atrophy following meningitis; (2 of these cases, however, might possibly have been of cerebral tumour)

As an example of unilateral blindness, I mention the following case:-

Boy aet. 16. Had epidemic meningitis when two years old, but recovered, with no known sequelae.

Short time ago he thought sight was not very good, and on examination was found to have complete blindness with well marked optic atrophy of one eye, the other being perfectly normal.

From the completeness of the vision of the other eye, the lesion must have been in front of the optic chiasma, and this lesion was probably initial optic neuritis resulting in destruction of the nerve fibres either at once or by contraction of the purulent exudate later. In the other eye the neuritis, as so often happens, had cleared up completely, if it had ever been present.

The other affections of sight mentioned are

purely the result of infiltration of the eye itself with infective matter, spreading right forward along the optic nerve.

DEAFNESS

This is universally recognised as being the most common sequel to severe cerebrospinal Meningitis. It may occur with other lasting complications, but is in a vast majority of cases found by itself, - being double, absolute, and permanent without exception as far as is known definitely.

Frequency. Love states that $1/5$ to $1/6$ of all cases of acquired deafness, as obtained from many Europeans and American statistics, are secondary to Meningitis.

From Census taken in U.S.A. in 1889 over 9209 cases of acquired deafness (so defined from Congenital) 31.1% were due to this cause, scarlet fever being the next most common with 29.3%.

Moos of Heidelberg in 1881 reported 64 convalescents from Epidemic Meningitis. Of these:-

38 were Deaf-mute.
 20 " Absolutely deaf.
 5 " "Dull of hearing"
 1 Heard well.

In addition, 32 of them had a staggering gait.

Larszynsky reported 50 cures. 10% Deaf.

With serum treatment, this sequela, as already shown, has markedly become more rare, eg.

Robb shewed 68 recoveries, 7.4% Deaf (no serum) and simultaneously treated

22 recoveries, 4.5% Deaf (serum)

Flexner over 307 do. 5.9% Deaf, but many with his serum. of these not injected till very late in disease, after deafness has asserted itself.

Causation.

Von Ziemssen suggested the cause to be simultaneous middle-ear infection: in view of the fact that the deafness is bilateral and complete, with no sign of disease otoscopically, this view is now discredited.

The following possibilities remain:-

- (1) A destruction of 8th Nerves by inflammation or its after effects.
- (2) Infection of Internal Ear itself, and destruction of its functioning parts.
- (3) Compression by hydrocephalus.

This does not account for the implication of the auditory nerves alone, with no other symptom of increased intracranial tension.

- (4) Thrombosis of Nutrient vessels to 8th Nerves.

Moos suggested this view, based on the facts that the 7th and 8th nerves, though

running together, have a separate blood supply, and that the 8th Nerve is so much oftener involved of the two.

(5) Involvement of Bulbar origin of 8th Nerves.

From the nearness of the 8th Nerve trunks to the seat of most intense inflammation - the base, together with the pathological conditions of these nerves usually found P.M., from the fact that the auditory nerves break up into the finest of filaments before they reach the internal ear, and that the internal ear is an extremely delicate mechanism which would be irretrievably destroyed by any inflammatory change affecting its structural parts, the true causes are in all probability (1) and (2), either together, explaining the early and permanent deafness, or else (1) alone, with post-inflammatory contraction of cicatricial tissue gradually obliterating the nerve fibres themselves (vide Cases 23, 25, 26) thus causing deafness of later and more gradual onset.

The comparative escape of the 7th Nerves depends on:-

- (1) The stoutness of this nerve is preserved throughout.
- (2) It does not end in a highly specialised sense organ which cannot be repaired when once damaged, and so the nerve inflammation, as in other nerves (6th for example) can pass off

leaving no permanent harm done.

Clinical Features.

Voltoolini's disease was formerly thought to be a primary inflammation of labyrinth, but is now accepted as being a slight attack of basal inflammation spreading along auditory nerves to internal ear.

The characteristic symptoms are:-

- (1) A short feverish attack, perhaps with
- (2) Severe cerebral symptoms, followed by
- (3) Deafness, deaf-mutism and staggering gait.

Post-meningitic deafness is very constantly associated with vertigo and disturbance of equilibrium (vide Case 22) accounted for by simultaneous involvement of the semicircular canals, -tinnitus not being complained of as a rule; they are always worst in the mornings on getting up.

Horsley pointed out that these symptoms might be due to:-

- { New growths in 8th nerve, involving cochlear fibres.
- { Certain lesions of Medulla and cerebellum.
- { " " of area of orientation (Posterior 2/3 of Temporal Lobes)

These lesions, however, are deepseated and not due to a meningitis, and therefore may be disregarded here.

Prognosis.

The deafness is, as far as authentically known, incurable.

DEAFMUTISM.

Frequency. In going through the records in the Edinburgh Deaf and Dumb Institution, difficulties arose in arriving at accurate statistics because of the indefinite nature of the histories.

Of 317 cases admitted between 1870-1909 of children between 6-16 years, 5.3% were ascribed to:-

(Meningitis
(Inflammation of Brain
(Water on Brain.

and of these, 3 dated from the 1907 Epidemic.

To Donaldsons Hospital (Deaf & Dumb side), 1488 cases have been admitted since 1854; in 360 the condition was said to be acquired, and, of these, 18% were ascribed to "Meningitis", 4 dating from the 1907 Epidemic.

Naturally many of the latter cases have not yet come under treatment at all, but it is significant that the Glasgow authorities deem a new school necessary in order to educate the children deafmute after the 1906-7. epidemic in that town.

Roth noted in 1874 that all the children in a Deaf and Dumb Institution in Upper Franconia, 42 in all, dated from this disease.

Moos found 38 deafmutes in 64 convalescents from Epidemic Meningitis.

Courtellemont states that half cases of acquired Deafmutism are post-meningitic.

Causation.

Secondary to the absolute deafness, in children who have not as yet learnt to speak. Elder children, (as Case 19) although deaf, do not become mute, and no search for a Central lesion is necessary.

Prognosis.

Seems to depend on 4 factors:-

- (1) Age of patient.
- (2) Degree of deafness (nearly always absolute)
- (3) State of intelligence.
- (4) Treatment followed.

I saw 7 of these post-meningitic cases in the Edinburgh Deaf & Dumb Institutions, and took notes of 10 more; none of them shewed any sign or symptom of Hydrocephalus, Mental Deficiency, Blindness, or other pathological change.

The headmaster assured me that in his experience the post-meningitic cases are particularly bright and intelligent, being easily and satisfactorily taught, and capable of leading useful lives.

The great point is to commence tuition before the deaf child has forgotten all the words, if any, that it ever knew, and from which its education may be built up. Late cases, however, are not hopeless, viz:-

- (a) Had meningitis at .6; no tuition till 11 years old, when absolutely deaf mute. He was discharged after 4 years speaking and lip-reading well, although of course in the expressionless manner characteristic of these patients.
- (b) Meningitis at 3 years; since been absolutely deaf-mute. Admitted aet. 7 years, and after 5 years in school, was discharged lipreading and speaking very well, being highly intelligent.
- (c) Meningitis at 6 weeks; since been absolutely deaf mute, very dull, and "peculiar" in mind. Admitted aet. 12 years, and after 3 years tuition intelligence was vastly improved and was discharged lip-reading and speaking well.

The various methods of teaching deaf-mutes are not considered here.

ILLUSTRATIVE CASES. (Selected)

CASE XIX. Deafness, also ankylosed joint.

Boy aet. 7.5.

Severe epidemic attack with Leptothrix found in cerebrospinal fluid. Treated with Flexner, and slowly recovered, having had pain and swelling in Right ankle joint, earlier, leaving it stiff. Absolute deafness noticed as unconscious state passed off.

Seen 1½ years later.

Well and thriving in every way. Right ankle joint firmly ankylosed, and he is quite deaf.

Speaking as well as he ever did, will read aloud, etc. Very intelligent, no headache or sign of hydrocephalus.

DEAFMUTISM.

CASE XX .

Boy aet. 3.6.

Moderately severe, but prolonged attack of sporadic form, vaguely ill for 2 months, but gradually recovered and was noticed to be deaf.

Seen 1 year later.

Although speaking well before illness, has gradually become mute. Now deaf-mutism is absolute, although he is bright and intelligent, and will run messages, etc.

In other ways is perfectly normal child.

CASE XXI. Coming on after hydrocephalus.

Boy aet. '8.

Severe attack of sporadic form, developing distinct hydrocephalus, with gaping of sutures. Symptoms gradually disappeared, and was discharged well, though quite deaf.

Seen 4 years later.

Thriving well. Cranium ossified, and no sign of

hydrocephalus. Can only utter a few inarticulate noises.

Seen 2 years later.

Strong, healthy, and very intelligent. No sign of hydrocephalus, but is absolutely deafmute.

CASE XXII. With marked staggering gait.

Boy aet. 2.8.

Severe epidemic attack, treated with Flexner, deafness noticed as he was recovering. When began to get about, noticed he staggered and fell a great deal; he often held head as if pained, but was discharged speaking well.

Seen 6 months later.

Quite deaf; no sign of hydrocephalus. Gradually forgetting his words; gait is steadier, though often falls, specially to left.

Seen 4 months later.

Absolutely deafmute, but sharp and intelligent; growing well, very cleanly in habits. Much steadier on his feet, but usually worst in mornings.

CASE XXIII. Late onset of deafness.

Boy aet. 3.

Very severe epidemic attack; ill for 4 months, then gradually recovered completely.

Soon after went home noticed he was not hearing

well, and about 6 months from onset was absolutely deaf. Gait never seemed impaired, no muscular weakness.

Seen 2 years later.

Has gradually lost speech, and is now absolutely deafmute. Bright and clever, no sign of hydrocephalus, staggering gait, or other abnormality.

CASE XXIV. Probable gradual onset.

Girl aet. 2.7.

Severe epidemic attack; gradually recovered, and deafness first noticed during convalescence, onset probably being gradual. No abnormality of gait or other symptom. Speech as good as before illness.

Seen 2 years later.

Well developed and nourished; no sign of hydrocephalus. Absolutely deaf, and has gradually forgotten all words except a few simple ones. Very intelligent and active.

CASE XXV. Deafness came on late; deafmute educated.

Boy aet. 3.10.

Very severe attack of sporadic form, with optic neuritis.

Discharged after 5 weeks apparently cured; seeing well and talking as before illness, putting on weight fast. No sign of hydrocephalus or deafness noted.

Seen 1 year later.

Been quite deaf since "soon" after discharge, and is now gradually losing speech. Very sharp, intelligent, and cleanly in habits.

Seen 9 years later.

Been in Deaf & Dumb Institution for 2 years. Absolutely deafmute, and seemed very dull, on admission. Now lip-reads, and is learning to speak well. General intelligence improving greatly, and headmaster is sure he is not mentally deficient in any way.

CASE XXVI. Deafness came on late. Delayed walking.

Boy aet. 1 year.

Severe attack of sporadic form; gradually recovered, but did not walk for 6 - 7 months, although doing so quite well before illness.

About $2\frac{1}{2}$ months after onset, and being well in every way, noticed he was getting deaf, and in week or two was completely so. Has never spoken at all, but always bright and intelligent.

Seen $1\frac{1}{2}$ years later.

Absolutely deafmute; very intelligent, will indicate, or lead people to, what he wants.

No sign of hydrocephalus; sees, walks, and feeds himself well, but uncleanly in his habits.

MENTAL DEFICIENCY.

I have not included here those cases of mental change associated with progressive hydrocephalus, nor those where neglected deafmutism is the primary cause, and where appropriate teaching has such excellent results.

The clearness of the mind is often a striking feature of an attack of cerebrospinal meningitis, therefore it is not surprising that mental defect is an uncommon sequel to it in older patients, this danger naturally being greater the younger the child.

Neither Bevan Lewis nor Tredgold mention meningitis specially as a cause of amentia, but Loof's statistics shewed that in 3.7% of all idiots the condition was due to cerebrospinal meningitis.

I have notes of only 3 cases, of varying severity. In 2, the children had temporary amaurosis during the acute attack, and one of them had occasional fits for 4 years afterwards, which then completely ceased. All three were late in walking, speaking, etc. but no special sign of cortical irritation was exhibited during the acute meningitis except in the one with severe and persistent convulsions.

The presence of other sequelae does not necessarily imply any degree of mental enfeeblement; the 2 cases of severe spastic diplegia (30-31) although backward through lack of education, were quite

intelligent, with excellent memory.

The Varieties are merely those of degree:-

- { (1) Delayed Development, i.e. slow in speaking, (or 'Backwardness') walking, etc. or Persistent enuresis.
- { (2) Distinct Mental Defect, perhaps very slight often with sluggishness, bad memory, and fits of uncontrollable temper, up to
- { (3) Complete Idiocy.

CAUSATION.

The delayed mental development is only what is seen after many acute illnesses of childhood, and the patient is merely backward, slowly assuming the mentality corresponding to his age, after which no difference is noticeable. The condition is exactly similar to that caused by abnormally slow development of the nerve cells, as of any other part.

Charles West's dictum "that a mentally deficient child would be abnormal for any age, whereas a backward child is merely abnormal for its own age", states the distinction well.

The cause here is probably a degree of paralysis of the nerve cells due to the toxins set free, and which is but slowly recovered from.

As regards the definite grades of mental defect, up to complete idiocy, there must be some degeneration of nerve tissue, - probably from the antecedent meningo-encephalitis with adhesions, etc. in process

of healing causing cicatrisation and consequent contraction of the parts. The seat is likely to be the frontal lobes.

PROGNOSIS AND TREATMENT.

The milder forms of mental defect can usually be very much bettered by special and persevering tuition. Here the Elementary Education Act of 1899 has done good work in instituting special classes, under the School Board, for these children. In the mildest forms, this may make all the difference as regards a useful life, while in the more marked forms may render things better both for the child and his relatives.

For the unimprovable cases, there is nothing to be done except an Asylum or special guardianship.

ILLUSTRATIVE CASES.

CASE XXVII.



Girl aet. .9.

Severe attack of sporadic form, and completely

blind when discharged 10 weeks later.

Seen again aet. 10 years.

Was quite blind for 7 months after discharge, since then sight been perfect.

Had few fits up to 5 years old; none since.

Speaking 1.8. Walking 3.; both as well as any other child.

Went to school at 6 years, and has not missed a day since, and is now in Second Standard, (about average).

Memory is very bad; absent-minded and thoughtless very stubborn, with fits of temper; 'takes up' with children much younger than herself, and will not make herself useful about the house.

Frequent nocturnal enuresis.

Physical examination revealed no abnormality, sight being excellent and fundi normal.

Seen 4 months later.

Much as before. Very dull and slow of comprehension, with frequent nocturnal enuresis.

CASE XXVIII.

Girl aet. 7.

Sporadic attack of medium severity; quite blind for many weeks after discharge though otherwise cured; since then sight been perfect.

Walking 3.9

Speaking 4 years.

No idea of Cleanliness till aet. 6.

Seen again aet. 8.8.

Very babyish in conduct and speech; wont play with other children, but is very easily pleased.

Always restless, will feed herself dirtily, but will not dress herself at all.

Big and strong for age. No evidence of hydrocephalus, but has slight divergent squint. No paralysis or rigidity whatever.

CASE XXIX.

Boy aet. 1.1.

Severe attack of sporadic form.

Seen 2 years later.

Has never 'come on'. Hears all right, but often pays no attention and was brought to hospital because his brother, 18 months younger, was leaving him far behind in general mental development.

Is well developed and nourished, so sign of hydrocephalus and has not the physiognomy of mental defect.

Circumference of head $19\frac{1}{2}$ ".

Speaks only word or two.

Sees and hears well.

Gait is slow and deliberate like that of a younger child; no idea of cleanliness; feeds himself very dirtily; sucks forefinger.

Seen 9 months later.

No increase in size of head.

Improving in every way; holds head firmer and straighter, and more agile on feet, though often falling.

Asking for things more, and will name what he sees.

Memory very bad - will forget mother after day or two's absence.

Still dirty in habits, but will hold water longer now.

PARALYTIC SEQUELAE.

These may be either:-

- (1) Temporary,
- (2) Permanent,

and either localised, viz: of an ocular muscle, or extensive, as a diplegia.

Moreover, the paralysis may be of either the Upper or Lower Motor Neuron type, the former being the more common.

The various and indefinite paralyses seen with hydrocephalus are not considered.

Causation.

Various conditions due either to spasm or paralysis of muscles are common during acute meningitis.

In many cases these disappear as convalescence sets in, but in others the paralyses either remain for a considerable time, (Case XXXII) or are permanent. In addition, paralytic symptoms may not assert themselves for some time after apparent complete recovery (Case XXXI).

In one case (XXXIII) although the paralysis had to a large extent disappeared, the child still has persistent convulsions, after 3 years.

Two conditions are apt to be confused with the original meningeal inflammation, causing mistaken diagnosis:-

- (1) Acute Polioencephalitis,
- (2) Poliomyelitis Anterior Acuta.

As regards (1), the onset of the disease in children is very similiar to acute meningitis, and the diagnosis in many cases can only be cleared up by lumbar puncture. The paralysis is usually absolute from the beginning, and most often is a hemiplegia, although para- and diplegias are also seen. Head retraction is not present as a rule. It must be remembered, however, that there must be a certain amount of underlying brain inflammation even in cases of leptomeningitis.

As regards (2), it is now certain that epidemics of this disease do occur. (Treves; Römer). The latter author was unable to find any organisms in the cerebrospinal fluid or tissues, but produced a like disease in animals by inoculation of the infective nerve substance into their spinal canal. He therefore concludes that the active organism cannot be stained by the ordinary means, and compares it to the virus of rabies.

Courtellmont, on the other hand, described 16 cases of Flaccid Paralysis, occurring in children and in every way resembling poliomyelitis anterior acuta.

The diagnosis of epidemic meningitis was based on the nature of the cerebrospinal fluid, with meningococci in every case, and 50% of these cases were said to be "abortive", the others "ordinarily severe".

The onset of the paralysis was early, sudden,

and not accompanied by pain, except at the very onset, and the constitutional disturbance was, in half the cases, very slight.

Its extent was variable, - like poliomyelitis anterior, - and the course was similar to this disease in that it tended to clear up somewhat, restricting itself to one or more groups of muscles permanently.

We must therefore conclude that paralysees of the lower neuron type may occur in meningitis, due either to a special susceptibility of certain motor cells of the anterior horn, thrombosis of their blood supply, or to a neuritis of the Spinal nerves implicated. The latter brings the condition on to a par with the implication of cranial nerves, but differs in that it is never completely recovered from, as are the cranial palsies (notably 6th and 7th), and probably due only to transient conditions, as oedema.

Hobhouse suggested that the special tendency to paraplegia was due to gravitation of the infective material to the lower part of the spinal canal. Against this is the fact that there is no authentic record of any lower motor neuron type being completely recovered from, and unfortunately the characters of the temporary paralysees (Cases XXXII - XXXIII) are not very definite.

SPASTIC PARALYSES.

The evanescent forms can best be accounted for by

a poisoning of the upper motor neuron and suspension of function, either from a Toxaemia, or pressure from the inflamed covering membrane, with its accompanying oedema. The permanent forms by definite structural changes.

Regarding the latter, the process is:-

- (1) Primary actual destruction of nerve cells by the acute inflammation, or
- (2) Postinflammatory contraction with cicatricial changes either of pia mater, or of brain tissue itself.

I have 3 cases:-

CASE XXX.

Diplegia first noticed as primary acute symptoms passed off, and gradually becoming more marked.

CASE XXXI.

Had 3-4 months of perfect health after the acute meningitis, and then spastic diplegia slowly developed.

CASE XXXII.

Paraplegia, passing off after 1 year.

Seat of Lesion.

Is probably cortical. Borne out by the similarity of these cases to Infantile Cerebral Paralysis due to trauma, haemorrhage, etc. and by the fact that spastic hemiplegia is a recognised sequel to acute Meningitis (Courtellmont). Case XXXIII shows disappearance of paralysis, but persistence of

convulsions, with definite "signal symptom".

Mandoul describes a case of typical cortical facial paralysis, postmeningitic, and without any hemiplegia or involvement of 6th nerve.

The motor tracts might be involved in their superficial course through the crura or pons, here being surrounded by the subarachnoid "cisterns", or by compression in their course through the cord.

The internal capsule has been suggested as the site, on the analogy that tuberculous meningitis is sometimes ushered in with hemiplegia due to spread of inflammation outwards from the lateral ventricle.

CASE XXX. Diplegia immediate sequel.

Girl aet. 3.4.

At 3 years had very severe attack of sporadic form, with numerous fits, and generalised twitchings; the latter still present, although acute symptoms gradually passed off. She now presents typical picture of a spastic diplegia with marked contractures; weak jerky movements, frequent involuntary twitchings of face and limbs, and nystagmus of irregular type being present.

Hearing and sight good, memory unimpaired.

Six months later.

Much as before; can speak better on wakening in morning, but muscles seem slowly to "stiffen up".

Mind quite clear; memory good.

Four years later.

Very little improvement; cannot sit by herself. Apt to spasmodically draw up arms and legs, retract head or give strange inarticulate cries if excited. Can slowly and inco-ordinatedly perform voluntary movements, throwing part into required position. Soft palate is drooped and inactive, but speaking better, with large choice of words.

One year later.

Much as before. Easily made to laugh or cry; but, although little facile, is learning simple lessons.

No headache or fits, and eye-grounds are normal.

Four months later.

Can now pull herself up, and sit a little. Left hand is most used, but movements as before, fingers closing spasmodically on anything grasped; when excited limbs and face are spasmodically jerked about.

Memory quite clear, even for events of some years ago.

CASE XXXI. Diplegia delayed 3-4 months.



Girl aet. 3.3.

Severe attack of sporadic form, with marked convulsions, and unconscious for 3 weeks.

Pronounced almost hopeless (Dr John Thomson), but gradually recovered and eventually could run about quite well and strong as far as known; intelligent, no fits and speaking, seeing and hearing well.

Absolutely all right for 3-4 months, and then it was noticed that legs were becoming weak and "knockknee", and gradually present condition developed.
Seen 4 years later.

Shows typical and wellmarked spastic paralysis of both lower limbs, left arm, and right to less degree, and is accompanied by generalised athetoid-like movements.

Walks a very little, with characteristic gait.

Intelligence and memory seem excellent, but she is a little facile. Hearing and sight good, eye-grounds normal, and speech, though babyish and low-pitched, is not definitely altered. Tongue unaffected.
5 months later.

No change, but sometimes complains of pain about lower dorsal region; nothing found.

Said to be quick at picking up information and is learning to read and write a little, the grasp of right hand being very fair.

No convulsions or headache.

CASE XXXII. Temporary paraplegia.

Girl aet. 8.6.

Very severe attack of epidemic form, in hospital four months and slowly recovered.

When convalescent noticed she could not stand or use her legs, merely collapsing.

She could not sit up for long, and Right lower extremity was completely paralysed. Left almost so, and they always felt cold.

Very intelligent hearing and seeing well.

Seen 6 months after onset.

Right lower extremity wasted and completely paralysed.

No spasticity.

Left " " No evident wasting, but movements very weak, and cannot bear weight of body, although complete range is possible.

Looks blue, with patches of congestion.

Both shew ———— { No sensory disturbance.
Patellar, achilles, and adductor jerks markedly +;
Double Babinski and ankle clonus.

She has incontinence of urine and faeces, now gradually improving; no weakness of upper limbs or trunk muscles, all reflexes being sluggish.

Seen 2 years later.

Continued as above for about 1 year, then began to move legs little more and 2 years from onset could

walk quite well, though apt to fall if hurried, and lately this has passed off. No rectal or bladder symptoms.

Went to school 8 months ago and is rapidly making up ground lost. Memory excellent and no sign of abnormality whatever, though unduly easily tired.

Seen 5 months later.

Healthy child; now in 3rd Standard (average for age 4th), no muscular weakness and can hop equally well on either leg.

Lower tendon jerks slightly +; no clonus or Babinski.

Hobhouse's case was very similar to last.

PERSISTENT CONVULSIONS.

In Case XXV, convulsions continued for 4 years after recovery otherwise, but a slight degree of mental deficiency remained although the fits entirely ceased.

In Case XXXIII, the child had at first paralysis of right arm, and frequent fits, and now, after 3 years the paralysis has almost disappeared, although the fits have continued. Here the left side of body is never affected while in the fit, the movements being unilateral entirely, beginning in the arm.

Triboulet described one case he attended for acute cerebrospinal meningitis, who developed typical epileptic fits some years later. He has seen occasional similar cases before.

I could find no other record of epilepsy ascribed to meningitis.

ILLUSTRATIVE CASE. No. XXXIII.

Boy aet. 6.

Severe attack of epidemic form, fits being marked feature and beginning in first day or two.

As convalescence set in, noticed he could not walk, that his right arm was quite paralysed, and that he had incontinence of urine and faeces. He was having fits daily, and was dull and stupid.

Was discharged in this condition, 5 months after onset.

After 3 months could walk all right, no trouble with urine or faeces, could use Right arm a little, but still having fits daily. The twitching began to Right arm, and frequently rendered him unconscious.

After about 1 year, fits began to diminish in frequency and intensity, no weakness of legs, and Right arm became slowly stronger, but with occasional twitchings independent of the convulsions.

General physical and mental condition improved very much.

Seen 3 years after onset.

Fits, though sometimes severe, only occur once in 2-3 weeks, and he retains consciousness.

He knows when fit is coming on, - will run in off street. Gurgling in throat, then right arm, eye and side of face begin to twitch, and finally the leg, and movements becoming more violent.

Left side never affected; does not pass urine or bite tongue, but is very dull and sleepy afterwards.

Has never been to school, and is backward for age. Memory uncertain, but answers questions very intelligently.

Hears, sees, speaks, and runs about perfectly, inco-ordination nowhere impaired, and can stand well on tiptoes, muscularity all over being good and equal.

Knee jerks slightly +, as are all tendon jerks,

no ankle-clonus or Babinski.

Upper extremities, {	R. 25
Dynamometer {	L. 50

No ocular symptoms; eye-grounds normal.

Seen 3 months later.

Condition unchanged, but power of Right hand slowly improving.

Seen 4 months later.

Fits been more frequent; mother ascribes it to cold weather, and says they are always **worst** under these circumstances.

Seems as bright mentally.

PERSISTENT ATAXIA.

One case is described.

As regards the seat of lesion, some authors suggest that tabes dorsalis might at times be due to spinal meningitis, causing sclerosis of the posterior columns. The assumption is based on the following:-

- (1) Frequent abolition of knee-jerks during acute meningitis.
- (2) During after-progress - i.e. period of sequelae, often get:-

{ Diminution in knee-jerks.
 " " pupil reactions.
 { Lymphocytosis of cerebrospinal fluid.

The semicircular canals could hardly be involved apart from the cochlea as well. Millar has recently described cases of "acute Tremor" in children, the lesion probably being in the cerebello-rubro-spinal tracts; to these, however, this case hardly corresponds.

A cerebellar encephalitis is possible, but it will be interesting to note how much this case improves, as at first, for 4 months, the child was unable to walk at all. At present, however, there is no indication of muscular weakness.

CASE XXXIV.

Boy aet. 8.

Healthy till 7 months ago, when took severe

attack of sporadic form. Unconscious for week, internal strabismus and lost sight and speech completely for a month. Unable to walk at all for 4 months, since then gradually improving.

Is well developed and nourished, muscularity equal and good; no sign of hydrocephalus.

Intelligence, memory, attention, all excellent.

Gait is ataxic, with reeling element; has coarse tremor of outstretched hands and arms with incoordination of voluntary movements.

Rhombergism well marked, tending to fall back.

Knee-jerks + +.

Other reflexes unchanged.

No sensory disturbance, hears well, no vertigo.

Speech high-pitched, and distinctly staccato.

Sight good; well marked double internal strabismus; no nystagmus or other ocular symptoms; pupils react well to light and accommodation.

Marked mitral stenosis present.

Left, r View by Transmitted Light.



Colour Photograph.

Pneumococcal
Leptomeningitis.

from middle ear disease,
(chronic).

I shall now deal briefly with the commoner varieties of Suppurative Leptomeningitis - where organisms are found in cerebrospinal fluid. The forms with sterile cerebrospinal fluid have been already described (page 16).

PNEUMOCOCCAL.

This may be brought about in two ways:-

- (1) A blood spread, e.g. from a Pneumonia.
- (2) A spread from local infection, e.g. from the middle ear, or external trauma. In this sub-division, of course, other organisms - staphylococci, etc - may be present instead, and therefore all will be considered together.

(1) BLOOD SPREAD.

This form is almost always fatal. I found records of 19 cases in all, with no recovery. Auché in 1904 reported what seems to be the only authentic cure.

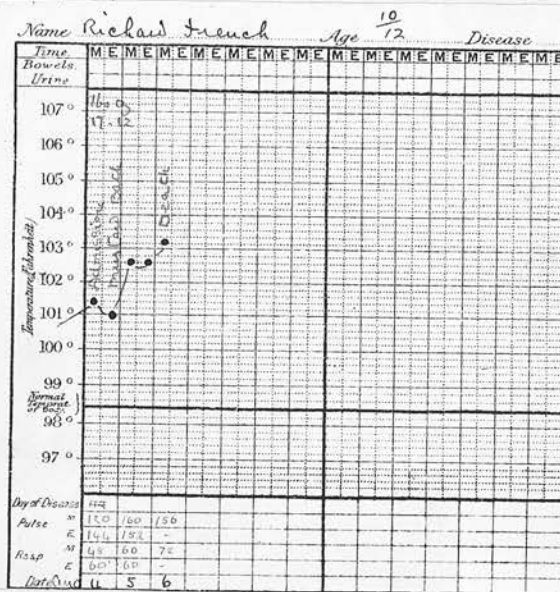
Boy aet. 5, with lobar pneumonia at left base. This rapidly cleared up, but characteristic symptoms of an acute meningeal affection asserted themselves, and the cerebrospinal fluid was turbid, under pressure, and contained typical pneumococci in numbers.

After 5 weeks illness, with repeated lumbar puncture, the child began to recover, and eventually

was completely cured.

The following are notes of a characteristic fatal case:-

CASE XXXV.



Boy aet. 10.

Presented typical signs and symptoms of broncho-pneumonia. Fontanelle not tense, nor was abdomen retracted.

After 5 days illness, general rigidity was first noticed, with head-retraction and rolling movements of eyes. No squint or fits, but fontanelle became very tense.

Next day rigidity intense, eyes fixed and staring upwards, and little irregular twitching of face and limbs.

Pupils gradually dilated (no optic neuritis), Cheym Stokes breathing set in, and died 2 days after first meningeal symptoms.

P.M.

Diffuse purulent pneumococcal Leptomeningitis of vertex and base of brain, besides consolidation of lung present.

(2) SPREAD FROM LOCAL INFECTION.

I collected notes of 12 cases with this condition present - all fatal. The causative organisms are most commonly Pneumococci, Staphylo, or Streptococci, and the Meningitis, though at first localised, tends to spread rapidly.

The initial lesion may be due to trauma, or disease elsewhere, but in a vast majority of cases is chronic middle ear disease - possibly acute otitis media in babies before closure of the petrosquamosal suture.

It may be caused by direct organismal spread, or through the medium of a septic thrombosis, and the prognosis has hitherto been practically as hopeless as in (1).

More recently, however, several cases of cure have been reported after early and thorough operation, notably by McEwan, and Barker in this country.

The first essential is to verify the diagnosis by lumbar puncture, and if positive, to at once clear out the initial focus of infection by operative means.

Gauthier advises the following procedure in

addition.

- (A) If mild case, repeated lumbar puncture, with injection of some fluid, as collargol, to combat the infective fluid.

Crowe first pointed out, and proved experimentally to be useful in this connection, that Urotropin given by the mouth is excreted in quantity by the cerebrospinal fluid.

- (B) In more severe cases, to at once open the membranes of brain and drain the subarachnoid space.

Attempts have been made to wash out the entire tract by means of 2 openings - 1 cranial, 1 spinal - but hitherto with no success.

The use of antisera and vaccines does not yet seem to have had a fair trial, but no case should be despaired of until operative means have been tried, as indicated.

WITH TYPHOID FEVER.

Although meningism is common at onset of this disease, true purulent leptomeningitis with isolation of *B. Typhosus* is rare.

Osler in 1,500 cases of typhoid fever, had only 1 case, and 5 of the serous form.

Of 2,000 Munich cases, only 11 shewed meningitis, but proportions of purulent and serous are not noted (Osler).

In babies dying of gastro-enteritis with meningeal symptoms, occasionally the paratyphoid bacillus can be isolated from the purulent cerebrospinal fluid.

In both, the disease seems to have been universally fatal.

WITH INFLUENZA.

Purulent Leptomeningitis with Pfeiffer's bacillus as the causative organism is not a common complication.

Slavik described the first authentic case in 1899, of a fatal case in a child aet. 9 months. Since then, cases have been reported at intervals.

Cohoe recently collected 26 certain cases from the literature; these were confined entirely to children, and 56% under age of 1 year.

The mortality was 85%; 3 recovered, but one was left with a complete and permanent hemiplegia.

WITH CONGENITAL SYPHILIS.

This disease is described as occasionally causing hydrocephalus, and presumably may do so in two ways:-

- (a) By a basal meningitis, obliterating cerebro-spinal foramina.
- (b) By a chronic inflammatory process of ependyma, and of epithelium covering choroid plexuses.

Fournier collected 170 cases of hydrocephalus of this origin.

With antisppecific treatment, 6 were apparently cured, and 5 slightly improved, the remainder (93.6%) dying.

Gee and Barlow, when first describing post-basie meningitis in 1878 noted 2 cases, aet. 3 and 8 respectively, who had both had typical congenital syphilis, but without having ever had any meningeal symptoms.

They died of marasmus, and P.M. old patchy meningitis was found.

BIBLIOGRAPHY.

- AUCHE. ... Revue Mensuelle d. Maladies d'enfance
January 1904.
- ARMAND DELILLE. Thèse de Paris, 1903.
- BARLOW & LEES. Clifford Allbutt's System. Vol. VII.
- BILLINGS. ... Journ. of Amer. Med. Assoc. June 2nd, 1906.
- BALLANCE ... Surgery of Brain & Spinal Cord 1909.
- BEACH ... Brain 1889.
- COURTELLEMONT. Accidents Nerveux consecutifs aux
Méningites aiguës simples, 1904.
- COHN. ... Berl. klin. Woch. January 11th, 1909.
- COUNCILMAN, MALLORY & WRIGHT. Amer. Journ. of Med. Sc.
1898.
- CROWE. ... Johns Hopkins Hosp. Bull. April 1909.
- COHOE. ... Amer. Journ. of Med. Sc. 1909.
- DEBRE. ... La Presse Medicale. Mai, 1909.
- DURET. ... Proc. Internat. Cong. on Tuberculosis,
Paris, 1905.
- DOPTER. ... Bull. de la Soc. Méd des Hôp. XXVIII
p. 39.
- FERRIER. ... Functions of the Brain, 2nd Ed.
- FLEXNER. ... Journ. of Exper. Med. Jan. & Sept. 1908.
- FOWLER. ... Review of Neur. and Psychiatry,
April 1907, January 1909,
- GEE & BARLOW Reports of St. Barts. Hosp. 1878.
- GAUTHIER ... Annales des Mal. de l'oreille, etc.
Mai, 1909.
- HARTMAN ... Deafmutism.
- HOBHOUSE ... Lancet November, 1897.
- HORSLEY ... Transact. of Roy. Soc. of Med. 1907-8

- Archives*
- HOLT ... Amer. ~~Journ.~~ ^{Archives} of Pediatrics, Vol. XX 1908.
- HILL. ... Cerebral circulation: Allbutt's System, Vol. VII.
- HALLIBURTON. Journ. of Physiology, Vol. X.
- HILDESHEIM Practitioner. Aug. and Sept. 1905.
- HILTON ... Rest and Pain.
- JOSLIN ... Amer. Journ. of Med. Science, Oct. 1900.
- KER ... Practitioner, Jan. 1908.
- KOPLIK ... Trans. of Amer. Ped. Soc. Vol. XX 1908.
Amer. Journ. of Med. Sc. April 1907.
" " " " " 1905.
- KNOX & SLADEN Amer. Journ. of Ped. Vol. XX 1908.
- LEWIS ... Textbook of Mental Disease.
- LOVE. ... Diseases of Ear, also Deafmutism.
- LOOFT ... La Mening. Cer. Spin. Ep. en Norvège 1875
1897
- LANGMEAD ... Practitioner 1907.
- MILLAR ... Brain CXXV. 1909.
- MANDOUL ... Arch. Gén. de Méd. January 1906.
- MACDONALD ... Thesis for M.D. Edin. 1907.
- MARTIN ... Brain. CXXVI 1909.
- NETTER ... Supp. to 20th Cent. Pract. of Med. 1903.
Bull. d'Acad. de Med. May 4th, 1909.
" " " " I XI. p. 300.
- OSLER ... Principles & Pract. of Med. 1909.
- ROMER ... Muench. Med. Woch. No. 49 1909.
- ROBB ... B.M.J. Feb. 1908.
- ROSEWARNE ... Lancet. Oct. 30th, 1909.
- RIEBOLT ... Deut. Med. Woch. November 1906.
- ROLLESTON ... British Journ. of Childr. Dis. Oct. 1909.

- SNELLEN ... B.M.J. November, 1907.
- STILES ... Proc.Internat.Cong.of Tuberc.
Washington, 1908.
- THIEMICH ... The Diseases of Child. Pfaundler &
Schlossmann.
- TAYLOR ... Amer. Journ. of Med.Sc. 1906.
- TREVES. ... Brain. XXXII 1909.
- TRIBOULET ET LIPPMAN. Soc.Med. des Hôp.17th Jan.1902.
- TREDGOLD ... Amentia.
- VAILLARD Bull. d'acad.de Med. April 27th ,1909.
- WATSON CHEYNE & SUTHERLAND. B.M.J. Oct.1898.
- WIDAL ... Revue Mens.de Med.et de Thérap.I.1909.
- ZIEMSSSEN ... Cyclopaed. of Med. Vol II (Amer.Trans)
-