

the remaining 33, evidence was either vague or absent.<sup>6</sup> Only 13 showed a severe infection. For this reason, clinical estimates of its proportion are apt to be fallacious unless examination is made in the early weeks, on repeated occasions, and knowledge is included of the parents or their previous children. On the hypothesis that syphilis is causal, however, these clinical deductions should prove correct:

1. In those severely tabetic, there should be evidence of syphilis, especially of cranial syphilis, co-existing or sequent. Of the present series, 38 had extensive softening, both sides being affected, with confluence of areas. Of these 6 showed severe syphilis, 13 mild forms, and in 19 syphilis was absent on all counts.

2. Where cranial syphilis co-exists, the greatest amount of craniotabes should coincide with the greatest activity of the syphilitic process in that instance.

3. On subsidence of the inflammatory condition of the bones, the craniotabes should be found to diminish *pari passu*. This should occur at least to a partial extent.

Craniotabes, however, in cranial syphilis I find to follow the pressure conditions, without relation with the syphilis. Among my records are cases where all signs of cranial and other syphilis were reduced under treatment; yet during or after their removal, craniotabes appeared. In a number of others, an existing postnatal area behaved during a course of mercury as though totally uninfluenced.

A boy, aged 9 weeks, with rather severe syphilis also involving the cranium, showed an area of second degree (postnatal). Vigorous treatment by inunctions was then carried out for four weeks, before the end of which all evidence of syphilis had disappeared, together with considerable gain in weight. The original area remained as before, and one of larger size had meanwhile developed on the opposite bone.

The curious view has been advanced by Marfan that syphilis can only be causal when co-existing with rickets. But this clearly breaks down unless syphilis can be proved present in nearly every case. It happens that a clinical diagnosis is far more weighty in infancy than in later years, as nearly all congenital cases give evidence within six months of birth. I can assert the absence of syphilis, therefore, without misgiving in certain whole families furnishing craniotabetics; and in a few it has been possible to maintain a scrutiny much closer than ordinary. The following is an instance:—

The first (male) child of young parents had an entirely uneventful infancy, and is now 3 years old. The next (female) had mild rickets, and is now 4 years. The third (male) had fetal and extensive postnatal craniotabes, and is now, at 12 months, definitely rachitic. I have been in very close professional touch with this family for over eight years, during which time no trace of syphilis has appeared. The father, aged 29, shows no physical signs, and his venereal history is negative.

Such evidence as the foregoing, taken together, seems to rule out the causality of syphilis. The more rational view is, I think, as follows:—

Granting that the incidence of syphilis is much too high to be dismissed as adventitious, we may suppose it to favour the true cause in some manner now obscure. Assuming this cause to be rickets, syphilis will therefore favour the existence of rickets. Hochsinger has concluded that syphilis can hasten its onset; and this, if correct, would help to explain why pressure is likely to produce craniotabes in a syphilitic subject. But one is driven to grant that rickets not seldom exists during the period when the foetal head comes to assume a more or less constant position in the lower uterine segment.

The following case may be cited as relevant to the present argument.

The first child (female) of apparently healthy young parents had foetal, and at the second month postnatal, craniotabes, which underwent partial development. No syphilis, but well-marked rickets appeared in second year. During the first pregnancy the father joined the Army, and on a return on leave the mother conceived again. The next child (male), born 13½ months after the first, had foetal followed by large postnatal areas, and at four weeks an extensive macular syphilide, rhinitis, slight hydrocephalus, &c. He responded well to treatment, though the craniotabes continued to spread, as in others. The foetal area in the first child was moderate in size, involving the left parietal; in the second larger, involving both bones; and his postnatal areas were also larger.

This is offered as a possible illustration of syphilis in the rôle of accomplice. That the first child is a latent syphilitic is conceivable, but is not an impartial reading of the data.

In imagining a method by which syphilis might favour the cause, the added effect of plus intracranial tension seemed to merit inquiry. Gauging its degree by the fontanelles and amount of venous turgidity, I recorded 41 cases of cranial syphilis during the first four months, finding craniotabes in 9. In 16 the tension was relatively high, and 5 of these had craniotabes. Probably the effect of tension is negligible, as in many it cannot be shown to be increased.

While it is clear that mercury is without influence on craniotabes, the therapeutic test for rickets cannot easily be made on young infants, both through the dietetic limitations and the lack of a "specific" for this disease. Experiments, however, both dietetic and medicinal, had been begun on selected cases, which have had to be temporarily relinquished.

<sup>6</sup> Of 100 cases collected by Lees and Barlow (Trans. Path. Society, 1881) 47 were certainties, 40 more were probables.

## THE NERVOUS SEQUELÆ OF CEREBRO-SPINAL FEVER.

BY CECIL WORSTER-DROUGHT, M.B. CANTAB., &C.,  
TEMPORARY CAPTAIN, R.A.M.C.; NEUROLOGIST AND OFFICER IN  
CHARGE, CEREBRO-SPINAL FEVER WARD, ROYAL HERBERT  
HOSPITAL.

PRIOR to the introduction of repeated lumbar puncture and the intrathecal administration of specific anti-serum as routine measures in the treatment of cerebro-spinal fever many of the relatively few cases that recovered appear to have suffered from some permanent disability, either mental or physical.

The older writings on the subject abound with references to the frequency of infirmities met with in survivors from the disease. Some allowance, however, must be made for the fact that, owing to the absence of accurate diagnosis by an examination of the cerebro-spinal fluid obtained on lumbar puncture, polio-encephalitis and the so-called meningeal form of acute anterior poliomyelitis were often confused with meningitis; consequently many of the paralytic sequelæ of the former diseases were erroneously attributed to meningitis. Nevertheless, serious disabilities frequently followed cerebro-spinal fever, by reason, no doubt, of the compression to which the brain structures were subjected during the course of the disease by the increased tension of cerebro-spinal fluid unrelieved by lumbar puncture.

Contrary to popular supposition, it is an undoubted fact that in recent years serious sequelæ have been considerably less frequent than formerly. These improved results are not only due to the relief of intracranial pressure afforded by repeated lumbar puncture, but also to the shortening in length of the usual course of the disease occasioned by the use of antimeningococcal serum administered intrathecally.

In infants, as formerly, the sequelæ remain the most serious.

Batten, for instance, writing in 1915, states that in the endemic form affecting infants and occurring annually in London, only 15 per cent. of cases recover completely; 35 per cent. survive but exhibit some permanent defect—either mental impairment, hemiplegia, diplegia, or blindness, while the remaining 50 per cent. prove fatal. In infants internal hydrocephalus is a most frequent complication, its development and course being considerably modified by the anatomical condition of the infantile cranium; the intracranial pressure brought about by the accumulation of cerebro-spinal fluid in the ventricles not only leads to compression of the cerebral tissues, but also expends itself in producing bulging of the fontanelles and separation of the cranial sutures.

In the infant, therefore, the nervous structures of the cerebral cortex are subjected to a considerably more gradual pressure than is the case when, as in the adult, the cranial capacity is a fixed quantity. These facts may account for the occurrence in children of permanent disabilities such as diplegia, blindness of central origin, and mental impairment, which at the present time are observed in adults only with extreme rarity.

The following description of the conditions of nervous origin which may result from an attack of cerebro-spinal fever is based chiefly upon a study of a series of 120 recovered cases coming under the observation of the writer.

### Mental Changes.

In the adult mental impairment is very rare. During 1915–17, of 120 patients recovered from cerebro-spinal fever, mainly adolescents and adults, I met with no instance in which permanent mental enfeeblement resulted. Bourke, Abraham, and Rowlands, however, state that of 77 military cases recovered from the disease (1915) feeble-mindedness was present in two.

Among the minor psychological changes Netter mentions conditions of irritability and puerility, while other French authors describe marked loss of attention or memory, moroseness, morbid shyness and morbid egoism. During early convalescence I have observed that some patients may exhibit unevenness of temper, anger being easily evoked, while inability to concentrate the attention for any length of time is relatively frequent. Such changes rarely persist, but disappear after a variable period.

Puerility occurred in one case out of 120.

The patient, aged 23, had survived a severe attack of the disease; duration approximately 40 days. When first beginning to walk about he would attempt to carry out practically anything he was told to perform, no matter how absurd it might appear—e.g., walking on his hands and knees, lying under bed, &c. After a week or two, however, he recovered his normal mental equilibrium.

Three patients of the 120 recovered cases complained of defects of memory.

The first, seen three months after discharge from hospital, stated that he had occasional difficulty in remembering things that were told to him or orders it was his duty to carry out.

A second patient, four weeks after termination of course of illness, complained that he had forgotten the names of several of his intimate friends and also of places he had visited. When seen a year later his memory had greatly improved, but he considered that he could not remember facts as well as prior to his illness.

The third patient was employed in clerical duty at a military dépôt; on returning to work he found that he had great difficulty in adding up columns of figures that previously he had managed with ease; no other psychological symptoms were complained of. Six months later his defect was still present.

Foster and Gaskell mention one recovered case in which there was complete loss of memory. As a concomitant paralysis of the right arm was present, however, the amnesia possibly owed its origin to a definite and localised cortical lesion. Six months later this patient's memory was almost completely restored, although some weakness of the arm persisted.

As regards permanent mental defects following the disease in infants, Looft estimated that 3·7 per cent. of 539 idiots owed their condition to an attack of meningitis. Netter, without doubt rightly, attributes a considerable number of such cases to insufficient serotherapy. Cases in which dementia results are often suffering from chronic hydrocephalus, which in some instances may persist for a considerable period. In all cases with dementia care should be taken to exclude hydrocephalus, even though apparent recovery from meningitis has occurred, before the patient leaves hospital.

#### *Sensory Symptoms.*

**Headache.**—Patients may continue to complain of headache for some time after convalescence is established and Kernig's sign is definitely absent.

In the majority of cases the headache entirely disappears after a few months; in others, however, it persists for longer. Of the 120 recovered cases already mentioned only 8 continued to suffer from headache three months after becoming convalescent; a few stated that the symptom only occurred after exertion, and in one case it was merely nocturnal.

Persistent headache of a severe nature may be due to a certain degree of hydrocephalus, which later may give rise to serious symptoms and even produce a fatal result.

**Pain in the back.**—Pain, weakness, and stiffness in the back are almost invariable complaints during the early stages of convalescence; in consequence, an awkward gait may persist for some weeks.

The majority of patients cease to experience backache about 3–4 months after termination of course of illness. Several, however, may complain of weakness in the back for many months after recovery; in some the symptom is only noticed on stooping, while in others pain is present in damp weather. Hyperæsthesia of the spine may occasionally persist well into convalescence.

That the pain and weakness in the back are due to the effect of the disease itself rather than to that of lumbar puncture is shown by an observation by Foster and Gaskell. A certain case in which lumbar puncture was performed once only, owing to the presence of auricular fibrillation, recovered completely from meningitis after a course of about 40 days; the patient, however, exhibited the symptom of pain in the back to a greater degree than other cases on whom a dozen or more punctures had been performed.

Also, I have known cases recovering from an abortive attack of the disease who had had no more than one or two punctures complain of pain and weakness in the back for a longer period than patients who had passed through a severe illness, necessitating the performance of many lumbar punctures. In all recovered cases I have carried out an examination for possible lesions of the cauda equina resulting from the punctures; of upwards of 120 cases, however, in no single instance could the slightest lesion be demonstrated.

**Pain in the limbs.**—Pain in the legs, persisting for more than three months after recovery, is a comparatively rare sequel. Occasional pain was complained of in two only of the 120 cases, the leading question not being put. Only one patient volunteered the information that he experienced occasional pain in the arms.

#### *Motor Defects.*

**Ocular muscles.**—As lesions of the nerves supplying the muscles of the eyeball usually improve coincidentally with recovery from meningitis, permanent strabismus as a sequel is most uncommon. When present it is usually the result of involvement of the sixth cranial nerve.

Of the 120 cases only 2 exhibited an ocular palsy persisting for longer than three months after recovery from the disease.

The first patient, who during the attack of cerebro-spinal fever had had complete paralysis of both external rectus muscles, exhibited when seen three months after his discharge from hospital, complete paralysis of the left external rectus, while some weakness still remained in that on the right.

The second patient, examined six months after recovery, showed some nystagmus and marked weakness of one external rectus muscle.

Bourke, Abraham, and Rowlands, of 77 cases recovering, met two with permanent strabismus.

As a rule, the pupils remain somewhat dilated for some weeks after establishment of convalescence, but eventually become normal. Persistent myosis or inequality in the size of the two pupils is extremely rare. Permanent nystagmus also is most infrequent.

**Limb palsies.**—Paralysis of limbs, developing during the course of the disease, may persist for several months after recovery from meningitis. Monoplegias due to the involvement of spinal nerve roots almost always disappear eventually, although a considerable time may elapse before function is fully restored. As an example the following case may be mentioned:—

A child aged 6 years, suffering from cerebro-spinal fever, exhibited on the sixth day of illness considerable weakness of both arms. Three days later patient was unable to abduct either arm at the shoulder, the deltoid muscles being paralysed and flabby; paralysis of right biceps and triceps was also present, no degree of active flexion or extension being possible at the elbow. The supinator, biceps, and triceps jerks were unobtainable in both limbs, and hyperæsthesia was particularly marked in the right. By the fourteenth day there was a fair amount of wasting

of affected muscles in right arm. Meningococci were present in cerebro-spinal fluid until ninth day of disease, and recovery from meningitis, as shown by the evacuation of a clear and sterile cerebro-spinal fluid, was not complete until the thirtieth day.

Fourteen days after recovery movement in the left arm had returned, but the right still showed paresis of the deltoid, biceps, and triceps. No response to faradism was obtained in these muscles, and with galvanism A.C.C. was greater than K.C.C. in the deltoid; in the biceps and triceps A.C.C. was about equal to K.C.C.

Full power was recovered in the affected arm about seven months after recovery from meningitis.

If a monoplegia or paralysis of a group of muscles persists without improvement, it is probably dependent upon definite changes in the anterior cornual cells.

Hemiplegia, occurring as a complication of cerebro-spinal fever, usually terminates in almost, if not quite, complete recovery.

For instance: In one case of the series, a boy aged 15 who ran a recrudescence course of over 80 days, hemiplegia developed about the fifty-first day of illness. By the termination of the course of meningitis (eightieth day), and about 30 days after appearance of hemiplegia, the paralysis of the arm had quite disappeared. Seven and a half months later, all that remained of the lower limb palsy was weakness of calf muscles and inability to flex or extend great toe; the other toes exhibited weak movement. Both patellar clonus and ankle clonus, previously present, had disappeared, but on the affected side, the knee-jerk and tendo Achillis jerk remained brisker than those in the opposite limb, and the plantar reflex was still extensor.

Cerebral hæmorrhage, occurring during the course of cerebro-spinal fever in middle-aged patients with pre-existing nephritis, may lead, if the patient recovers, to permanent hemiplegia.

Flaccid paraplegia, fortunately a very rare complication, may show some improvement but is usually permanent.

Some degree of weakness in the legs and unsteadiness of gait is frequent during the early stages of convalescence.

In a few instances, according to Sophian, exaggerated knee-jerks associated with a true ankle clonus and extensor plantar reflexes, may be elicited. I have frequently observed ataxia with a positive Romberg's sign and difficulty in walking; in many cases, exaggeration of the deep reflexes is also present. In no instance, however, in the absence of a definite upper neurone lesion has one found ataxia associated with either definite ankle clonus or a true extensor plantar reflex. Romberg's sign was not observed in any case in which a period of one month had elapsed from the time of the patient's beginning to walk.

One man, aged 30, was met with, who had had an attack of cerebro-spinal fever at Belfast eight years previously; for a considerable period following his recovery he had complained of weakness affecting the legs. When examined he stated that he still felt weak and "shaky" in the legs after violent exertion; he exhibited, however, no abnormal physical signs.

**Peripheral neuritis.**—This condition is a very rare complication of cerebro-spinal fever.

Of two cases seen in which definite neuritis affected the lower extremities, complete reaction to degeneration in the muscles supplied by the affected nerves developed in neither case. In one case the tendon reflexes were beginning to return, and sensation was nearly normal within three months of recovery from meningitis; the second patient, after a similar period, exhibited normal cutaneous sensation, but both the knee and ankle-jerks were still absent, and the vibration sense remained much diminished.

**Muscular rigidity.**—Rigidity of the cervical muscles persisting for any considerable period after recovery is extremely rare.

As a rule, neck rigidity is absent before or within a week or so of the cerebro-spinal fluid returning to normal. In the following case, however, actual head retraction persisted long after the course of meningitis, and even when the patient was up and about.

A youth, aged 19, was admitted to hospital acutely ill on the second day of a recrudescence attack of cerebro-spinal fever; four days earlier he had had an abortive attack elsewhere. On admission considerable head retraction was present. This symptom was still as marked on the twenty-fourth day of illness, by which time the cerebro-spinal fluid obtained on lumbar puncture was quite clear, sterile, and contained no more than the normal number of cells. At times occipital retraction had been so extreme as to cause difficulty in swallowing.

One month after the termination of the course, the patient having been up and about for over a week, there was still a tendency to head retraction, with inability to bring the head quite into the vertical position. Massage, &c., was then instituted and four weeks later there was some improvement; he remained, however, unable to flex the head beyond the perpendicular while standing in the erect position. Finally, it was not until three and a half further months had elapsed (five and a half months from termination of course of meningitis) that the patient was able to flex the head sufficiently for the chin to reach the chest.

Kernig's sign, in the majority of cases, persists for the first 5–14 days of convalescence.

In a few cases the sign may still be present for 20 or even 30 days following the evacuation of normal cerebro-spinal fluid and in the absence of all symptoms of hydrocephalus. Some rigidity of the hamstring muscles can occasionally be demonstrated 2–3 months after recovery.

#### *Other Nervous Conditions.*

**Aphasia.**—Persistent aphasia has been noted in rare instances. The only case of our series in which this complication occurred had fully recovered the faculty of speech before the termination of the course of the disease.

**Epilepsy.**—Sainton mentions epilepsy as a rare but occasional sequel of recovery from cerebro-spinal fever.

This observer mentions one case in which the crisis began with an aura of gastric origin—nausea and vomiting, followed by loss of consciousness and epileptic convulsions. The cerebro-spinal fluid between the crises was normal, but the fluid obtained immediately after the seizure showed a slight lymphocytosis. The general condition of the patient was good. Voisan reports a somewhat similar case.

Petit mal is also stated occasionally to occur.

**Neurasthenia.**—Considering the severity of the usual course of the disease process in cerebro-spinal fever, I have found the occurrence of true neurasthenia following recovery from the disease surprisingly uncommon.

It is contended by Dejerine and others that to constitute true neurasthenia, in addition to a state of neural fatigue, there must exist a condition of continued emotivity; the non-adaptation of a continuous emotive cause, to which is often added an obsessing preoccupation, produces a condition of neural exhaustion, and the sum-total of the phenomena resulting justifies a diagnosis of true neurasthenia.

The appearance of neurasthenia following recovery from cerebro-spinal fever, therefore, would depend to a large extent upon the temperament and inherent or acquired tendency to emotivity of the individual, as well as upon the severity of the disease. Chronic cases, it may be remarked, are more liable to become neurasthenic than those exhibiting a shorter course. Six patients only of the 120 cases recovered could definitely be said to show signs of true neurasthenia when examined 4–6 months after recovery from meningitis; of these six patients, two at least were neurasthenic prior to the attack of cerebro-spinal fever. Of the remainder the following case serves as an example.

The patient was examined six months after recovery from a severe attack of cerebro-spinal fever lasting approximately five weeks. He complained of frequent headache, soreness in the limbs, and of fatigue upon the slightest exertion; his appetite was poor and he slept badly.

**Physical signs:** The patient was thin and perspired freely during the examination although it was winter. The pupils were somewhat dilated, equal, and reacted sluggishly to light and accommodation. There was a well-marked tremor of the outstretched hands; the superficial reflexes were brisk and the knee- and ankle-jerks exaggerated. No ankle clonus was elicited and both plantar reflexes were flexor.

Two other patients exhibited a somewhat similar condition; one, in addition, complained of occasional loss of memory. Another recovered case, seen four months after his discharge from hospital, complained of sleeplessness but showed no other evidence of neurasthenia.

A slight tremor of the outstretched hands is frequently observed during the first few months following recovery from the disease.

One patient examined five months after recovery from meningitis closely simulated a case of disseminated sclerosis.

The man, aged 26, complained of occasional inability to use the left hand and also of frequent diplopia. On examination there was distinct rotatory nystagmus on lateral deviation of the eyes, together with weakness of the left external rectus muscle. The pupils were somewhat dilated but reacted normally to light and accommodation; the optic discs showed nothing abnormal. There was a coarse tremor of the outstretched hands, more marked on intention, together with some incoordination of the left arm. The left hand-grip was considerably weaker than that on the right; the left supinator, biceps and triceps jerks were brisker and of greater range than those on the opposite side. Both the knee and tendo Achillis jerks were brisk and exaggerated but equal on the two sides; ankle clonus, however, was absent and both plantar reflexes were definitely flexor. The abdominal and epigastric reflexes were also well marked and speech was normal.

The presence of the abdominal reflexes and a bilateral flexor plantar response alone serve to distinguish the above condition from disseminated sclerosis.

Poster and Gaskell mention a recovered case exhibiting exaggerated knee-jerks, volitional tremors, weakness of the bladder, and pallor of the optic discs.

#### Special Senses.

**The eye.**—Blindness may result from (1) damage to the structures of the eyeball; (2) permanent changes in the nervous tissues associated with vision.

1. **Damage to structures of eyeball.**—Corneal ulceration may leave permanent opacities, and loss of vision is the usual sequel to iridocyclitis. This latter complication had occurred in one case of the 120; when examined three months after recovery from meningitis he was able only to distinguish light from darkness with the affected eye. Panophthalmitis is almost invariably followed by complete blindness in the affected eye.

A large vitreous hæmorrhage, associated with optic neuritis, occurred in one of our cases during the course of illness. When seen three months after recovery, the vitreous was practically clear and vision in the affected eye was 6/9.

2. **Permanent changes in the nervous structures.**—In adults, blindness of central origin is very rare; when present, it is usually due to optic atrophy and is permanent and incurable.

In infants suffering from the posterior basic type of the disease, blindness, according to Langmead, develops in about 30 per cent. of cases. Although at first the optic discs may exhibit no definite change, optic atrophy usually develops at a later stage. In most instances the blindness is due to changes in the visual centres situated in the cerebral cortex or the occipital region.

No instance of blindness of nervous origin occurred in the series of cases under review.

**The ear.**—Deafness is the most common of the serious sequelæ met with at the present day.

The disability is almost always bilateral and if persisting during convalescence, is usually permanent; some recovery, however, has been known to occur in a few instances after an interval of two or three months.

Below the age of 7 or 8 years permanent deafness usually leads to an associated mutism; even though speech has begun it gradually dis-

appears. Statistics of the proportion of acquired deaf-mutes who owe their defect to cerebro-spinal meningitis are widely divergent, the number as estimated by different observers varying from 8 to 45 per cent. The condition appears less common in the posterior basic type of the disease than in the more ordinary types. There is little doubt, however, that permanent deafness resulting from cerebro-spinal fever is much less frequent now than formerly. The condition is probably brought about by atrophy of the auditory nerve and possibly some destruction of the cochlea.

Permanent and absolute deafness was present in 2 cases only of the 120 recovered from cerebro-spinal fever. Partial deafness (20 per cent.) of central origin resulted in one other case.

#### Vaso-motor and Secretory Disturbances.

In the majority of cases a well-marked *tache cérébrale* can usually be elicited for many weeks, or even months, following the disease.

Profuse and easily elicited perspiration, apart from a condition of neurasthenia, was present in some cases of the series up till two or three months after recovery from meningitis.

#### The Reflexes in Recovered Cases.

The following observations are the result of a study of 120 cases recovered from the disease and seen at various periods from two months to one year after recovery. One case of hemiplegia is excluded.

**Knee-jerks.**—In five cases only were the knee-jerks found absent, in spite of the employment of the usual methods of reinforcement.

Of these five cases, in two patients their absence was dependent upon the presence of definite peripheral neuritis, the tendo Achillis jerks also being absent, together with loss of both epicritic and protopathic sensibility over certain areas of the lower limbs. In two other cases the tendo Achillis jerks also were not elicited, although there was no definite evidence of neuritis. The last case exhibited absent patellar reflexes, but the tendo Achillis jerks were brisk.

In 10 cases the response to tapping the ligamentum patellæ was very slight.

In 30 cases the knee-jerks were distinctly brisk and exaggerated, but in no instance was this condition associated with either true ankle clonus or a definite extensor plantar reflex.

In the remaining 75 cases the knee-jerks could be described as moderate.

Marked inequality in the reflexes of the two sides was noted in three instances only, and was unassociated with any other physical signs.

**Tendo Achillis jerks.**—This tendon reflex is usually proportional to that obtained at the knee.

In one case only, mentioned above, were the tendo Achillis jerks elicited in the absence of the patellar reflexes. In two instances the tendo Achillis jerks were brisk, but the knee-jerks excessively slight.

Inequality of the ankle-jerks on the respective sides was observed in two cases, one of which also exhibited a corresponding inequality in the knee-jerks.

In no instance was true ankle clonus obtained. Three cases showed what might be described as unilateral pseudo-ankle clonus, unassociated with any signs of an upper neurone lesion.

**Plantar reflexes.**—In all cases plantar stimulation yielded a flexor response.

**Abdominal reflexes.**—It would appear that cerebro-spinal fever may occasionally lead to permanent loss of the abdominal reflexes.

Failure to elicit these reflexes occurred in three cases; of these, no patient exhibited any other abnormal physical signs and in each they had been obtained in the earlier stages of the attack of cerebro-spinal fever.

In 14 cases the abdominal reflexes were only very slight, while in eight they were more than usually brisk. In all the remaining cases these superficial reflexes were moderate. Inequality was noticed in two cases only.

#### Summary.

A summary of the more serious sequelæ occurring in the series of 120 cases is as follows:—

Strabismus ... ..	2 cases.	Hemiplegia ... ..	1† case.
Blindness (peripheral)	4 cases.	Neurasthenia (true) ...	2 cases.
(one eye) ... ..	1 case.	Deafness (absolute) ...	4 „
Monoplegia ... ..	1* „	„ (partial) ... ..	1 case.

\* Function fully restored about 7 months after recovery from meningitis.

† Recovery nearly complete as regards motor power 7½ months after onset.

From this table it will be seen that including neurasthenia 10 patients only of the 120 (8·3 per cent.) suffered from more or less permanent sequelæ.

Of the military cases recovering from the disease—94 in number—as far as I am aware it was necessary sooner or later to discharge as medically unfit for further Army service in any category whatever 11 cases only (11·7 per cent.).

Of these, one patient was neurasthenic prior to developing cerebro-spinal fever, and in addition had double otitis media of old standing; in the ordinary course of events he would have been discharged quite apart from the attack of cerebro-spinal fever. In four further cases discharge was also recommended on account of neurasthenia.

The presence of permanent deafness accounts for two others.

One patient each was invalided on account of one of the following disabilities: persistent strabismus, blindness (peripheral) of one eye, residual weakness of the foot remaining after hemiplegia, and lastly, in a patient aged 41, persistent pain in the lumbar region on exertion.