POLIOMYELITIS AND POLIOENCEPHALITIS.

Thesis submitted for the degree of
Doctor of Medicine by

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This thesis is respectfully submitted for the degree of Doctor of Medicine, and is compiled from the case sheets and records of over six hundred cases of poliomyelitis which have been admitted to hospital.

When it became obvious that a relatively large number of cases of poliomyelitis would be occurring, I asked for the co-operation of the local Medical Officers of Health of the region from which we admitted, in notifying me of the cases which occurred in their districts. I then compiled questionnaires (see appendix) copies of which have been distributed since the poliomyelitis epidemic of 1947.

These questionnaires were distributed as follows:-

1. To the general practitioner treating the patient.
2. To the Medical Officer of the Isolation Hospital to which the patient was first admitted.
3. To the Medical Officer in charge of the admitting hospital or Rehabilitation Centre.

I personally checked the information given on these questionnaires.

When cases were notified to me but not admitted to hospital, a simplified questionnaire was sent to the patient or to his relatives and later checked. By so doing, it was hoped that some particular point would be observed. These questionnaires were also compared with the more detailed ones. By this method it was hoped that a carefully selected and observed series of over six hundred cases would yield a definite clinical picture of these poliomyelitis patients.

A reassessment of my case notes has brought out additional points and combined with my more recent cases, I tentatively submit the following observations.

Poliomyelitis and polioencephalitis are endemic throughout the world, but periodically they become epidemic and unlike the other infectious diseases they appear to be increasing in frequency. The first major epidemic of this acute infective disease occurred in Great Britain in 1947, and it was followed by a second in 1949. From previous experiences in other countries which have been ravaged by this disease, it is certain that these two epidemics will be followed by others.

The popular term "infantile paralysis" should, in my opinion, be strictly avoided for the following reasons:

1. The disease is by no means confined toinfants: the tendency during recent epidemics has been for a much larger proportion of adults and adolescents to be the victims.
2. This infection is only one of the numerous causes of paralysis found in infants.
3. Only a small proportion of the affected patients develop paralysis.

Indeed, judging from experience in the years 1947 and 1949, of every 100 suspected patients, 5 are likely to die; 9 to be severely paralysed; 18 to have some degree of residual paralysis; 38 to suffer either no ill-effects, or to be left with a slight degree of paralysis, whilst in the remaining 30, the initial diagnosis of poliomyelitis is disproved.

Furthermore, the term "acute anterior poliomyelitis" appears to be out-dated by the recent pathological findings both in experimental animals and in humans. It has been shown conclusively that the lesions due to the virus are not confined solely to the anterior horn cells of the spinal cord. Any part of the central nervous system, including the sympathetic and parasympathetic nervous systems may be involved, and so give rise to the different types of the disease mentioned later in the thesis.

It is therefore my intention to use throughout this work the term poliomyelitis to denote acute inflammation of the grey matter of the spinal cord, and polioencephalitis to denote acute inflammation of the grey matter of the brain.

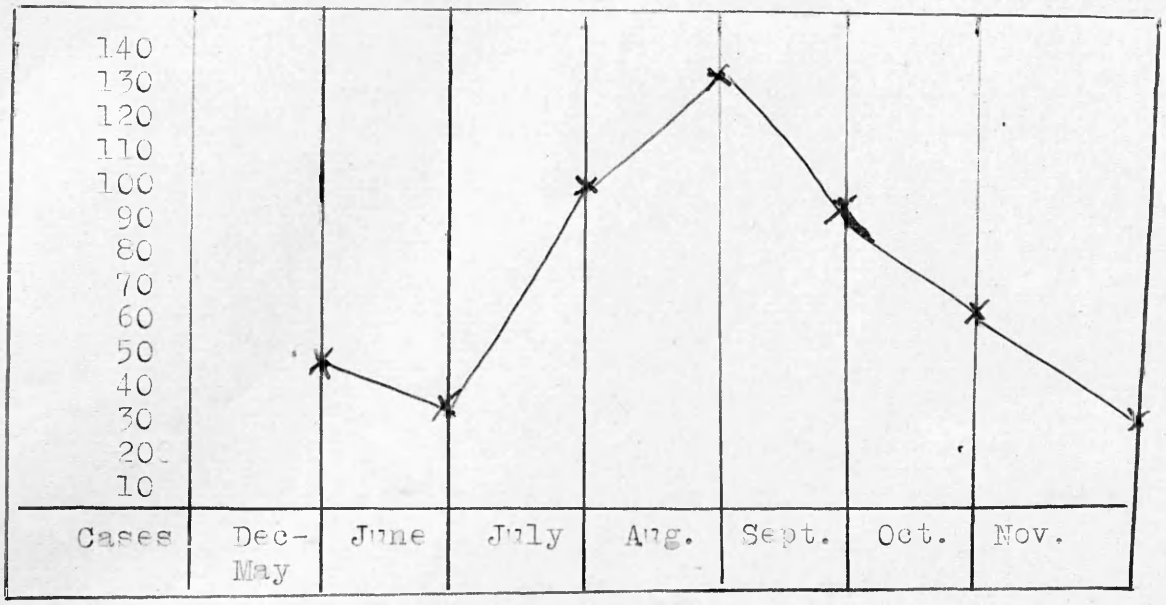
The marked diversity of the symptoms from country to country, from epidemic to epidemic, and from individual to individual, greatly complicates the picture. Signs and symptoms predominant in one epidemic may be entirely lacking in a subsequent outbreak in the same area. This may be due to the type and virulence of the virus, the dose of the infection, the portal of entry, the natural or acquired resistance of the patient, or to other factors as yet undiscovered.

Epidemiology.

First of all it must be stated that unlike other infectious diseases, poliomyelitis is increasing both in frequency and severity. Since the end of the nineteenth century, there has been a steady increase in the number and extent of epidemics throughout the whole world.

During the second world war, observers found that poliomyelitis was a common infection in the tropics, and an important point noted was that the white troops were much more severely affected than the native troops. McAlpine(1945) also made the observation that British officers were affected five times more frequently than other ranks. It has been suggested that whereas officers, even in the field, frequently ate their meals in a mess where crockery was communal, other ranks ate from their individual mess tins.

In this country, the highest incidence of poliomyelitis has occurred during July, August and September. The graph following shows the month of onset of the disease in my series of cases.



Most investigations tend to show that although poliomyelitis occurrs in an epidemic form at all seasons of the year, the number of paralytic cases is much higher during the summer months. It is interesting to note that the seasonal epidemic graphs of poliomyelitis, gastro-enteritis and typhoid fever are very similar.

In the early stages of the outbreak, direct or indirect contact between many of the cases may be demonstrated, but in the later stages this is impossible. The frequency of the infection amongst contacts appears to be proportional to the closeness of their contact with a proved case.

Only a very small percentage of infected cases develop paralysis, and many temporary carriers do not contract the disease owing to their lack of susceptibility.

The following table shows the number of days between the onset of the illness and the first appearance of paralysis in various age groups in my series of cases.

Days	Age Groups.		
	0-5 yrs.	5-15 yrs.	15+
1	25	29	22
2	19	28	24
3	21	28	21
4	20	23	25
5	18	16	17
6	17	12	13
7	14	13	12
8-21	28	22	28
	162	171	162

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When poliomyelitis has followed tonsilleetomy, adenoidectomy or an associated tonsillitis, it is possible that the trauma or bacterial infection may open up peripheral neural pathways for the invasion of the virus.

Anderson et al (1950) stated that the risk of developing poliomyelitis was at least three times as great amongst those undergoing tonsillectomy as amongst a comparable group not undergoing this operation, and the risk of bulbar infection was eleven times as great. This was confirmed by Seigel (1951) who stated that the most desirable months for elective operation seem to be the winter months which are farthest removed from the poliomyelitis season. I agree with these observations.

It seems to me that epidemics occur in the countries with the highest standards of living. It may be that in primitive civilizations infants are repeatedly exposed to infection and thus acquire a lasting immunity, but if the sanitation is improved, then epidemics will occur. The increase in the number and size of the epidemics, the change in the incidence to the older age groups and the greater mortality in adults may be due to better modern sanitation.

I also noted that the great rarity of infection amongst those nursing cases of poliomyelitis, despite the presence of the virus in the faeces, was in direct contrast to that which

occurs in cases of typhoid and dysentery.

Dornedden (1933) expressed the opinion that preliminary catarrh or injuries were predominant factors in paving the way for the virus.

It was found that six of our cases in the age group 0-5 years gave a definite history of injury one to seven days before the onset of the paralysis. Of the 5-15 age group, one gave a history of appendicitis with drainage twelve days previously and here the paralysis affected both legs, the abdominal muscles and the erector spinae. Four cases over 15 years of age admitted to strenuous exercise twenty-four hours before the onset of paralysis of both legs.

It is seen that the seasonal incidence of the disease in Great Britain seems to coincide with the appearance of soft fruits in the shops, and the disease may therefore be spread by the ingestion of unwashed and diseased fruits and vegetables affected by the neurotropic virus. The fruit may be affected by the virus from a sprayed droplet infection, contaminated hands, or directly from faecal-feeding flies or from dust.

Family outbreaks of poliomyelitis are fairly common and although often only one child becomes paralysed, the others may have minor illnesses compatible with abortive poliomyelitis and the virus can usually be isolated from them. In our series of 600 cases, 25 occurred where a definite relationship could be established of contact with another case of poliomyelitis in the same family. The first case usually showed definite clinical signs with paralysis, whilst the other relative or relatives showed only the initial clinical signs with minimal or no paralysis. The majority occurred within the first week, this agreeing with the observations of other workers.

It is now generally accepted that the majority of children are relatively immune during the first year of life, even when in close contact with a definite case, and this has been attributed to a passive immunity derived from the mother. If a baby does develop the disease, it was usually of a very mild nature. In our series of cases, 16 were under one year of age, nine being females and seven males. One or both legs were usually affected, but in three cases the left arm was involved. One of these also had a paralysis of the seventh cranial nerve.

When poliomyelitis is a sporadic endemic disease and during the first epidemics experienced by a country, approximately 90% of the paralytic cases occur in children under five years of age. Craster (1916) reported that 86% of 1360 cases were under 5 years of age and Blencke(1933) found that 70.3% of 1695 cases were also in this group. In later epidemics the proportion of cases occurring in the later age groups increases with each epidemic, about 50% occurring in the 0-5 age group and about 80% occurring in the 0-10 group. For example in 1907 in Massachusetts, only 7% of cases were over 15 years of age, but in 1945, this had increased to 25%. In the 1947 epidemic in the British Isles, approximately one third of the cases were in each of the age groups 0-5, 5-15 and over 15 years. Horton and Rubinstein (1948) attributed this change in the age distribution to an alteration in the age of the population and to an increase in the number of non-paralytic cases reported.

It is nevertheless now generally agreed that the age incidence has altered greatly during the last few decades and that except during epidemics, cases are rare in the adult population. The following table shows the age incidence in my series of 600 cases.

Age	No.	Age	No.
0-1	16	10-15	64
1-2	32	15-20	45
2-3	64	20-25	42
3-4	58	25-30	33
4-5	34	35-40	51
5-6	22	40-45	32
6-7	25	45-50	5
7-8	23	50-60	1
8-9	28	60-70	1
9-10	24		

It will be noted that approximately two-thirds of the cases were under 15 years of age and approximately one-third under 5 years.

It is also important to note that patients from rural districts had a greater tendency to be drawn from the older age groups than those in urban areas. It is of interest that the oldest recorded case was that of a man aged 68 years.

It was also noted that the largest proportion of abortive cases were in the latter age groups, and the maximum number of non-paralytic cases appeared to be in the 5-14 age group.

Horstmann (1946) observed that ~~in~~ the majority of the paralytic cases were in the 0-4 age group and other observers have stated that the great vascularity of the spinal cord about the second year of life may contribute to the susceptibility at this age. In 1948, Bradley and Gale made the important observation that the severity of the disease was much greater in the later age groups; in other words the tendency to severe paralysis increases with advancing years. My series confirmed this.

It should be noted that in the earlier epidemics, the limbs and trunk appear to be mainly involved, but in the more recent epidemics, the incidence of brain stem involvement has been particularly high.

There seemed to be a slight proponderance of males over females and the ratio usually quoted is 5:4. This is

specially true in patients under 5 years of age. It therefore follows that there is an excess of females if the patients are over 5 years of age.

The next table shows the number of male and female cases at the various ages in my series.

	Sex			Sex	
Age	M	F	Age	M	F
0-1	7	9	10-15	32	32
1-2	17	15	15-20	23	22
2-3	33	31	20-25	18	24
3-4	30	28	25-30	16	17
4-5	20	14	35-40	30	21
5-6	11	11	40-45	16	16
6-7	12	13	45-50	2	3
7-8	13	10	50-60	-	1
8-9	13	15	60-70	1	-
9-10	12	12		138	136
	168	158			

It must however be admitted that the epidemiology of poliomyelitis is still undetermined. This is due to the following main factors:

1. The route of the infection is unknown.
2. The manner of transmission is unknown.
3. The uncertainty of making an early and definite diagnosis.
4. The absence of any specific test for susceptible persons., sub-clinical and abortive cases.
5. There is no suitable test for past infection.

Pathology.

Although not a recognised pathologist, I have always taken great interest in the pathological specimens obtained in this series.

Poliomyelitis is caused by a neurotropic virus which acts directly on the nerve cells. The brunt of the attack appears to fall on the ventral horns of the cervical and lumbar enlargements of the spinal cord, but the nerve cells in the

cerebrum, cerebellum, brain stem and automatic nervous system may also be affected. In some cases the virus is destroyed before the nerve cell is damaged to any great extent, and then only a temporary paralysis or paresis results. In other cases, it continues to multiply during its period of activity, resulting in a total destruction of the motor cell, and a varying degree of permanent paralysis.

It was formerly taught that an attack of poliomyelitis conferred a life-long immunity by the production of anti-bodies, but although second attacks of the disease are rare, they are not unknown. Bridge et al (1946) reviewing the literature, found reports of only thirty-one instances of second attacks, and Alves and Pugh (1941) added another. One of our cases gave a history of an attack at the age of six months when the erector spinae group of muscles was affected. The second attack was at the age of thirty-three years and both legs were then involved.

On the subject of morbid anatomy and histology, the whole central nervous system can be affected to a greater or lesser extent.

The virus of poliomyelitis was observed to produce lesions in the cerebral cortex, the basal ganglia and the thalamic and hypothalamic nuclei. The mid-brain, cerebellum, pons, medulla oblongata, spinal cord and the autonomic nervous system were also involved. The lesions in the cerebral cortex were confined mainly to the motor and pre-motor areas, as the virus appears to have a special affinity for the large pyramidal cells in these regions, whilst in the cerebellum they are mainly confined to the roof nuclei, the vestibular nuclei and the reticular formations.

The histological changes are always found to be much more widespread than the clinical evidence would indicate. A very severe inflammatory reaction may be present but without any evidence of correspondingly severe damage to the nerve cells.

On the other hand, I found an extensive destruction of the nerve cells without any accompanying oedema or marked vascular changes, and the inflammatory reaction may be minimal or absent.

I found that the pathological findings were greatly altered or modified according to the length of time which had elapsed between the initial onset and the death of the patient. In fulminating cases, involving the cerebral cortex, mid-brain, basal ganglia and cerebellum, the nerve cells showed practically no change. In cases in which the brain stem was involved, and which lived for twenty-four to forty-eight hours, there was usually a large number of neuronal lesions in the medulla. These were most marked in the region of the nucleus ambiguus, the dorsal nuclei of the vagus nerve, and the nucleus solitarius. In less acute cases, where death had occurred after the onset of the paralysis, the whole of the central nervous system was found to be slightly or moderately oedematous, the brain stem congested and the blood vessels in the anterior horns of the spinal cord distended.

In these less acute cases, if lesions were looked for in other parts of the brain, they were found fairly frequently in the substantia reticularis, in the floor of the fourth ventricle, in the substantia nigra and around the aqueduct of Sylvius. Less frequent and less severe lesions may be detected in the corpus striatum, the globus pallidus, and the hypothalamic nuclei.

I tried to correlate these pathological findings in the central nervous system with those of the clinical findings, and the following observations were recorded.

Softening around the basal nuclei with a definite loss of the ganglion cells was a common finding in cases which have suffered from stupor, coma or disorientation before death. Hysterical and psychological manifestations were

also attributed to lesions of the hypothalamic centres. If there had been marked dysphagia and dysarthria, the lesions were usually found around the nucleus ambiguus.

Sheinker (1947) reported upon a pronounced inflammatory and degenerative tissue reaction in the region of the vagal nuclei, and this appeared to substantiate further the theory of the spread of the virus from the alimentary tract to the central nervous system by means of the afferent and efferent fibres of the vagus nerve. Certain other observers have pointed out that the nuclei of the seventh and twelfth cranial nerves are also often involved. The ganglia of the fifth, ninth and tenth cranial nerves which carry afferent impulses from the pharynx have also shown signs of invasion in a large number of human cases.

In our series, the olfactory bulbs and tracts were examined histologically and they were almost invariably found to be normal. In cases which have died following manifestation of bulbar poliomyelitis, the spinal ganglia may also be found to be of normal appearance.

In the spinal cord, I found that the initial attack appeared to be focused upon certain groups of motor neurones, or to be spread amongst the individual nerve cells in the anterior horns. An important point noted was that if the attack was bilateral, it was usually of an asymmetrical nature.

If the spinal cord was sectioned and examined at different levels, it was found that certain areas showing marked involvement were interspersed with other areas of apparently normal tissue.

I found that typical lesions were most pronounced in the brain stem and anterior horns of the spinal cord at levels corresponding to the paralysed muscle group.

A partial or complete destruction of the nerve fibres in the anterior commissure can also be found. A similar condition may be present in the anterior nerve roots and

muscular branches of the peripheral nerves.

Polymorphonuclear leucocytes are seen outside the blood vessels within a few hours of the onset, but they are later replaced by lymphocytes and plasma cells. It was noted however, that there was no fibrinous exudate. The blood vessel walls are thickened and the perivascular sheaths become distended with lymphocytes and other mononuclear cells. Some of the smaller blood-vessels were thrombosed and small haemorrhages were also present. These appearances were most marked in the grey matter of the spinal cord.

The extent of the perivascular infiltration of the small mononuclear cells appeared to be proportional to the vascularity of the area and it was most evident in the floor of the fourth ventricle and in the cervical and lumbar enlargements. Mayr (1932) found that the spinal cord was most frequently affected below the fourth lumbar segment. This infiltration was most evident in the anterior fissure where the larger veins leave the spinal cord.

Infection may ascend or descend in the grey matter of the spinal cord and in this way several segments were involved. The chief method of spread appears to be along the nerve fibres, e.g. Clarke's column, but probably both sensory and motor fibres can transmit the virus.

Neumann (1950) stated that he thought that the pain complained of was probably due to the involvement of the posterior roots and their spinal ganglia. The posterior horns of the grey matter was also affected in many cases and this caused motor dysfunction by interference with the spinal reflex arc.

Other observers have stated that the most commonly affected parts are those in the regions containing the cell bodies of the internuncial neurones. Attempts have accordingly been made to explain the muscle spasm, alienation and muscular inco-ordination by these lesions, but I have

not observed enough cases to pass a definite opinion.

In a few cases the inflammatory processes extended into the lateral columns of the spinal cord, whilst in the rare cases in which there was an impaired appreciation of the pain and temperature senses, the spino-thalamic tracts were found to be involved.

Fairly recently it has been expostulated that the virus may also extend along the sympathetic fibres to their ganglia. Clinically this has been demonstrated by cyanosis, sweating and disturbances of micturition which may develop before the onset of the paralysis. Also at post-mortem the sympathetic ganglia showed evidence of having been invaded. Some of the ganglion cells are swollen and have clear cytoplasm, but others may be shrunken and no nucleus is seen. The evidence therefore strongly suggests that the virus could travel by the visceral afferents.

Regressive changes took the form of sclerosis or vacuolation of the cells. Degenerative alterations in the neurofibrils and disintegration of the cell walls and nuclei were also seen. The presence of abnormal or increased lipoid deposits in some of the cells were also observed.

Let me now consider cases in which death has occurred during the first few weeks. Here I found an irregular congestion of the leptomeninges over the affected area of the spinal cord and some haemorrhage into the ventral horns. These changes were found to be most marked in the cervical and lumbar enlargements and characteristically they were asymmetrical.

Examination of the anterior nerve roots showed that they had become atrophied and the anterior horn cells appeared to be diminished in number. Some of these cells were seen to be shrunken, distorted or degenerated. There was also an increase in the neuroglia and a coarsening of the delicate

reticulum which the fibres usually form.

Let me now review the pathological changes which may also be observed in the pharyngeal mucous membrane, skin, muscles, bones, ligaments, lymphatic glands and other tissues and organs.

As early as 1912, Neustaedter described the pathological changes which he had observed in the pharyngeal mucosa. He found that it looked anaemic and that it had a glistening and oedematous appearance. In some cases, a serous frothy exudate was present and this persisted for some weeks after the onset of the paralysis. This was confirmed.

The skin should next be examined and it was often found to be hypersensitive and tender during the acute phase. This however could be missed as it lasted in the majority of cases for only a few days. The real cause could not be definitely established but it was attributed to the affection of the posterior nerve roots and the posterior columns of the spinal cord. At a later stage, the skin became less elastic and thickened with disappearance of the subcutaneous fat.

As there was frequently a marked disturbance in the circulation in the involved extremity, vasomotor changes should be looked for in the convalsecent and residual stages of the disease. It usually manifested itself in blueness and coldness of the limb, and perspiration may be excessive. Trophic changes were very commonly present in the late convalescent and residual stages and the patient could suffer greatly from chilblains and ulcers.

The skeletal muscle fibres obey the "all or none law", so that the destruction of a single anterior horn cell results in paralysis of a minute portion of the muscle. It therefore follows that the more nerve cells involved, the greater will be the weakness of the muscle. If all the nerve cells were destroyed, the related muscles will be completely

paralysed, resulting in atrophy and fibrosis or there may be a fatty replacement of the muscle fibres. These changes result in a permanent loss of function or contractile power.

The virus may not cause irreparable damage to the nerve cells and therefore the paresis or paralysis may not be permanent and there is then only a transient impairment of function. In the incomplete lesions it was found that some portions of the muscle function normally whilst others degenerated.

In the bony skeleton, it was found that the bones of the affected limb or limbs underwent atrophy and decalcification. Their shape could also be altered and this, coupled with the above changes, renders them less resistant to pressures and strains. These changes may be attributed to disuse of the limb, alterations in the local blood supply or to primary neurotrophic disturbances.

In a child who has suffered from poliomyelitis, the affected limb grew more slowly than its fellow, and the younger the victim, the more noticeable was this effect, especially if the lower limb was the part involved. This factor may lead to a marked deformity when coupled with the action of the unaffected muscles.

The joints may also be affected but this was usually the result of inadequate treatment or faulty supervision during treatment. If the appropriate treatment is not instituted early, the capsule and ligaments could be subjected to severe strains. This will eventually lead to a marked weakness of the joint, as the result of the stretching of these structures. The joints will therefore become unduly mobile or flail and subluxation or dislocation may occur.

Another deformity found frequently is pes cavus which is due to contracture of the plantar fascia.

Prophylaxis

Compulsory notification should include abortive and non-paralytic cases as these play a major part in the spread of the infection, being much more prevalent than the frank paralytic hospital case.

All children who show any evidence of illness such as fever, nausea, vertigo, retention of urine, pain in the head, neck, back, limbs, chest or trunk, with perhaps some stiffness of the neck and spine should be isolated in bed for one week. They should have the maximum amount of fresh air and sunshine. Other contacts should be carefully observed and at the first sign of illness they should be quarantined for two weeks.

The isolation period usually recommended is three weeks, but as 50% of patients are still excreting the virus in their stools after this time, it is obviously theoretically too short. Brown et al (1948) showed that 20% of the family contacts excrete the virus, and therefore ideally isolation should include the whole family. To be effective, rigid quarantine should be enforced for three to eight weeks, i.e. until the virus can no longer be isolated from the stools. Not only is this rigid isolation impractical, but it is ineffective in preventing the spread of the disease because of the large number of sub-clinical cases, abortive cases and carriers who walk about undetected.

1. Schools and nurseries.

Unless there are definite contra-indications, schools should not be closed because this will not control the infection and it may even tend to increase it. Children who are free to gather in groups in playing-grounds and streets are more likely to come in contact with adult carriers.

If a child is known to be an intimate contact, he should be kept away from school for three weeks from the date of exposure. Owing to the great susceptibility of very young

children, nurseries should be closed if more than one case occurs in them.

2. Crowds.

All persons should avoid crowds and attendance at places where overcrowding can occur, e.g. cinemas, theatres and parties. It is interesting to note that in our series we encountered fairly frequently a history where all the members of the household suffered with "head colds".

3. Swimming-baths and paddling-pools.

Only a limited number of children should be admitted at one time and then only for a restricted period, because exhausting exercise may predispose to the paralytic stage in an infected child.

Hired swimming-suits and towels should be washed in hot water and laundered before re-use and the floors of the changing rooms disinfected.

All fresh water paddling-pools should be drained, but river bathing is perfectly safe provided that no sewage pipe drains into the river.

4. Exercise.

Strenuous and unusual forms of exercise must be avoided at all costsduring the preparalytic stage as this may determine the onset of a severe or even fatal paralysis. Fatigue must be avoided after minor injuries, operations and sunburn. Six of our cases gave a history of bathing and exercise rapidly followed by the onset of paralysis.

5. Personal hygiene and posture.

Parents should insist on children washing their hands after the use of the toilet and before meals.

6 Food.

Personal hygiene must be of the highest order in all those handling food. Water of doubtful purity and unpasteurized milk should not be drunk, and fresh fruits and vegetables should be washed, peeled or scraped before consumption.

Protective foods should be given in adequate amounts.

7. Flies and vermin.

Anti-fly measures and destruction of vermin should be intensified, and the greatest care taken in the disposal of garbage, excreta and sewage. Food bins should have tightly fitting lids and be emptied regularly.

8. Operations and injections.

In the early period of the 1947 epidemic, cases occurred which seemed to be adversely affected by local injury, operation injection or some other physical factor. Paralytic poliomyelitis usually developed four to twenty-one days after the local trauma. The paralysis was maximal or confined to that part of the body which was the site of the trauma. Several of our cases had a previous history of recent acute tonsillitis, tonsillectomy or dental extraction. Three had had a severe attack of measles seven to ten days previously, and in one family, a brother and sister gave a history of recent measles. It is advisable that dental extractions, tonsillectomy and all elective operations on the ear, nose and throat and intestinal tract should be avoided during an epidemic. Mass inoculation against diphtheria and whooping-cough should be restricted to the non-epidemic periods of the year.

One of our cases had an attack of acute appendicitis one month after admission, and the operation was followed by an increased weakness of the right leg.

9. Deformities.

Lastly, if poliomyelitis is suspected, a splint should be applied to protect and support the joints in the position of neutral muscle pull. The tissues should also be kept warm.

These precautions may help to prevent deformity and minimize disability.

Clinical Features.

It must first of all be stated that this disease requires the diagnosis to be made on purely clinical grounds, because as yet the virus cannot be detected by any specific laboratory test. The attacks appear to be irrespective of previous health, social class or environment. The symptoms may be so mild that no definite diagnosis can be made and yet the patient may be highly infective. Certain contacts can act as carriers and spread the infection, although they themselves appear to be perfectly healthy. Early and accurate diagnosis is therefore essential so that adequate measures can be taken to limit the spread of the disease and to minimize the patient's suffering.

Numerous classifications of poliomyelitis have been attempted, but it is my intention to stress only the main clinical features which can be observed at the beginning of the illness and after the various regions of the nervous system are involved. It is important to note that only a few signs and symptoms may be present and that one type may overlap the other. Also the first symptom may be the onset of paralysis or mental confusion without any other prodromal symptoms and further the disease may be arrested at any stage. To avoid missing the earliest signs, a systematic examination is necessary.

The severity of the signs and symptoms varies greatly in different epidemics, and there is a marked difference in the distribution and severity of the paralysis in each outbreak. During the 1947 epidemic in Great Britain, the distribution was as follows:

Paralysis of limbs and trunk	63%
Cranial nerves chiefly affected	10.3%
No paralysis present	23.3%

In the majority of cases, the diagnosis can be made within twenty-four hours of the onset of the symptoms,

especially during an epidemic. As a rule, after the onset of fever, the earliest paralysis to appear is that resulting from damage to the cranial nerves. It must not however be assumed that cranial nerve palsies are a very common occurrence as it is rare for more than one third of the cases to be so affected.

Careful and repeated observations are necessary if complications are to be detected and treated promptly. In some cases of poliomyelitis when the patient is very ill, it is extremely difficult and sometimes quite unjustifiable to make an accurate day to day detailed clinical examination.

There are without doubt a large number of abortive cases which are missed or remain undiagnosed and which because of this mild attack are later found to have a natural resistance to the virus.

Abortive infection.

It is most important from the epidemiological point of view to remember that poliomyelitis may be represented in a particular area entirely by cases of the abortive type. Even during an epidemic, the diagnosis may be only presumptive. In this period of the infection, the poliomyelitis has not progressed beyond the systemic stage in a person who has been exposed to, or resides in the same house as a known case. There are no signs of inflammation of the central nervous system nor is there any permanent damage to health.

These abortive cases should never be admitted to hospital because admission is justifiable only when neurological signs develop. For this reason, daily observation by the physician is necessary so that the earliest signs can be detected. There is no doubt that non-patalytic and often ambulant cases play a much greater part in the spread of poliomyelitis than the frank paralytic case in hospital.

The prodromal stage may last for a few hours to fourteen days, and the symptoms are those which are common to certain

The pulse is usually only slightly or moderately increased in rate whilst the respirations remain unaltered in rate, rhythm and depth, but occasionally there was pain in the chest.

Rarely there was a definite circumoral pallor and occasionally a mild diffuse erythema over the whole body. The patient may also suffer from profuse sweating. Epistaxis was recorded in one of our cases aged 11 years.

In 30% of cases the patient complained of a sore throat, which on examination showed a varying degree of redness,, but oedema and inflammatory exudates were rarely seen. Sore throat was complained of by 30% of my cases, but Pohl(1947) gave a figure of 10% in his review. The distribution was as follows:

Age group	0-5 years	- 21.6%
"	5-15 years	- 40.7%
"	15+ years	- 42.1%

19% of our cases had catarrh of the nose and 16% had catarrh of the chest. The distribution was as follows:

	Catarrh of nose	Catarrh of chest
Age group 0-5 yrs	18.3%	13.3%
" 5-15 yrs	22.2%	14.7%
" 15+ yrs	12.2%	34.7%

The complaint of sore throat occurred eight times more frequently in cases in which bulbar symptoms ultimately developed, than in those which eventually showed paralysis of spinal origin. Paralysis was therefore most carefully watched for in these cases.

Headache is usually one of the first symptoms. It occurred in 75% of cases within twenty-four hours and may be frontal, vertical, occipital, temporal or generalized. Between 50% and 90% of patients complained of headache in the 0-5 and 5-15 age groups whilst the main distribution in this series was 45.5% for frontal headache in the 0-5 age group, and 92.6% for the 5-15 age group. Occipital headache

varied from 15% to 25.3% in the 0-5 and over 15 age groups, respectively. Although moderately severe, the headache is rarely intense, but it may be persistent or throbbing in character. Clinically I found it very difficult to be sure of this symptom in infants.

The tongue is usually furred and nausea and anorexia are frequently present, giving rise to marked distress. Loss of appetite was present in 59% of my cases, and the distribution was as follows:

Age group 0-5 years - 61.6%
" " 5-15 years - 70.4%
" " 15+ years - 52.6%

Vomiting occurred early in 60% to 80% of cases and is usually of sudden onset, repeated and forcible, but lasts only for about forty-eight hours. In my series, vomiting varied from 45%, 59.2% and 42.1% in the 0-5, 5-15, and 15+ age groups.

Diarrhoea may also be present in this stage, and was found in 13%. 9.6% and 6.7% of my cases in the 0-5, 5-15, and 15+ age groups respectively.

General muscular aching and lethargy, or restlessness and fretfulness may be present, but these symptoms were more commonly found in the preparalytic stage.

In this stage, the changes in the cerebrospinal fluid were not specific but only suggestive.

If the disease does not progress any further, it is usually found that the patient remains lethargic for several days.

Preparalytic stage.

In the preparalytic stage there is practically no difference in its symptomatology and that of the current infectious diseases, but it is comparatively easy to make the diagnosis during an epidemic if nuchal rigidity or stiffness of the spine were also present.

A history of exposure to infection was usually but not invariably obtained, but the abortive stage may be so short

that it is impossible to distinguish between it and the preparalytic stage which lasts only from one to three days. On the other hand, there may be an interval of one to three days during which time the patient feels remarkably well. It was most important to determine the onset of the signs of involvement of the central nervous system as compared with the onset of the initial febrile attack, this being usually thirty-six hours.

This stage was characterized by all the signs and symptoms of the abortive type along with those of involvement of the nervous system but without any evidence of paresis or paralysis. The severity of the meningeal and spinal symptoms however, varied greatly from case to case.

In a few cases after the initial rise of temperature observed during the abortive stage, there may be a secondary rise. This secondary febrile phase was usually the first to be onserved by the physician, but its true significance may be missed owing to it being mistaken for the primary one. The temperature usually subsides in three to four days by lysis, rarely by crisis, in both the paralytic and non-paralytic types, but it may remain elevated for ten days. When the patient becomes afebrile, the signs of meningeal irritation disappear, and there is only a minimal risk of further progress of the disease.

The pulse may be rapid or slow and this may be due to an early bulbar involvement.

The general symptoms in the pre-paralytic stage may be slight or severe, but in themselves they were not diagnostic. Mental irritability may be one of the earliest symptoms and it was usually followed by drowsiness which may be very marked in some cases, or occasionally it may alternate with periods of intense irritability. In a few cases however, the excitability persisted and even increased. In other cases, the patient was nervous, fretful, restless, apprehensive and anxious-looking,

or lethargic, apathetic, prostrated or stuperose. Rarely the patient may be critically ill, comatose or suffering from convulsions. 2% of our series were markedly comatose and 3% had generalised convulsions, the distribution being as follows:

	Comatose.	Convulsions.
Age group 0-5 yrs	2.6%	3.9%
" " 5-15 yrs	-	2.4%
" " 15+ yrs	4.0%	3.0%

The patient may lie quietly in bed resenting being disturbed. Definite signs of meningeal irritation may be present, the headache being intense and vomiting being a prominent symptom. Occasionally the sleep rhythm is inverted, the patient being very restless during the night and sleepy during the day. 45% of our patients suffered from insomnia, and 5% had marked stupor, the distribution being as follows:

	Insomnia	Stupor
Age group 0-5 yrs	41.6%	5%
" " 5-15 yrs	55.5%	3.6%
" " 15+ yrs	43.8%	6%

One of our patients, a girl of 23 years, suffered from marked emotional upset. It was noticeable that when convulsions occurred, it was usually in the younger patients, whilst delirium was noticed in patients over 30 years of age.

Gastro-intestinal atony was frequently present and gave rise to obstinate constipation and abdominal distension, but in approximately 10% of cases, diarrhoea was present with very offensive stools. Constipation may also be caused by inactivity, weakness, paralysis of the abdominal muscles, loss of habit or fear of pain. In our series, constipation was present in 56% of cases, in the following age distribution:

	Diarrhoea	Constipation
Age group 0-5 yrs	3.5%	50%
" " 5-15 yrs	7.0%	65%
" " 15+ yrs	12%	63%

The number of patients who complain of bladder symptoms varies greatly, and figures between 2% and 65% have been quoted.

An important point is that these symptoms including dysuria were noted sometime before the onset of paralysis of the limbs. The patient complained early of difficulty in starting micturition or there was a complete retention of urine with an accompanying overflow incontinence. The paralysis was transitory and recovery usually complete within a week, Retention of urine was present in 7% of our cases. A urinary output chart should be kept and other disturbances of the bladder watched for. True incontinence may occur from the onset but it invariably recovers, although it may take several weeks to do so. If infants are affected, the involvement of the bladder may manifest itself by the persistence of enuresis for a number of years. Incontinence occurred in 5% of our cases, in the following age distribution:

	Retention	Incontinence
Age group 0-5 yrs	10%	5%
" " 5-15 yrs	5%	3%
" " 15+ yrs	10%	10%

The remote urinary complications are due to stasis, residual urine and infection from catheterization.

The superficial reflexes, e.g. the abdominal and cremasteric are diminished or lost early. The tendon and skin reflexes may be exaggerated in the preparalytic febrile stage or they may be lost early and be asymmetrical.

It has been said that general hyperaesthesia is a constant finding in the early stages, although it varies greatly in its intensity. Other observers whilst agreeing that hyperaesthesia can occur either generally or locally, declare that it is seldom seen. Severe sacral pain may however be present in patients who often later develop a severe lower limb paralysis. The patient may be seen to lie in a peculiar position in an attempt to ease the pain which is increased by pressure on, or stretching of the muscles. Any attempt to persuade him to assume a reasonable posture either passively or by his own efforts, is markedly resented.

Hyperirritability as demonstrated by local pain, paraesthesia and stiffness of the neck and back muscles is usually attributed to involvement of the nerve roots, internuncial neurones, posterior root ganglia or their surrounding meninges. Areas of hypoaesthesia and anaesthesia are rare.

Changes in the cerebro-spinal fluid were seen in this stage.

It will be obvious that a definite diagnosis cannot always be made at this stage, and it has been estimated that about 95% of cases will make a complete recovery without developing any further symptoms, but all cases were watched carefully for another two weeks in order to exclude poliomyelitis definitely.

Paralytic Stage.

In the 1947 epidemic, of every one hundren persons who showed clinical signs and symptoms of infection, less than five developed into a frank paralytic case. The clinical features varied greatly and any of the abortive and preparalytic symptoms may have been present in a patient who has now developed paresis or paralysis. It is important to note that the symptoms in the preparalytic stage give no indication of the degree or distribution of the ensuing paralysis. To delay in making a definite diagnosis until the paralysis has set in is quite unjustifiable, because valuable time is lost before the initiation of the appropriate treatment.

Walton (1907) stated that no extension of the paralysis was to be expected after the fever had subsided. In the so-called "dromedary type" of temperature chart, the febrile illness usually lasts from one to three days to be followed by a period of one to four days in which the patient feels remarkably well. This however is followed by a second rise in temperature which lasts for one to three days and is usually accompanied by a spread of the paralysis. Another rare type was that seen at the

end of two or three weeks of apparent inactivity and was manifested by an increased pain and tenderness of the muscles and a marked spread of the paralysis.

If the temperature remains elevated, the paresis may progress but to a less extent on each subsequent day. In other cases, each extension of the paralysis was associated with a temporary rise of temperature. The degree of elevation of the temperature did not appear to be of any diagnostic significance, although a prolonged fever appeared to affect the diagnosis adversely.

Nissen (1947) stated that the tongue was moist and of a cold bluish colour. In all cases with paralysis, he noted that there were small macules similar to flea bites around the margin, and these changes lasted for about two weeks. I have been unable to verify this although several of our cases had furred tongues.

Anoxia could be due to abductor paralysis of the vocal cords or from obstruction of the airways by pools of mucus, saliva or vomitus. Other causes may be a reflex closure of the glottis, and pulmonary oedema with a consequent decrease in the alveolar absorptive surface. If there is a diminished cough reflex, aspiration may be required to remove obstructive fluids from the lungs. Anoxia produced damage to the nerve cells, and overaction of the weakened respiratory muscles increased fatigue and may actually increase the paralysis.

Occasionally the patient may be incontinent of faeces from paralysis of the rectal sphincters and unfortunately he may remain so permanently.

Paralysis of the bladder is frequently associated with paralysis of the lower abdominal and thigh muscles. In 40% of cases in one series in which both legs alone were severely paralysed, there was a retention of urine for two to three days, normal control being then spontaneously re-established. In the spinal form of paralysis, the bladder symptoms and dilatation of

the ureters with a secondary pyelonephritis were attributed to an involvement of the sympathetic nervous system.

As a rule, the paralysis appeared about the second to the fifth day after the onset of fever, or one to two days after the onset of the meningeal symptoms. As observed by Russell in 1947, the meningitic symptoms usually abate and the patient feels better before the paralysis develops. On the other hand, the paralysis appeared extremely quickly without any warning especially in children in the 0-5 age group. The paralysis may however be delayed for several days, but in over 95% of cases the maximum degree of paralysis is seen between the first and fifteenth days of the onset. In a small percentage of cases, the paralysis may only become obvious after the second week. In my series of cases, the time which elapsed between the onset of the disease and the first appearance of paralysis was as follows:

No. of days.	No. of cases.	Percentage.
1	35	16
2	46	20.6
3	44	19.8
4	32	14.4
5	14	6.3
6	10	4.5
7	16	7.2
7-14	14	6.3
14-21	7	3.2
21-28	5	2.1

Paralysis may be maximum in its severity and distribution at the onset, or it may, during epidemics progress for one to three days especially in adolescents and adults. Walton(1907) observed that the onset of paralysis was more retarded in adults than in children, and that the extension from one group of muscles to another was less rapid. One hundred and sixty seven of my cases, i.e. 66% showed a sudden onset of paralysis, and 57 cases i.e. 33% showed a gradual one. The paralysis was increased within twentyfour hours in 94 cases (43%), was stationary in 57 cases (25%), and decreased in 69 cases (32%).

In 47% of cases the paralysis was of an ascending type and was descending in 53%. Frequent thorough muscle testing showed a spread of the paralysis for several days after the onset. I found it essential in the early stages to give a very guarded prognosis.

During an epidemic, well over 50% of infected patients will recover fully without showing any signs of paralysis.

Loss of sensation can usually be detected if looked for clinically; for example a loss of pain and temperature sense can be detected in the first week of the illness. It can be bilateral and occur on the opposite side below the lesion. The patient complains of pain in the feet during the night but not during the day. He also complains of severe girdle pain and in one of our cases this feeling of constriction around the waist was very marked for ten days before the onset of paralysis of both lower limbs and abdominal muscles. Another case complained of pain and weakness in the legs for twelve hours before the onset of the paralysis, whilst another complained of numbness of both legs.

In the age group 0-5 years the loss of sensation appeared to be widely distributed, e.g. the sole of the foot, the lateral aspect of both legs, the plantar aspect of the foot, etc. These symptoms are usually only temporary and may last for two to five days.

The tendon reflexes diminish rapidly after the onset of the paralysis and finally disappear or they may appear to be normal at the onset and in a few hours the paralysis may develop. It is important to note that the character of the reflexes can change from one examination to another. Where the disease progresses slowly, the deep reflexes may at first be exaggerated, but rapidly tire when repeatedly stimulated. In 93 of our cases where the knee jerks were recorded, 38 were normal, 41 were diminished and 14 absent. In no case

were they exaggerated. It should be noted that there can be a complete loss of the knee jerks and yet there is no detectable paralysis.

In some cases, with severely paralysed lower limbs, the intensity of the inflammation in the affected region of the cord, leads to a spreading oedema into the white matter. This in turn leads to a transitory extensor plantar response in one or both limbs.

In the later stages, poor circulation and trophic changes result in local sensory disturbances.

Before leaving this section, it is necessary to draw attention to the important fact that has now been fairly well established, namely, the performance of any exercise after the onset of the febrile illness is extremely dangerous and may leave the patient extensively and permanently paralysed. Even the restlessness exhibited by some patients may be harmful.

Myelitic form of poliomyelitis (one case)

A common form is characterized by an upper and lower motor neurone lesion accompanied by a temporary paraesthesia.

There is flaccid paraplegia with extensor plantar responses. Retention of urine is also found. There is a sensory loss over the abdomen and residual spastic paraplegia.

Neuritic form of poliomyelitis (two cases)

The patient complains of severe pain worse on movement in the affected limb. There is tenderness on pressure of the nerves and muscles. There may however be no paralysis, but if there is, it is followed by wasting. The tendon reflexes are lost and there may be disturbances of sensation.

POLIOENCEPHALITIS. (35 cases.)

Bulbar form of poliomyelitis.

Adults were much more likely to develop this form of poliomyelitis than infants and the majority of deaths occurred in this group.

The bulbar type of poliomyelitis varies greatly in frequency

in differant epidemics and Kelleher (1947) found that 30% of the cases seen in the first part of the 1947 epidemic were of the bulbar type. There is no doubt that there is an increase in the proportion of cases of polioencephalitis.

The preparalytic stage appeared to be shorter than in the purely spinal type, lasting one to two days, and it may be preceeded by high fever, rapid pulse, severe headache and nuchal rigidity. The fever may last for ten days and it can be prolonged even longer if secondary respiratory infection occurs, Severe, forceful and unremitting vomiting may also be present along with a sore throat and laryngitis.The patient shows evidence of extreme malaise and drowsiness, or intense excitement, marked restlessness, irritability and apprehension may be the chief signs,

As Boines (1947) pointed out, an early diagnosis of polio-encephalitis can be made if the muscles supplied by the cranial nerves are carefully examined. The first indication of paralysis is dysphagia or a slurring of the speech. Weakness or loss of power of coughing or an ineffective cough may also be present, and secretions may collect in the mouth. and air passages.

Wolfe (1894) reported on a case with involvement of the first, third, fourth, sixth, seventh, and ninth cranial nerves, and Steiglitz (1897) described a case in which the facial palsy preceeded the constitutional symptoms and consequent paralysis by five days. In my series, 35 cases had various combinations of involvement of the cranial nerves of which the following were the most common. Seven cases had involvement of the 9th cranial nerve and in four cases, the 7th and 9th were both involved. Other combinations noted were the 3rd and 10th, 5th and 7th, 7th and 12th, 9th, 10th and 11th, and in one case, the 9th, 10th 11th and 12th.

In my series we observed unequal pupils in 3% of patients, diplopia in 6%, nystagmus in 4% and photophobia in 19% the

age distribution being as follows:

	Unequal pupils	Diplopia	Nystagmus	Photophobia.
Age group 0-5 yrs	6%	1%	2%	14%
" " 5-15 yrs	1%	6%	4%	12%
" " 15+ yrs	4%	10%	6%	20%

One case suffered from intermittent blindness extending over a period of three days.

Seventh Cranial Nerve.

In my series, there were 15 cases of paralysis of the 7th cranial nerve one of which was bilateral.

Facial nerve paralysis is seldom seen as the only sign of paralysis and is usually accompanied by an involvement of the palate.

If complete recovery from the facial paralysis does not occur within one year, then there will be little or no further improvement.

Cerebral form of poliomyelitis. (21 cases)

In this type, the frontal ,parietal and occipital areas of the cerebrum are involved.

According to Powell (1937) the most important points are the signs volunteered by the patient or his relatives, such as nuchal rigidity, drowsiness etc. which are not present when the patient first comes under the observation of the doctor.

In the less severe cases, there is fever with flushing of the face, vomiting, general malaise and listlessness. This may be accompanied by troublesome yawning, diplopia and insomnia or somnolence. In the more severe cases, there is an extremely rapid tremor and twitching of the facial muscles and of the extremities. In the most severe cases, the patient may become drowsy or even comatose, whilst in others there may be unilateral or bilateral convulsions extending over several hours. This may be followed by complete recovery, hemiplegia or paralysis of one limb with a subsequent retardation in the

growth of that limb. Mental deterioration may occur, or athetosis, choreiform movements or epilepsy may be the sequelae.

In adults the mental symptoms may be very marked, and the patient may be irrational and suffer from hallucinations, marked confusion and anxiety. Other cases may be apprehensive, fretful and distressed out of all proportion to their pain or discomfort. Other symptoms which may be present are hyper-excitability, restlessness, irritability and delirium.

Cerebellar form of poliomyelitis.

The patient complains of intense headache, vertigo and vomiting on moving the head. Nystagmus may also be present.

The ataxia in poliomyelitis is generally regarded as cerebellar in origin and if so it is usually accompanied by retraction of the head and nuchal rigidity. This ataxia may however be cerebral or spinal in origin. A female patient aged 22 years showed all the signs of acute cerebellar ataxia but did not develop any paralysis. The signs persisted in varying intensity for eleven days before the onset of the illness. Acute cerebellar ataxia occurred in 2% of our cases and vertigo in 18%, in the following age distribution:

	Ataxia	Vertigo
Age group 0-5 years	1%	14%
" " 5-15 yrs	4%	20%
" " 15+ yrs	1%	22%

Very rarely there was also emotional instability, gross ocular tremor and tremor of the head and limbs.

Summary.

1. The incubation period from the time of exposure lasts for seven to fourteen days.
2. The systemic phase lasts for one to fourteen days and the following clinical features may be observed:

a. Sick, fretful, restless patient.
b. Moderate fever.
c. Malaise.
d. Head cold.
e. Sore throat.
f. Injected pharynx.
g. Nausea.
h. Vomiting.
i. Intestinal upset.
j. Enlarged cervical glands.
k. Occasional pain in chest and limbs.
l. Weakness.

95% of cases make a complete recovery whilst 1% to 5% progress to the preparalytic stage in one to four days.

3. The preparalytic stage lasts for seven to fourteen days, but may be as long as three weeks. The patient has an expression of impending disaster and is most apprehensive. He looks acutely ill, has a temperature of 102°-104°F, and may be completely prostrated. The face is flushed and the tongue furred. There is severe headache and backache and there may also be an increasing stiff neck and/or back. The muscles may be sore or tender and there is pain in the neck and spine when bending the head forwards. Tremor and ataxia may also be present. The reflexes are altered and may be exaggerated and asymmetrical. Occasionally there is vomiting, drowsiness, irritability, coma and nystagmus. Abnormalities are detected in the cerebrospinal fluid. The cells are usually increased in number up to 250/c.m.m. and are chiefly lymphocytes. The globulin test is positive, and the glucose contents may be normal or increased.
4. The paralytic stage usually lasts for thirteen to eighteen days but may be as long as five weeks. 50% of cases recover without signs of paralysis, but 10% to 50% are found to have some degree of paralysis. In this stage, there is an early acute period when the lesions are active, and there is muscle paresis or a flaccid paralysis. The reflexes are usually diminished or absent.

Respiratory failure.

It is most important to recognise the difference between respiratory failure due to involvement of the respiratory centre and affection of the cervical and thoracic cord with paresis or paralysis of the primary muscles of respiration. It is not always possible to differentiate between the different types of respiratory failure and occasionally they may both be present in the same patient. Paralysis of the respiratory centre was found in only 4% of our cases and was distributed

as follows:

Age group 0-5 yrs - nil
" " 5-15 yrs-3%
" " 15+ yrs -6%

1. Involvement of the respiratory centre.

This may be preceeded by a rise in temperature and pulse rate, cyanosis, sweating, anxiousness and apprehension. Periods of mental confusion and hiccup were often present.

Involvement of the respiratory centre was evidenced by and irregular jerky type of respiration which was completely without rhythm, or alternatively a sudden respiratory failure occurred. The respirations were shallow in depth and the intervals between them became more and more prolonged until the breathing stopped completely. Pulmonary oedema occurred and Cheyne-Stoke's respirations usually preceeded the fatal termination. The patient died suddenly of respiratory failure usually within twenty-four hours of admission to hospital. The above symptoms occurred in spite of the fact that the intercostal muscles and diaphragm were normal and remained unparalysed.

Involvement of the respiratory centre and the cranial nerve nuclei is usually accompanied by involvement of the circulatory centre.

In the early stages, the face became flushed and the lips bright red. The pulse rate, which may be regular or irregular, increased to 150 or more per minute and was thready in character. Tachycardia and irregularity of the heart were recorded, and also hypertension especially in children. In most cases however the blood pressure fell, the skin became cold and clammy and the patient was apprehensive, restless and confused.

2. Spinal type of respiratory failure.

This is due to the involvement of the motor cells supplying the respiratory muscles and may not be clinically obvious for several days after the onset. The diaphragm and intercostal muscles may be affected separately, but one is usually involved to a greater degree than the other.

Grawitz (1896) reported on the first case of an adult with paralysis of the diaphragm due to poliomyelitis. This I found to be present in 10% of cases, in the following age distribution:

Age group	0-5 yrs	- 2%
"	5-15 yrs	- 14%
"	15+ yrs	- 4%

As long as the temperature is elevated and the acute illness persists, the respiratory muscles should be watched frequently for the earliest signs of impairment of function.

If there is early paralysis of the cervical muscles, the shoulder girdle or upper extremity, there is always the possibility of respiratory paralysis. Again, if there is paralysis of the lower extremity with an accompanying upward extension, then the diaphragm and intercostal muscles are fairly frequently affected.

As respiratory failure sets in, the respirations become increasingly difficult and more rapid and shallow. Dyspnoea is present and the cough reflex is weakened. As the result of the paralysis, the excursion of the chest wall and/or diaphragm is diminished. The patient becomes fatigued, restless, irritable and unable to sleep. Asymmetric and other abnormal but perfectly rhythmical movements of the chest may be noticed.

Later, the respirations become extremely rapid and the accessory muscles of respiration are brought into action. Occasionally the sternomastoid and trapezius are markedly contracted, thus holding the thorax in the position of inspiration and therefore making breathing most difficult.

The alae nasae dilate and cyanosis develops, gradually becoming more marked. The patient is disinclined to talk, and there may be twitching at the corners of the mouth, but consciousness is usually retained until the end.

Cerebrospinal fluid and blood.

Cerebrospinal fluid.

In any large epidemic, there is usually a marked discrepancy in the findings, and naturally the value of lumbar puncture in the diagnosis of poliomyelitis has been much discussed.

Dennig and Tartter (1933) advised the examination of the cerebrospinal fluid in doubtful cases, and remarked that in an epidemic period , many cases may have fever without any significant changes in the cerebrospinal fluid, and yet are suffering from a mild or abortive attack of poliomyelitis.

During the period of the systemic infection, the cerebrospinal fluid is usually normal and remains so during the prepatalytic period.

During the general infectious period, and the latent period, lumbar puncture is usually negative, but with the penetration of the choroid by the virus and the onset of meningitic signs, lumbar puncture is usually positive. This is also true if there are signs of an ataxic tremor, slight unrecognised weakness or a definite paralysis.

If the pressure happens to be raised, it usually lies between 150 mm. and 200 mm. of water. In 90% of cases, the fluid is clear and colourless, whilst in the remaining 10% it is faintly opalescent. A fine pellicle formation may appear on standing. This net-work of fibrin and contained blood-cells may be present within a few days of the onset of the paralysis or if the protein content is over 90mgm. per cent.

It should be stressed that the cell content of the cerebrospinal fluid should be examined immediately after its

collection because the number of polymorphonuclear leucocytes falls rapidly if the fluid is kept.

There is a definite progressive increase in the number of cells during the preparalytic stage. The average count is 50-200 per cmm. and it is found that the polymorphonuclear leucocytes predominate in the first few days, to be succeeded later by the lymphocytes which usually outnumber the polymorphonuclears by the end of the second week. In some cases however, the cell count may be normal at the end of this time, and at this stage the protein content, glucose and chloride levels are almost invariably normal.

In about 90% of the paralytic cases, there is an increase in the cellular contents to between 30 and 900 cells per cmm. but occasionally this may rise to 2,000 per cmm.

During the first week of paralysis, the cell count may remain high, but usually by the seventh to tenth day, it is starting to fall and reaches normal about the twentyfirst day. Coinciding with this fall in the total number of cells, the percentage of polymorphonuclear leucocytes falls rapidly and that of the lymphocytes rises.

Large mononuclear cells and plasma cells are also occasionally found.

If there are signs of bulbar involvement, the total number of cells is usually small.

Some cases with high cell counts were of the abortive type whilst some with extensive paralysis showed the presence of only a few cells. Some observers argue that a high cell count shows a protective mechanism and that a low count indicates that the resistance of the patient is low, but this is by no means a satisfactory explanation. With the establishment of paralysis, the cells tend to disappear rapidly.

The protein content of the fluid is usually normal in about 40% of cases during the first week of the disease, and then rises steadily to reach its maximum about the end of the third week.

This may be in the region of 300 mgm. per 100 cc. of fluid or as high as 800 mgm. It then falls gradually during the next two to three weeks, but it may remain high for as long as six to eight months.

It must be emphasized that any alteration in the number and character of the cells in the cerebrospinal fluid bears no relationship to the type and severity of the disease, nor is there any correlation between the increase in the protein content and the extent of the paralysis.

The majority of workers report an increase of globulin in the cerebrospinal fluid and it frequently remains high for about a month. The chloride and glucose content of the cerebrospinal fluid usually remains normal. The colloidal gold curve is invariably abnormal and shows a rise in the paretic but more so in the leutic zones.

Blood.

It has been stated that there is no correlation between the clinical types of poliomyelitis, the severity of the paralysis, and the elevation of the blood sedimentation rate. Neither does the level of the white cell count afford any guidance as to the ultimate prognosis.

The blood count is very variable and in exceptional cases, a leucopenia may be present. The monocytes and lymphocytes show no characteristic features and the picture is that usually seen in any other acute infection. Leucocytosis may be present and is usually a lymphocytosis. In one of our cases with a white cell count of 6,000, 48% were polymorphonuclear leucocytes, 46% lymphocytes, and 4% eosinophils. In another case where the white blood count was 16,000, the eosinophils were 22% and eosinophilia was present for several weeks. Extensive search failed to reveal any explanation.

Anaemia does not appear to be a predisposing factor to the onset of poliomyelitis nor does it invariably occur in the convalescent stage.

In polioencephalitis, a slight leucocytosis and a moderate hypochromatic anaemia have been reported.

The blood sedimentation rate may be normal in uncomplicated cases or it may be raised in 50% of cases at the start of the disease. This elevation makes the test of little help in differential diagnosis, but if the rate is markedly raised, the diagnosis is more likely to be that of cerebrospinal meningitis.

Certain observers have attempted to ascertain whether a patient with any particular blood group is more liable to contract poliomyelitis, and it has been stated that patients with blood group O are relatively susceptible, whilst group B patients are normally resistant. My own results compared with other observers are shown in the following table:

Blood Group	% population in each group	% of cases in each group.	
		Taylor	Other observers.
AB	3	0	0
A	42	55	30
B	9	5	5
O	46	40	65

It would therefore appear that the blood changes in poliomyelitis although varying from case to case, are of very little diagnostic significance nor does any particular group appear to be unduly susceptible.

MUSCLE.

Muscle involvement is usually more widespread than was at first suspected. It may be accepted that if one muscle is considerably affected, other muscles are nearly always weakened

The less common muscular signs and symptoms are probably due to virus lesions in the motor cortex, vestibular nuclei, reticular formations or other parts of the extrapyramidal tracts. These are twitchings, tremors and tenderness which

may very greatly in intensity and have been observed in some epidemics in 95% of cases.

Muscle tremor.

Muscular tremors are usually present in the upper limb and are seldom seen in adults. If the patient is asked to extend the arm and to hold it in this position, fine muscular tremors may be noted. The tremor frequently indicates the localization of the paralysis and may precede it by a few hours. Occasionally however the tremor may be present but no paralysis develops. In cases with tremor, the voluntary movements are apt to be jerky in character, and in fulminating cases the tremor may involve the whole body. Jerky movement of the limbs was noted in 22% of our cases and twitching of individual muscles in 12% the age distribution being as follows:

	Jerks	Twitchings
Age group 0-5 yrs	13%	5%
" " 5-15 yrs	33%	18%
" " 15+ yrs	7%	17%

Muscle tenderness may be entirely absent in muscles which subsequently become paralysed, or there may be the same distribution of tenderness and subsequent paralysis. On the other hand, there may be an equal distribution of tenderness in all the affected muscles and yet there is an unequal degree of paralysis. It has also been stated that the paralysed muscles may be the most painful. This is especially true in the case of the deltoid which may later be found to show no evidence of recovery.

During the acute stage of the illness which may last from one to six weeks, one of three types of pain and tenderness may be elicited from the muscles:

1. Pain due to muscle spasm which is variable in amount.
2. Pain elicited on palpation or deep pressure.
3. Pain elicited on stretching the muscles.

Each of these three types of pain is quite distinct and care must be taken to differentiate between them.

The muscle pain and tenderness may be localized or have a segmental distribution, and it is invariably most obvious in the neck, back and extremities. Pain in the limbs occurred in 21% of my cases and lasted for days or weeks after the temperature became normal. The age distribution was as follows:

Age group 0-5 yrs 30%
" " 5-15 yrs 28%
" " 15+ yrs 18%

The pain is usually evoked or exaggerated by touch, pressure or movement, and the site of the pain may denote subsequent severe paralysis. Muscles which are the least affected by paralysis may be the most tender but on the other hand, some cases with severe and widespread paralysis are painless throughout. The pain may be persistent and may indicate an abnormal condition within the affected muscles, but apparently healthy muscles may also be acutely painful and tender. Contracted muscles when put on the stretch may be painful, and many of them have no voluntary contractile power.

Muscle tenderness lasted in the majority of my cases for periods varying from one day to one week and in the minority it lasted up to five weeks. This is shown as follows:

Time in days	No. of cases	Time in weeks	No. of cases.
1	15	1	113
2	29	2	26
3	21	3	16
4	15	4	3
5	12	5	2
6	5		
7	16		
	total 113		

Degree of muscle tenderness.

This was particularly noted in 24 cases . There was slight tenderness with severe subsequent paralysis in 9 cases. The muscles of the legs and arms were very tender but there was no resultant paralysis in 8 cases, whilst there was moderately severe pain with slight paralysis in 5 cases. In the remaining 2 cases both pain and paralysis was slight.

Muscle spasm.

In my opinion, there are two types of muscle spasm. The first type is the early spasm of the acute stage which lasts for a few days. This transitory spasm is never a therapeutic problem as it usually disappears spontaneously. It is relieved by moist heat or by sympathetic paralysants. The second type is the spasm which persists for more than a month and it is most troublesome as it may interfere with the general treatment of the patient. It is reflex contracture due to a pain stimulus and it is frequently present along with stiff joints and contractures. It is only present if there is a limitation of movement in a particular limb.

As Luft and Muller (1947) pointed out, the occurrence of spasm does not appear to depend on the degree of injury to the peripheral motor neurones, and there is no relationship between the degree of paresis and the frequency and intensity of the spasm. These observers are also of the opinion that spasms may be due to vasomotor disturbances.

One of my cases of muscle spasm was treated by a para-sympathetic block with an immediate increase in the temperature of the left leg which became more relaxed, and passive movements became possible. Spasticity returned on the second day, but after manipulation it relaxed and by the end of the first week a full range of passive movements was obtained.

In one of my cases, spasm of the psoas muscle was followed a week later by a flaccid paralysis. This case was first diagnosed as a tuberculous hip joint.

The incidence of muscle spasm appears to differ greatly in different countries and in this country has so far been rare except as a manifestation of meningeal irritation.

Richards et al (1947) pointed out that some patients during the first few days complain of a very slight stiffness of the neck and back muscles which later increases and may or may not be accompanied by paralysis. It may be of minimal intensity and disappear rapidly when the inflammatory process in the cord subsides. It may on the other hand, spread extensively to other parts of the body and persist for weeks or months whether treatment is given or not. The presence of spasm is shown by the painful sensitivity of the peripheral tissues, e.g. nuchal rigidity, stiff back and shortened hamstrings. Nuchal rigidity is caused by inflammation or irritation of the dorsal nerve root ganglia and meninges and all degrees are met with. It was present in 28% of my cases the age distribution being as follows:

Age group 0-5 yrs	26 %
" " 5-15 yrs	40%
" " 15+ yrs	26%

The head can be flexed without any resistance through a fairly large arc, whereas in meningitis the head is definitely retracted.

Kernig's sign is positive in a large number of cases but it is frequently absent in infants. In 19% of our cases, this sign was positive and head retraction was present in 14%, the age distribution being as follows:

	Kernig	Head retraction
Age group 0-5 yrs	21%	15%
" " 5-15 yrs	15%	18%
" " 15+ yrs	17%	12%

A stiff back, due to muscle spasm, which occurred in 55% of my cases, may be diagnostic if other local causes can be excluded.

The age distribution in cases with a stiff back was as follows:

Age group	0-5 yrs	53%
"	5-15 yrs	50%
"	15+ yrs	60%

If the patient is placed in a sitting position, he will support himself by placing his hands behind him in the so-called tripod position (Amoss's sign). Difficulty in maintaining the sitting position may be due to spasm or shortening of the hamstring muscles, and if the patient is made to sit with his knees flexed over the edge of the bed, it will be found that he can straighten his back fully. If, whilst in this position, he is asked to touch his knees with his nose or lips, he is unable to do so. In a few cases, the back rigidity produces such severe pain that even slight flexion is impossible. The patient therefore assumes the position of opisthotonos, and if the spasm of the neck and back muscles is prolonged, it may result in considerable shortening of the muscles in this position.

Spasm of the respiratory muscles may lead to marked dyspnoea, whilst spasm of the abdominal muscles may simulate appendicitis, as was seen in two of my cases.

Careful investigations of muscle spasm seem to show that it does not initiate the development of muscle weakness. Spasm however has been said to impair the muscle function, and its relaxation reduces the number of, or completely prevents deformities. This relaxation can therefore in itself be considered a sign of improvement.

Muscle spasm may cause contractures and deformities by muscle imbalance, and if not adequately treated, there is loss of function, contraction, fibrosis and muscular atrophy. Pain and limited movement even after several weeks of treatment may be due to fibrosis, contractures or adhesions. These are the main

causes of muscle shortening, and should be treated by passive stretching by an experienced physiotherapist. Muscle spasm is aggravated by massage, premature weight-bearing and exposure to cold.

Paresis and paralysis.

In my series, 50% of cases were of sudden onset and in the other 50% the onset was gradual.

It has been stated that the apparent weakness or paralysis seen during the acute stages of poliomyelitis is the sum of at least three differenct effects:

1. Pseudoparalysis - resulting from pain and tenderness.
2. Temporary paralysis resulting from temporary loss of function of the anterior horn cells which can recover without axonal degeneration. Tested electrically, the muscles do not show the reaction of degeneration.
3. Permanent paralysis resulting from irreversible damage to the anterior horn cells.

It should be noted that the paralysis seldom spreads for more than about three days. During this period of progressive paralysis, the patient must be carefully observed for any evidence of respiratory paralysis due to bulbar or spinal involvement. If this complication arises, there is continued fever, and a slow and progressive paralysis up to the time of death.

The muscles appear to be involved in proportion to their activity or fatigue, and experience has shown that the most highly developed muscles are those which are the most severely affected, e.g. the forearm muscles in a violinist.

The paralysis may be masked by the presence of muscle pain and tenderness or the paralysis may be considered to be much more severe than it really is owing to a reluctance on the part of the patient to move the limb because of pain.

Involvement of the spinal cord results in a weakness or

flaccid paralysis of one or more of the muscles of the thorax, abdomen, back, and upper and lower extremities. This paralysis may affect a few of the muscle fibres in a muscle, any one particular muscle, a group of muscles, or any combination of muscle groups in either the arm or the leg. In the more severe cases, it may affect the whole arm or leg or any combination of the four limbs, with or without involvement of the muscles of the trunk. The muscular signs vary from those of minimal weakness to those of complete paralysis. It is always essential to examine and compare the muscles of each side because in some cases, very mild paresis can be detected only by so doing.

Seddon stated that the lumbar enlargement was involved in 86.3% of his cases and that there was an equal involvement of the two limbs. This has been confirmed in my series when both lower limbs were equally affected. Other observers however, have found that the left leg was more often affected than the right. There is no doubt that the legs are affected about twice as often as the arms and that the paralysis is usually more severe. The right arm was affected five times as frwquently as the left. Hemiplegia was not a common finding, but it was twice as frequent on the right side. All four limbs and trunk were affected in three cases. The anterior tibial, peronei, quadriceps and gluteal muscles tend to be constantly and severely affected. The proximal muscle groups are said to be more affected than the distal and this was true in my cases. The extensors of the knee, hip and foot are more often affected than the flexors. The abductors of the hip are more often affected than the adductors and the evertors of the foot more often than the invertors. The hip abductors seem to be more prone to bilateral involvement than the tibialis anticus, soleus and peroneal muscles.

If the muscles of the foot and toes are paralysed, the plantar responses are flexor or absent. Alterations in, or loss of the reflexes does not necessarily mean that paresis

or paralysis is present, but this is much more probable if the reflexes are asymmetric. A pseudo-Babinski sign may result from paralysis of the flexor muscles whilst the extensors retain their power of contraction.

Cases may occur with paralysis of one or both upper limbs and in which the knee or ankle reflexes or both may be diminished or absent and yet there is no sign of muscle weakness in the lower limbs. If these cases are followed up for a considerable time, it will be found that the reflexes recover fully in a large percentage of cases.

The arms are more frequently involved than the trunk, and the proximal arm muscles are affected more often and more severely than the distal ones. The right arm appears to be affected twice as often as the left. The deltoid is the muscle most frequently involved, the next in frequency being the biceps, triceps, brachialis, brachio-radialis and opponens pollicis. Paralysis of the deltoids and shoulder girdle may be an indication of an impending or an associated intercostal or diaphragmatic paralysis.

If the spread of the paralysis is of the ascending type, it usually commences in the legs and involves progressively the abdominal and back muscles, the thoracic muscles, and finally the neck and respiratory muscles. The paralysis may however halt at any stage and then show signs of regression, and it may eventually leave the patient with only a very slight residual paralysis.

In my series, the ascending type occurred in 30% of the 5-15 age group and 45% in the 0-5 age group. The dsecending type was found in approximately 60% in all age groups.

In the ascending and descending types, the progress of the active disease may proceed for several days but it is occasionally interrupted by a period of remission. In my series, 80% occurred in the first twentyfour hours. A most

peculiar type of remission is that where the limbs are involved one at a time at intervals of approximately twelve hours, the maximum spread having occurred in about four days. This type of case may end fatally from the involvement of the respiratory muscles.

The abdominal muscles may be affected in 80% of paralytic cases and this is usually bilateral. In cases of paralysis of the lower abdominal muscles if the head is raised, the umbilicus moves upwards. This indicates a lesion at the level of the tenth intercostal nerve (Brevor's sign).

If the spinal muscles are involved, and especially if this is combined with an asymmetrical affection of the abdominal muscles, a special watch must be kept for spinal curvature.

Usually the paralysis gradually diminishes, the distal muscles recovering more quickly and more completely than the proximal ones. Clinically some of the muscles are permanently paralysed and finally degenerate in whole or in part, depending upon the degree of damage to their controlling motor nerve cells. These completely paralysed muscles show the reaction of degeneration but the degeneration of a muscle is a very slow process. Other muscles undergo disuse atrophy whilst their controlling motor nerve cells are temporarily out of action; but with the resumption of the normal function of the cells, even very weak muscles may recover reasonably good or normal strength.

Muscle testing and charting.

If the patient is acutely ill, accurate muscle testing is impossible and unjustifiable. An experienced observer however should be able to obtain a reasonably accurate picture of the degree and extent of the muscle involvement, and this will allow adequate splinting to be applied at the earliest moment.

It has been stated that if an attempt is made to test

all the muscles in one session, the patient will become exhausted long before the rest is completed and therefore the later results will be inaccurate. In the early stages of the disease a more accurate assessment will therefore be obtained if the muscle testing is spread over two to three days. In this unit, all patients except small children are tested in one session and I have not observed any detrimental effect attributable to this practice. The picture may of course be obscured by the presence of excessive pain and muscle spasm.

On admission, each case should have a chart of the whole muscular system completed, otherwise a weakened muscle may inadvertently be missed. This chart which is part of the diagnostic procedure, forms the keystone of the rehabilitation programme and the whole course of treatment to the muscles depends upon its accuracy. It should be stressed that frequent thorough muscle testing might increase the paralysis and should be avoided.

The muscle chart shows the extent of the original involvement and the rate and degree of the return of muscle power. It therefore helps to determine the prognosis, always bearing in mind the interval between the onset of the disease and the initiation of adequate treatment.

The system of grading used by us is that recommended by the Peripheral Nerve Injuries Committee of the Medical Research Council and this gives the following valuations:

0 - no contraction present.
1 - flicker of movement which can be seen and felt.
2 - muscle contraction with gravity eliminated.
3 - muscle contraction against gravity only.
4 - muscle contraction against gravity and resistance.
5 - normal muscle contraction.

(Examples of useful types of muscle charts are given in the appendices.)

The tests are made by giving as much assistance as possible to the muscle being tested, and it is wiser to underrate rather than to overrate the power of a muscle. It need hardly be

mentioned that it is always the primary action of the muscle or single group of muscles which is tested. If it is impossible to test an individual muscle, then a single muscle group containing it may be taken and recorded.

In very young children and infants, accurate manual muscle testing is impossible and only a very rough estimate can be obtained. The patient may lie with the lower limb completely immobile, but if complete paralysis is not present, he will usually withdraw the limb when the sole of the foot is tickled. The movements of the upper limb can be tested roughly, by holding a toy in different positions in front of the child, and asking him to reach for it.

In this unit, we do not insist on the tests being carried out by the same person, but he or she must be a member of the poliomyelitis team. As a result we find that the records tally very well, and in addition one observation is always a check on another.

From the muscle chart a preliminary estimation of the extent and distribution of the major muscle weaknesses and an assessment of the factors producing deformity can be arrived at. Hyperaesthesia, muscle tenderness and spasm should also be noted. As passive movements can also be used to determine whether limitation in movement is due to muscle weakness or to some other predisposing factor, the ranges of these movements should always be recorded.

Notes on individual muscle paresis or paralysis.

In poliomyelitis, paresis or paralysis of an individual muscle is very rarely seen, and it is much more common to find that one or more muscle groups are affected.

It is also found that the paralysed muscles fall into one of three categories:

1. Those in which the anterior horn cells are only slightly and temporarily damaged. The muscles, if they do waste, will do so only temporarily and they will recover their

full function within a few weeks or months.

2. The majority of the anterior horn cells are seriously damaged but a few may escape. Atrophy is incomplete, and during the following two to three years, the muscles will partially recover their function, but they will always remain permanently weakened and atrophic.
3. The anterior horn cells are completely destroyed and the muscle fibres are replaced by fibrous tissue.

Although limps due to paralysis of individual muscles are frequently described, they are rarely seen. Limps when present are usually of a complicated nature, being due to the paralysis of several muscles.

Treatment.

Most observers attempt to fit each individual case into a specific treatment group, whereas in my opinion, each patient must be dealt with as an individual problem in order to obtain the best results.

Throughout the whole of the treatment, it is absolutely essential that all the members of the poliomyelitis team should remember that the condition of the patient is constantly changing in character. Whatever method of treatment has been decided upon, it must be repeatedly reviewed so that it can be adapted or changed as the condition of the patient alters with his response to treatment. These alterations should always be carried out gradually and with the greatest care, and children especially must be supervised during the growing period otherwise deformities may progress, sometimes with great rapidity.

The treatment should vary according to the age of the patient, the stage of the disease and the time which has elapsed between the onset of the disease and the commencement of adequately supervised treatment.

If cases are seen in the later stages, it is essential to know the extent of the original involvement and then to

ascertain the extent of the paresis, paralysis or deformities present, before considering what improvement there has been in the individual muscles. The type of previous treatment should also be ascertained and then, having considered all the relevant factors, a definite line of treatment should be embarked upon.

Let me now give a resumé of the essential points in the treatment of a case of poliomyelitis.

1. Absolute mental and physical rest during the acute stage, preferably in a darkened room.
2. Skilled nursing technique.
3. Careful diagnostic study.
4. The maintenance of correct body alignment of the affected parts, and checking at frequent intervals to prevent or limit any tendency to stretching or contracture.
5. Light casts or splints carefully applied where indicated, especially for night use in children to prevent stretching of the weakened muscles.
6. Warmth to promote the circulation and thus improve the nutrition and elimination of waste products from the paralysed parts.
7. Physiotherapeutic treatment to prevent stiffness of the joints and to counteract the effects of muscle spasm.
8. Accurate localized muscle re-education as soon as active motion is possible without pain or muscle irritation.
9. The avoidance of weight-bearing during the period of recovery.
10. The restoration of maximum function, joint stability and the maintenance of correct posture.
11. Constructive psychotherapy by encouragement and reassurance.
12. Over-treatment must be guarded against and adequate periods of rest enforced, as gross muscular fatigue must be avoided.

In the abortive case, the patient should be confined to bed in his home for two or three days after the temperature has returned to normal. In the preparalytic case the patient should be at complete rest in bed for at least four weeks, and in cases which have shown marked initial symptoms, for a period of not less than six weeks.

In the preparalytic stage, the doctor may be uncertain whether he should transfer the patient to hospital immediately and until this has been decided, the patient must be kept very quiet both physically and mentally by adequate doses of sedative drugs, so as to prevent restlessness and to promote sleep. At the first sign of bulbar palsy, hospital treatment is urgently required because of the danger of a rapid spread to the respiratory centre.

For prophylaxis of known or suspected infections, a normally practised aseptic technique is most important. The suspected case may be suffering from meningitis, rheumatic fever etc., and an efficient fever hospital is the best place to treat cases of this kind. If the case is one of poliomyelitis, barrier nursing for about three weeks is required, and it is therefore best to admit the case to an isolation hospital. Here the nursing staff have the necessary experience in the use of the respirator and in the nursing of tracheotomy cases. Special hospitals especially in this country are uneconomical and impractical from the medical, nursing and financial points of view. The main reason for this is that the disease is a seasonal one, and therefore the facilities would be out of use for long periods of time.

It is most essential to have a full team consisting of a physician, orthopaedic surgeon, physical medicine specialist, physiotherapist, occupational therapist, almoner and a fully trained nursing staff, all of whom combine their own special knowledge for the ultimate benefit of the patient.

In the paralytic cases, during the first four to six weeks,

the clinician is able to get a complete picture of the functional weakness. The proposed treatment may be conveniently divided into three stages, but we must remember that this division is not a rigid one but that one stage invariably merges into another.

Acute stage.

The acute febrile stage usually lasts for four to seven days and occurs during the three-week period of isolation in the fever hospital. During the acute stage expert nursing combined with rest and relaxation of the muscles are essential. The patient is not allowed to sit up and passive movements, active exercises etc., are contra-indicated.

All cases should be seen by the orthopaedic surgeon within twenty-four hours of the onset, and a muscle chart may be made out at this time to record the power of the affected muscles. If the patient complains of severe pain or is very apprehensive, it is better to postpone muscle testing for a day or two, bearing in mind that the earliest possible estimation of the paralysis is advisable.

At least 30% of all cases fail to develop detectable paralysis or develop a temporary paralysis which disappears in seven to fourteen days. In these cases, no specific treatment is indicated as recovery depends upon spontaneous return of the function of the anterior horn cells which have not been destroyed. The effects of pain and muscle spasm should be mitigated, but interference must be minimal whilst the infection is active.

Nuchal rigidity and stiffness of the back usually disappear spontaneously with the departure of the meningeal irritation. Lumbar puncture should be repeated only when symptoms of increased cerebral pressure are manifest, and can then be repeated daily until the pressure is normal.

All cases, especially those with spinal and abdominal muscle paralysis, should be nursed on a firm mattress which

can be made more rigid by the use of fracture boards, but a plaster bed or Bradford frame and restraining harness may be required for a restless child. The maintenance of the correct posture is essential, whether the patient is lying in the lateral, prone or supine position, and this is especially important during sleep.

When palatal paralysis only is present, the patient should be given semi-solid or soft foods, but if the muscles of deglutition are involved, he is unable to swallow saliva, mucus or vomitus. There is then a mechanical blockage of the upper respiratory passages and usually small regions of atelectasis occur. In these cases, the mechanical respirator is contra-indicated, unless a previous tracheotomy has been performed, otherwise the contents of the throat may be sucked into the lungs producing an aspiration pneumonia. The correct treatment is postural drainage as advised by Brahdy and Jenarsky (1934) and Stimson (1940). The patient lying in the prone position may be tipped 20° to 30° from the horizontal and a suction apparatus used with care and skill is useful in removing the mucus and saliva. Certain observers advise intranasal feeding with milk, glucose water, eggs, etc., but it must be used with the greatest caution otherwise collapse and death may follow the feeds. I prefer to give 5-10% intravenous glucose solution in the beginning, and if the patient vomits, gastric suction should be commenced.

Atelectasis of the lungs should be treated by immediate bronchoscopy, and aspiration by suction as advocated by Morrow and Stimson (1947).

Before there is any evidence of blockage, postural drainage and assisted coughing should be given. Any asymmetrical movement or deficient air entry should be noted. This is most important as it is well-known that the presence of air beyond the block greatly aids the assisted coughing and helps to dislodge the

mucus plug. The position of the patient also aids drainage towards the tracheal bifurcation, but the foot of the bed should not be raised more than 20°. If it is elevated more than this, we may get even greater distress owing to the pressure of the abdominal viscera on the weakened diaphragm.

Assisted coughing is the name given to sudden firm bimanual pressure on the chest which is synchronous with the patient's feeble coughing. It is kept up for twenty minutes at a time, and is repeated four times a day.

The main indications for tracheotomy are:

1. Irregular shallow breathing and/or periodically apnoeic breathing.
2. Agitation, extreme restlessness and apprehension preventing pharyngeal exploration.
3. Progression of bulbar involvement with increasing dyspnoea.
4. Presence of congestion and recurrent cyanosis.
5. Coarse râles in the chest and laryngeal stridor.
6. Stupor or exhaustion in which the patient is apparently unaware of the accumulation of secretions in the pharynx.
7. Inability to cough effectively.
8. Paralysis of the vocal cords (Bilateral abductor paralysis)
9. Intralaryngeal hyperaesthesia which has been demonstrated by laryngoscopy.

In spite of all the foregoing indications, it must be pointed out that no definite rule can be given as to when a tracheotomy should or should not be performed. All the clinical features in each particular case should be taken into account before arriving at a definite decision. It should however be performed preferably before cyanosis occurs, because if this sign is waited for, irreversible damage may have been done to an already virus-damaged central nervous system.

Nursing.

Once the physican has decided that hospital treatment is necessary, the patient should be transported in the recumbent position in an ambulance. A car should not be used, unless the patient can lie flat on a board with is adequately padded with blankets.

The nursing staff who are entrusted with the care of these poliomyelitis cases must be highly trained, because apart from the general nursing care of the patient, they will require to take all necessary precautions to prevent the spread of the infection. In addition they should be familiar with the deformities which may arise even in the earliest stages and with the splints and appliances which are employed to prevent or correct them.

Isolation of the patient, especially from children and young adolescents is necessary for three weeks after the temperature has become normal. This is usually carried out in an isolation hospital by the pavilion or cubicle system, but unfortunately cubicles and pavilions are still relatively rare in Great Britain. Admission to an isolation hospital is preferable in the first place because the patient may be suffering from some other infectious condition, and if no paralysis develops, it is more economical in beds from the orthopaedic point of view. Another important point in favour of the isolation hospital is that the members of the staff are adequately trained in the intricacies of barrier nursing.

It is however essential that all proved cases should have adequate orthopaedic supervision from the onset of the disease.

It is quite usual for the patient to be nursed in the general hospital ward by barrier nursing or preferably in a side ward, as quietness is an important part of the treatment. In the former case, a corner bed next to a window is to be preferred, as efficient ventilation helps to minimize risk of spread of the disease. The bed should be screened and the floor space

within the screens should be at least 12 feet by 10 feet. The furniture and walls should be wet-dusted and the floors and blankets treated with spindle oil.

Gowns should always be worn by the nursing and medical staff to prevent contamination of the clothes or uniform, and they should hang beside the isolated bed. To put on the gown, it should be removed from its hook by the loop and held by the neck. One arm after the other is then placed in the armholes and the gown fastened. The gowned nurse should then leave the bedside only to deal with used sanitary utensils, being careful not to touch ward furniture and doors whilst so doing. The gown should eventually be removed before washing the hands, and for removal it should be held by the outer part of the sleeves and drawn off. This care prevents contamination of the interior surface which hangs outermost when it is returned to its hook.

Facilities for washing the hands should be provided as this should be carried out after every nursing care and treatment. The majority of articles such as feeding and sanitary utensils can be in common use by the other ward patients provided that they are efficiently sterilized, disinfected or boiled after use.

Personal and bed laundry should be disinfected, either by steam or by soaking in carbolic lotion (1 in 40) for a few hours.

If visitors are permitted, they must wear a gown and mask, and are warned not to touch the patient or his bed.

Mental rest and quiet are most important, and the room should not be brightly lit.

The patient should be nursed flat in bed, the mattress being on a wooden frame or supported by fracture boards. Although maximum physical rest is essential in the acute stage, the position of the patient must be changed fairly frequently with extreme care and gentleness to prevent passive congestion in the dependent parts. Bedsocks should be worn to keep the limbs warm, as this maintains a good circulation and helps to relieve the

pain and tenderness. The skin must be kept clean and dry and precautions taken to prevent bedsores by means of air-rings, pads, etc., A bedbath is given night and morning and the bedclothes are kept free from wrinkles and crumbs.

The limbs must be handled very gently and the joints supported when the patient is moved or when splints are removed.

In extensively paralysed patients, retention of urine with distension of the bladder should be watched for. In the early stages, retention may be relieved by raising the head of the bed on blocks and by the application of heat to the abdomen. Next, if there iis no response to antispasmodic drugs, e.g. carbachol, catheterization, tidal drainage or suprapubic drainage may be required. If there is incontinence, great care must be taken to prevent bedsores.

Constipation may be troublesome but laxatives and cathartics should not be given. Repeated retention enemata of two to four ounces of warm olive oil and mineral oils, e.g. nugol by mouth are advised. Later when laxatives are permitted, compound liquorice powder can be given. Incontinence of faeces usually occurs only in very severe cases.

Whilst using the bedpan, the body should be kept in a straight line with the joints supported in the optimum position, but any splints in use should not be removed. The patient is turned on to his side and the bedpan placed in position. Folded blankets are placed above and below it, to support the trunk and lower limbs respectively. The patient is then turned gently on to the bedpan. Special beds or split mattresses assist greatly in its easy use.

To turn the patient on to his side, firm pillows or a folded blanket are placed alongside the body. The nurse supports the leg at the ankle and just above the knee, whilst an assistant supports the buttocks and shoulders.

As the patient is turned gently, the leg and arm rest on the pillows, the arm being supported at the shoulder level and the upper leg is brought slightly forward from the hip to avoid pressure on the underlying one. The pillow can later be made higher if there is much pain on moving the arm, thus preventing it from coming too far forward. If the abdominal muscles are weak and the abdominal wall sags, a firm supporting pad is indicated as this invariably gives added comfort.

To turn a child on to his face, he is moved en bloc, the pelvic and shoulder girdles moving together. The draw sheet, which has previously been placed in the long axis of the body is then drawn to the foot of the bed so that the feet project through the bars at a right angle to the lower limbs. When in the prone position, one or two pillows should be placed under the chest and thighs so that the abdominal muscles are free to carry out the respiratory effort. Pillows should also be placed under the knees to protect them from pressure.

If the child is restless, he should be nursed on a straight Bradford frame or on a plaster bed. If on the former, he is turned on to his face with his feet over the edge of the frame which should be high enough to prevent the pressure of the bed on his toes. If on the latter, the patient can wear the anterior cast in the prone position and the posterior cast in the supine position.

The nurse is responsible for ensuring that the position ordered by the physician is maintained so that stretching of the paralysed muscles is prevented. The position is maintained by sandbags, pillows or splints and a bed-cradle is used to bear the weight of the bedclothes. Once again it must be stressed that rigid immobilization during the acute stage is never called for as this invariably causes stiff joints and contractures.

The nurse must know how and why any apparatus is used, and a brief description of the positions to be maintained either

voluntarily or by splinting is now given. A full description however is given in the appropriate section:

1. Head and neck kept straight.
2. Back straight.
3. Shoulder is abducted to approximately 60°.
4. Elbows are flexed to a right angle.
5. Wrists are slightly dorsiflexed with the fingers flexed.
6. Thumbs are splinted in opposition.
7. Legs are kept straight with no medial or lateral deviation.
8. Knees are slightly flexed.
9. Foot is kept at right angles to the lower limb.

(It should be noted that these positions differ in certain respects from those advocated by Sister Kenny.)

This correct body and limb position must be maintained even in abortive and preparalytic cases. A careful watch must be kept to avoid pressure on the paralysed muscles by tight bandages and straps or by the splints and appliances themselves.

The patient should be encouraged to take plenty of fluids, especially during the febrile stage and the mouth is cleaned regularly. After the temperature becomes normal, a more varied nutritious diet, rich in vitamins is given.

Some observers advise the use of ice-bags applied to the back of the neck and spine and certain drugs to reduce the fever.

Pain may be relieved by the use of warm packs for five to fifteen minutes or by the use of an infra-red lamp. Neuritic and neuralgic pains are usually relieved by sodium salycilate in doses of from ten to sixty grains. Sedatives may be given for restlessness but only in very small doses, because of the danger of respiratory paralysis during sleep.

A careful watch must be kept for signs of respiratory paralysis, the chief of which are restlessness, anxiety, insomnia, increased pulse rate, sweating, cyanosis, dilatation of the alae nasae and difficulty in speech.

Even in the mildest case of poliomyelitis, the patient should rest in bed for at least three weeks.

In the preparalytic stage, when there are signs of meningeal irritation, lumbar puncture is essential to exclude meningitis, encephalitis etc., and preparations for this precedure must be made by the nurse.

In the preparalytic stage when the diagnosis is certain, lumbar puncture may be harmful and is unjustifiable unless there is clinical evidence of increased intercranial pressure.

If there are no contra-indications, the bed may be raised at the end of three weeks, as this allows the patient to feed himself and to take an interest in what is going on around him. It also helps to increase his respiratory excursion. In these cases, raising the head of the bed has definite advantages over a back-rest.

A portable reading lamp is of great benefit, and in certain cases mirrors and prism spectacles can be used to permit even severely paralysed patients to read whilst remaining supine. Even patients with complete paralysis of both upper limbs can enjoy reading a book by the use of an electric "page-turner."

The position of the bed-side locker should be changed at intervals to prevent constant turning towards the same side, as this may contribute to torticollis or scoliosis if there is weakness of the neck, abdominal or back muscles.

Nursing treatment of cases with respiratory paralysis.

The proper selection of patients for treatment in a mechanical respirator is essential not only from the point of view of the correct treatment, but also to economise in the use of respirators as during an epidemic the demand may sometimes exceed the available supply.

It is interesting to note that the first bulbar case treated by Drinker in 1929 died. Brahdy and Lenarsky in 1936 reported on 63 cases treated in the Drinker respirator, and pointed out that all these cases with involvement of the

respiratory centre had died.

Nielson (1946) made observations on 110 cases with respiratory insufficiency of whom 76 were treated in the Sanlin (Swedish) respirator. The mortality was found to be greatest in those patients with signs of bulbar lesions. At postmortem, there was emphysema of the upper part and atelectasis of the lower parts of the lungs, but only in the patients who were treated in the respirator.

It can therefore be concluded that the bulbar type of case should not be treated in a respirator if the respiratory muscles and the mechanics of respiration are still normal.

It has been suggested by Nielson (1946) that treatment in the respirator leads to the accumulation of carbon dioxide in the blood, and that the resultant acidosis contributes further to the fatal issue.

1. Respiratory embarrassment due to bulbar paralysis.

The patient is in danger of dying from involvement of the respiratory or cardiac centres or from choking due to an inability to swallow. Aspiration of mucus and vomitus into the bronchi may cause aspiration pneumonia or a fatal bronchial obstruction, and it is therefore important to keep the pharynx free from secretions, food and vomitus.

As excitement increases the flow of mucus, the patient should be kept as quiet as possible. Nausea associated with vomiting also causes increased secretions, but vomiting can be prevented by keeping the stomach empty. Fluids and carbohydrates are therefore given per rectum or intravenously in preference to nasal tube feeding.

As severe attacks of coughing and choking weaken the patient, postural drainage should be tried, by raising the foot of the bed 30°. Drainage may also be aided by turning the patient on to his face and by using a suction tube or an electrically driven suction apparatus. Because of the risk of trauma the latter should not be used more often than is necessary to keep the

patient relatively comfortable.

In extreme cases, tracheotomy may be necessary and it is then the duty of the nurse to see that a free air-way is maintained.

If there is a failure of the respiratory centre, intravenous saline with 25% glucose every four hours for twenty four hours and oxygen therapy have been recommended and the nurse must have the facilities for these constantly available. Oxygen therapy by means of a nasal catheter or a B.I.B. mask can be administered in an emergency but an oxygen tent is unsuitable. Lumbar or cisternal puncture may also be ordered to relieve the cerebral tension.

Different features of bulbar paralysis and spinal intercostal paralysis.

Features	Bulbar	Spinal-intercostal
Pathology	Nerve cells of respiratory centre in medulla	Nerve cells of anterior horn of cervical part of spinal cord.
Respiratory muscles	No paralysis	Intercostals and/or diaphragm paralysed.
Concomitant paralysis.	Usually cerebral motor nerves and their muscles esp. those of pharynx and palate.	Usually muscles of shoulder girdle.
Type of respiration.	Irregular in rate and depth.	Rapid and shallow.
Speech.	Nasal tone.	Short sentences.
Dysphagia.	Collection of mucus in pharynx.	Absent.
Mental state.	Disoriented, often stuperous but may be comatose.	Apprehensive and anxious, rarely disoriented.
Auxiliary muscles of respiration.	Not improved and often made worse by the respirator.	Immediate dramatic improvement.

2. Respiratory embarrassment due to paralysis of the respiratory muscles. (Intercostals and diaphragm).

These spinal cases are ideal for treatment in the mechanical respirator but it can also be used in cases where the respiratory distress is due to persistent spasm of the muscles of respiration, when the thorax will be found to be held in the position of expiration.

It is imperative that the late signs of respiratory embarrassment should not be waited for, before placing the patient in the respirator. The nurse should be warned to look for, record and report the slightest and therefore the earliest sign of impaired respiratory function, because they may last in the inital stage for only a very short time and may be followed by a period of apparent abnormality.

The early use of the respirator helps to prevent the exhaustion of the weakened muscles, ventilates the lungs adequately, controls the acidosis and avoids a fatal result from anoxaemia.

There appears to be very little doubt that anoxaemia has a marked deleterious effect and it may even induce a more rapid spread of the virus. The earliest signs of anoxaemia which must be looked for are anxiety, sweating, increased restlessness cyanosis and an increase in the pulse rate. There is also a quickening rate of respiration, loss of strength to cough, visible feebleness of chest and diaphragmatic movements and an inability to say one or two words with each breath. (ask the patient to count.) The patient is restless, irritable, fatigued and apprehensive. The accessory muscles of respiration, e.g. the sternomastoids and alae nasae are very active.

A mechanical respirator should always be "on hand" in a hospital and it should be instantly available for any case with bilateral involvement of the shoulder muscles, cervical muscles and upper extremities.

In the case of young children, the respirator should in

appropriate cases be used at the earliest possible moment, because if they survive the crisis there is every possibility of recovery. Unfortunately this is by no means true for adults, for whom the prognosis is very much worse.

Patients may be treated in various types of respirator, e.g. the Both cabinet, the Sanlin, Drinker or a special orthopaedic type with a cabinet large enough to permit the use of splints for the support of the arms and legs and so prevent contractures and deformities.

The importance of saving the life of the patient must not entirely obscure the necessity for protecting him from needless disability.

Certain observers prefer the Bragg-Paul type of pulsator in the late convalescent and residual stages, as the patient is then free to move about and also it is readily transportable.

These respirators are however a mechanical means of preventing death from asphyxiation, and the type used is really of little importance. They may require to be used for the duration of the patient's life.

The Both cabinet respirator.

As this is the usual type in use in this country, a brief description of the nursing technique will be given, as it is essential that the nurse should be entirely familiar with its mechanism.

Inspiration and expiration are both controlled in a box type of respirator and the action is so powerful that it can overcome quite marked degrees of muscle spasm.

The machine is tested before use and the speed of the artificial respirations regulated at 20, 26 or 32 pulsations per minute, according to the requirements of the patient. In our opinion, respiratory rates higher than 20 per minute are too high and are likely to produce a "washing out" of carbon dioxide and a condition of acapnia. We therefore

advise the maintenance of a lower rate of respiration than that which is usually recommended and the administration of oxygen to overcome any anoxia. In an emergency, such as the failure of the electric current or a break-down of the electric motor, the bellows can be hand-operated at the rate of 20 strokes per minute. The mattress and small pillow are prepared and a suitable rubber collar chosen. To insert the patient, the bed is pulled out, and the patient is placed on it his head being near the oval opening in the end cover. His head is now put through this opening, and he is pulled in a horizontal direction until his shoulders come against the cover. The special sponge rubber neckpiece is then fitted comfortably and not too tightly over a bandage previously placed around the patient's neck to prevent chaffing by the collar. The neck may be protected by lanoline or vaseline spread on lint and covered with gamgee tissue. The bed is then re-inserted into the cabinet and the end cover is bolted into position.

Before starting the motor, the nurse should make sure that the air passages of the patient are free from mucus, by aspiration if this is necessary. The motor is then started and a negative pressure of 12 to 18 ccm. of water is maintained by adjusting the valve on the top of the cabinet. This pressure gives an adequate ventilation of the lungs in a patient who has a complete paralysis of the respiratory muscles. In young children, the negative pressure should be between 10 an 14 ccm. of water.

Air leakage around the collar can be prevented by using cotton wool packing.

If so desired, the cabinet can be tilted with the head downwards to approximately 20° from the horizontal, the patient's shoulders being protected with pads of cotton wool. If the diaphragm is weak, pressure of the mattress in the prone position increases the difficulty in breathing,

The temperature inside the cabinet should be kept just above

the normal room temperature, i.e. 75°F.

As too much clothing obscures the movement of breathing, which must be under constant supervision, the minimum amount should be worn. An open-backed gown is advisable because of the ease with which it can be removed. Extra warmth can be supplied by bedsocks, well-protected hot water bottles, or by a lamp switched on inside the respirator.

Basic nursing care is essentially the same as for all patients, but a two-hourly schedule for changing the position of the patient should be organised. The number of assistants required for this depends on the position and the weight of the patient. Special care of the skin is necessary to prevent bedsores and excoriation around the neck by the rubber collar. The use of a rubber bedpan is most advantageous.

If the patient is of an age to understand, he should always be told that he is going to be nursed in a respirator and should be assured that it is only being used temporarily to give the respiratory muscles a rest. At the beginning, the patient may be apprehensive until his breathing becomes synchronous with that of the respirator, and when this is established, he usually falls into a peaceful and restful sleep. Synchronism may be established by increasing temporarily the negative pressure or by increasing the rate of the pulsations. If breathing is not synchronous with the respirator after five minutes, involvement of the respiratory centre must be suspected. If there is spasm of the chest muscles and diaphragm, hot packs may be applied to the thorax through the portholes. It is important to remember that the pressure should be increased to 25 to 30 ccm. of water for five minutes several times a day so that the lungs may be fully ventilated.

During the acute stage, it should be possible to carry out all nursing treatment without stopping the machine. The opening of a porthole should immediately follow an inspiration but the porthole should be closed as soon as possible. Certain

observers advise the administration of pure oxygen by mask for five minutes before the portholes are opened and that it should be continued until they are closed. If the cabinet is open for any length of time such as for changing of the bedclothes, accessory artificial respiration can be given by the McKesson resuscitator or by a Boyle's anaesthetic apparatus. In the latter case, the lungs are inflated by the rhythmic compression of the rubber bag whilst the face-mask is closely and firmly applied.

Eve's tilting stretcher or manual methods may sometimes be used in relatively mild cases whilst the patient is having orthopaedic treatment of short duration out of his respirator, but they should not be continued for ~~for~~ long periods as they are very distressing to the patient.

The respirator may be used continuously for days, weeks or months, thereby protecting the respiratory muscles from undue or cumulative fatigue. In this way, recovery may be more rapid and complete especially in cases where there is only a mild or moderate weakness of the respiratory muscles.

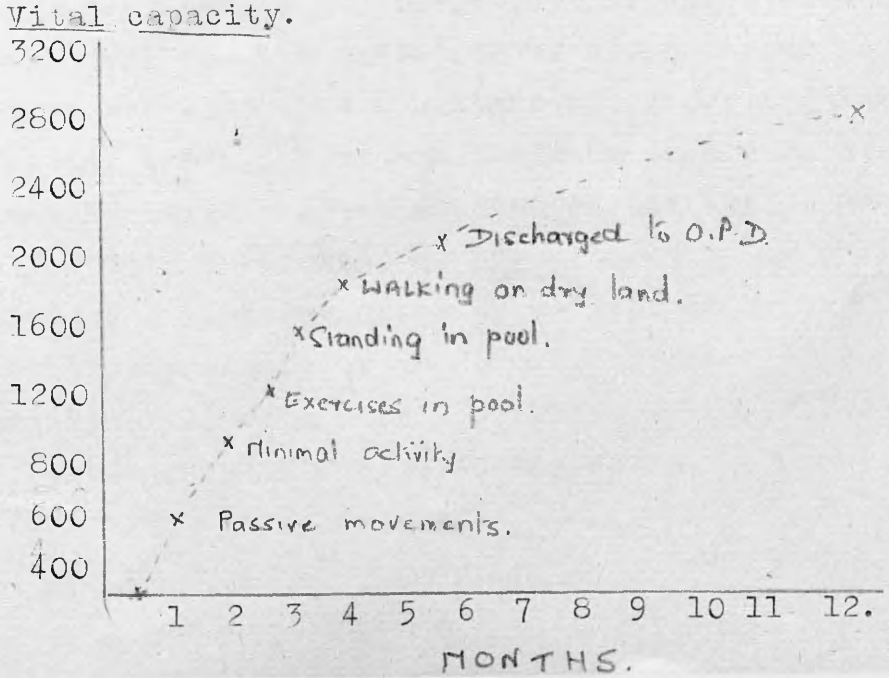
The mechanical respirator may also be employed as a therapeutic measure in order to rest the weakened muscles of respiration and to re-educate them towards normal power and balance. This may be achieved by putting the patient into the respirator for several hours a day or he may be allowed to sleep in it. One of our patients who had extensive paralysis of the whole trunk and limbs was nursed in an iron lung even during sleep. It was noted that respiration was carried on only by the accessory respiratory muscles. The vital capacity whilst lying was only 350 ccs. and when sitting 400 ccs.

Cases should also be treated in a respirator who show progressive weakening of the intercostal muscles, increasing respiratory embarrassment or fatigue of the overworked healthy muscles.

The respirator can also be used to treat insomnia which is due to dyspnoea.

The mechanical respirator must be discontinued gradually and this process may extend over several months. The tendency has been to remove the patient far too soon rather than too late. The effects of this early removal are cumulative over days or weeks and the premonitory signs and symptoms are headache, irritability, insomnia, anorexia and mild dyspnoea. These signs increase until an emergency arises which may result in the patient's death or prolong his retention in the respirator for several weeks. The correct method is to open the porthole for varying periods and to gradually increase this time during sleep. During the intervals when the patient is breathing without the aid of the respirator, a careful watch must always be kept for the onset of sudden dyspnoea, which will necessitate the immediate restarting of the respirator or the return of the patient to it.

Weekly estimations of the vital capacity should be carried out. This is an individual assessment and other things being equal, the removal of the patient from the respirator can usually commence when the vital capacity has reached 750-1000ccs



It will therefore be seen that the mechanical respirator tides the patient over a critical period and may keep him alive until the respiratory muscles recover.

In rare cases, with abductor laryngeal paralysis; tracheotomy and/or oxygen therapy may be required in patients who are being treated in a respirator.

One last point which is sometimes forgotten is that the respirator must be adequately serviced and there must always be an adequate and immediate supply of spare parts.

The Stanco respirator.

In this country the vast majority of hospitals dealing with cases of poliomyelitis are equipped with cabinet respirators of the Both type. In recent years, Stanley Cox Ltd have perfected the very latest type of respirator - the Stanco, which incorporates many new features. The whole apparatus, in cream enamel and chromium plate has been carefully finished to eliminate as far as possible the "coffin" appearance which undoubtedly had an adverse psychological effect upon most patients. Every consideration has been given for the comfort of the patient, including a very comfortable Dunlopillo mattress, four strip lights which provide both light and adequate heat, and a large port provided with quick action fasteners for the insertion of bedpan or urine bottle. There are also five miniature ports for the introduction of feeding tubes,, four arm ports on each side of the cabinet, and two large perspex windows on the top of the cabinet to allow full inspection of the patient. The pulsator unit can produce a negative pressure as high as 35 cms. of water whilst positive pressure up to 10 cms, of water is controlled by a separate valve. A simply operated device warns the attendant of pressure failure due to any cause.

Cuirass respirator.

This is not so certainly effective that it can be depended upon for the treatment of the initial stage of acute respiratory muscle paralysis when paralysis may progress quickly, but it may be used as an adjunct to the tank machine. It is useful during the process of weaning a patient from the respirator, for the chronic care of moderately paralysed patients and when a tracheotomy is necessary.

The Bragg-Paul pulsator.

This apparatus can be used even for very young children, as different sizes of air-belts are supplied. Air is forced into the chest belt at definite rhythmical pressures by electrically driven bellows. Each pulsation produces an involuntary expiration, and the release of the pressure allows the natural elasticity and recoil of the chest and diaphragm with a resulting inspiration.

One disadvantage of the Bragg-Paul pulsator is that it assists only during the passive phase. Movement of the tender and painful intercostal muscles by the pulsator may cause them to pass into spasm. Reflexly this spasm causes a marked variation in the respiratory rate, but the apparatus cannot control the constantly changing respiratory rhythm. The pulsator works at a rate of approximately 14 to 16 pulsations per minute and it can also be worked by hand. It is useful in cases with a very low vital capacity and the patient can sleep in it. Positive pressure oxygen therapy may be required if there is any suspicion of pulmonary oedema.

Blockage of the bronchi and collapse of the lung may result from bronchial catarrh. This is best treated by penicillin, postural drainage, gentle manipulations and frequent change of position. Atropine sulphate and sedatives are definitely contra-indicated.

Convalescent stage.

During the convalescent stage, the patient must receive an adequate amount of fresh air and sleep, and mental and physical fatigue must be avoided. The skin and muscles are kept warm and the correct posture maintained by the use of any necessary appliances.

The patient is given general tonics and an easily-digested nourishing diet.

The early symptoms of anoxaemia in a patient who has been in a mechanical respirator for several weeks or months are insomnia and headache and they should be treated by the administration of oxygen.

Bruce (1941) drew attention to the fact that vitamin B is supposed to increase the resistance of the nerve cells but its true value in this respect is still not definitely proved. Vitamin B does however improve the carbohydrate metabolism in the nerve cells and will therefore produce a more satisfactory oxygenation of pyruvic acid. Vitamin E is also given as it is said to improve the muscle tone, decrease capillary permeability and counteract the development of fibrosis.

Prolonged recumbency in a patient may result in renal calculi which may cause death from renal insufficiency. This is treated prophylactically by:

1. Regular and frequent changing of the position of the patient.
2. The administration of abundant fluids.
3. The reduction of calcium and Vitamin D in the diet.
4. The prevention and control of urinary infection.

As the patient often suffers from a prolonged illness, he may become irritable and apprehensive. The nurse must therefore show patience, understanding, sympathy, tact and great gentleness, as this helps to dispel fear and establish confidence.

On discharge of the patient, the nurse should make sure that both he and his relatives understand fully the fundamentals of nursing care required in his particular case, and that instruction has been given in the application and care of appliances etc.

Muscle re-education.

I propose to give only a brief description of muscle re-education, because this form of treatment should preferably be given by a physiotherapist who has been specially trained in the treatment of poliomyelitis.

It must first of all be pointed out that it is a mistake to imagine that the patient must keep the limbs active to counteract a spreading of the paresis. The patient should be left entirely alone until the maximum paralysis has developed and this usually takes three days. The only treatment necessary during this waiting period is to apply the appropriate splints to prevent any tendencies to deformity.

In performing a voluntary movement, all the intact muscle fibres do not necessarily contract, because the muscle can adapt itself to the amount of work it has to perform, and the more vigorous the exercise, the greater will be the number of fibres involved. When the function of a motor nerve is completely or partly destroyed, attempts are still made to produce the movement by sending impulses to the region along other intact nerves.

The main aim of muscle re-education is to produce hypertrophy of the remaining muscle fibres in an attempt to compensate for those which have lost their nerve supply. The treatment should also be directed towards maintaining good alignment both in weight bearing and in non-weight bearing. Controlled muscle activity and exercises to the unaffected part of the body are also given to maintain muscle tone and to prevent disuse atrophy, and they should be given as soon as muscle tenderness disappears.

There is no doubt that physiotherapy treatment should begin as soon as possible, because delay may be partly responsible for the development of contractures and an unnecessarily long prolongation of the convalescent period.

It is essential that right from the beginning and throughout treatment that the physiotherapist should have the full co-operation and confidence of the patient, and keep an accurate record on the patient's chart.

Whatever method of treatment is used, recovery will be extremely rapid if there is only a transient weakness of the muscle group.

Forrester-Brown (1938) stated that muscle re-education should begin immediately a "flicker" movement appears, and that the first consideration is passive and guided exercises

Technique of treatment.

No definite rule can be followed in connection with muscle training of a poliomyelitis patient.

The aim of treatment is to re-educate voluntary contraction in the affected muscle by individual and co-ordinated action, and to encourage activity by increasing the frequency of an exercise or making the muscle task harder.

The decision as to whether to increase the task of the muscle depends upon its ability to move the joint smoothly through its full range. It is therefore wiser to increase the frequency of the exercise until full range has been achieved. The last movement should be as well performed as the first.

It is essential to have the part under training stripped for observation. Fatigue must be avoided and it is more noticeable in a lessening of the muscle effort. Warming the tissues prior to performance is indespensable and a warm pool or radiant heat are both effective.

It must be assumed that no muscle is completely denervated, and therefore muscles which are apparently paralysed must be given training equally with recovering muscles.

A muscle which has had adequate treatment for a period of six months and which still remains paralysed, will not subsequantly regain any recovery of functional significance with the passage of time. All muscles except those of no apparent power should receive whatever progressive training is necessary to achieve maximum strength and it may be necessary to continue for as long as eighteen months or two years after the onset.

Training of a muscle of no apparent power.

The part to be exercised must be supported, carrying out the movement passively whilst the patient concentrates on the attempted movement, thus the impulses of movement are awakened, and the asistance given by the physiotherapist must synchronise with the patient's physical and mental effort.

To initiate reflex tension in the muscle, gentle stretching should be given from time to time. Passive movements through the full range should be repeated several times during treatment and in the majority of cases a contraction is seen after two to three weeks. The patient must be taught to watch the contraction, learning the feel of tension, and his full attention must be concentrated on the anticipated movement. The physiotherapist must assist the movement aiming to obtain the patient's maximum concentration in the middle range by lessening assistance at this stage. As muscle power returns, co-ourdinated exercises must be introduced but resistance must not be given until the muscle is able to perform the movement smoothly through its full arc. This should be taken as the criterion for further progressive resistance.

The judicious use of varied technique helps to avoid boredom for the patient during many weeks of treatment.

1. Manual control in the form of assistance or resistance is conveniently used in the early stages of muscle re-education.

The control can be graduated to correspond with the returning muscle power, each movement being undertaken with adequate support of the part to avoid joint strain. The change in leverage which takes place during movement is a guide to the amount of resistance to be given. The middle range is easier to achieve than the inner or outer range, therefore the resistance should be lightly applied at the beginning and at the end of the arc of movement and more strongly applied in the middle range. The resistance given should be slightly less than that which would stop movement.

The disadvantage of manual control is that both hands are occupied in supporting the limb so that the operator cannot be certain that the required muscles are contracting.

2. The re-education board.

This technique requires a large ply-wood board about three feet six inches square with an even-polished surface. This board is placed under the part to be exercised so that the limb rests on it, support is adequate and friction is greatly reduced by the polished surface. The physiotherapist guides the limb with one hand, steadying the fulcrum with the other.

For re-education of the muscles of the lower limb in adults, trick movements are difficult to prevent. For the upper limb, the board can be used for the re-education of the deltoid muscle. The board is placed in the nature of a back-rest, the shoulder is fixed with one hand whilst the other guides the arm in abduction. A short or long leverage can be made use of by either fixing the elbow, or the arm can be more fully extended. The re-education board can be placed on an inclined plane where gravity may assist the movement which takes place in a "downhill" direction, or gravity may resist the arc of movement which takes place in an "uphill" direction.

3. Suspension therapy.

The apparatus required is simple, cheap and can be readily erected in a limited space. Care is required in the selection

of suspension apparatus. A strong supporting frame should be of adequate height with slings and ropes of suitable strength.

The advantage of suspension therapy is that it leaves the operator's hands free, one hand being used to control the range of movement, whilst the other is free to palpate the tendon of the acting muscle. Assistance or resistance to the movement can be given manually by the operator. Friction is eliminated and gravity is counter-balanced, except as is explained later by changing the fixation point of suspension. Gravity can assist or resist muscle action. Trick action can more easily be detected and prevented. Isolated muscle groups can be re-educated whilst neighbouring muscles remain relaxed. The psychological effect on a comfortably suspended patient is marked; once he is aware of the rhythm of motion, he can pick up and follow movement more easily, thus encouraging greater effort. To obtain an accurate analysis of the power of trunk muscles, partial suspension can be used with advantage. One half of the body is lowered to rest on a fixed point whilst the other half is raised in suspension. Partial suspension can also be used for strengthening the weak trunk muscles.

The principles of suspension therapy.

There are three different types of suspension.

1. Axial suspension. The limb is supported in slings, the ropes of which are fixed at a point above the joint which is to be moved. The limb can therefore move in one plane from one axis in perfect balance. Axial suspension allows the fullest range of joint movement.

2. Pendular suspension. For this type, the axis of movement is moved to a point away from the joint. Friction is therefore not vertical, but the plane is truly horizontal.

The advantage of this type of suspension is that a particular muscle group can be activated whilst its antagonist muscles remain relaxed. By altering the fixation point, it is possible to obtain either assistance or resistance by gravity for the acting muscles, and the suspended limb will swing back to the fixed point. Only a half of the arc of the swing is used to give either uphill or downhill effect, and the limb then swings back in relaxation to the fixed point. Pendular suspension can be of great assistance in the early stage of muscle re-education. The patient can watch the movement and can be taught to synchronise thought and effort whilst the operator initiates a gentle pendular swing.

3. Spring suspension. This allows movement in multiple axes and planes and may be used to assist movement by making use of the recoil action and timing the recoil exactly with muscle effort.

The patient is suspended in the prone or supine position and can be taught co-ordinated exercises such as swimming and natural movements which are similar in character.

Co-ordination may be taught by rhythmical exercises to music. This is especially important in children who can associate certain movements with the music they hear.

In partial paralysis of the diaphragm and intercostal muscles, breathing exercises should be given daily and the vital capacity recorded every two weeks. Poor breathing habits cause bad posture, sleeplessness and dullness, and therefore patients should be taught to breathe deeply and rhythmically by the physiotherapist.

It must also be noted that alterations in protective supports are needed fairly regularly in children and adults to meet any changes in the muscle balance, but appliances should never be discarded without the permission of the orthopaedic surgeon.

Physical treatment may continue for two to three years, and during this period, it must be constantly supervised and adapted as the muscle power is regained.

Weak muscles begin to increase in strength within two weeks of the onset of the paralytic stage. Recovery is most striking during the first twelve weeks but gradually slows down, and after approximately two years of supervised treatment, it is doubtful if further recovery is possible. It should however be pointed out that a patient who has not had supervised treatmen -t from the beginning of his illness may show an increased rate and degree of improvement subsequent to the substitution of these exercises, and cases have been recorded where this has occurred after a period of as long as ten years. On the other hand, a limb which has recovered imperfectly from an old poliomyelitis paralysis may show an increased weakness after several years. This is probably due to the excessive use of the weakened muscle, and it is more commonly noticed in the arm than in the leg.

Permanently and completely paralysed muscles should be recognised as soon as possible. If there is no trace of contraction after three months, it will never recover any useful function.

Thermotherapy.

The metabolic rate of the tissues is increased locally and directly by heat. The effect of the increased temperature on the limb is to warm both the nerves and the muscles. As a result of this:

a. The threshold of the nerve, the neuro-muscular reaction to stimuli is lowered and also the muscle threshold itself is lowered.

b. The duration of the muscle contraction is shortened.

c. The refractory period of the muscle is shortened.

Although the exact mechanism is still not definitely known, there is no doubt that thermotherapy has the following beneficial effects:

1. It relieves pain and discomfort which are often considerable in the early stages.
2. It improves the circulation.
3. It stimulates metabolism.
4. It improves the efficiency of the muscles.

I have found that a Hubbard tank in which the water is kept at 100°F is most useful in the sensitive stage. The patients are able to relax and so obtain great comfort.

My experience has shown that carefully administered local heat in the form of short wave diathermy is particularly valuable for muscle tenderness and stiff joints, when it precedes other physiotherapy treatment. The mild heating aids in softening contracted tissues so that the range of movement can be improved.

It must be remembered that heat tolerance appears to vary from patient to patient and that the application of too much heat must be guarded against as this causes further weakness, and therefore diminishes the total remaining contractile power of the muscle.

It has already been stated that the efficiency of muscle action is improved by heat and therefore warming the tissues before exercise is indispensable to the performance of maximum muscle effort.

Pool therapy.

This is a form of treatment devised to avoid too early specific muscle re-education by the partial elimination of gravity by the water. It can also be used for general function and balance exercises, swimming exercises, training in walking and in diversional games.

Pool therapy allows great diversity of movement of the

joints, and it is usually carried out in a Hubbard's tank or a treatment pool. The Hubbard's tank is ideally suited for individual cases and for specific muscle training, but in units dealing with a large number of patients, a special pool is necessary. This pool should be of such a height above ground level that the physiotherapist can comfortably and conveniently control any particular movement. She must always be able to reach the patient throughout the treatment and to support the limbs where necessary. In the pool there should be two shallow bays, one twelve inches deep for children, and the other eighteen inches deep for adults. Part of the pool should be of a suitable depth, width and length to allow a free area for walking and swimming exercises and for games. The ideal pool should have two walking lanes each of which should be fitted with a set of parallel bars at a suitable height for children, possibly on a built-up base, and another set at a convenient height for adults.

In some treatment pools in America and in this country, the physiotherapy staff go into the pool with the patient. This is essential if the pool is deep or very large, otherwise the physiotherapist would not have full control of the patient.

Hydrotherapy in this centre takes place in a Hubbard's tank and the patient should not participate in this form of treatment until the temperature has been normal for at least twenty four hours. Also there must be no complicating factors such as pneumonitis, pathological skin conditions or respiratory or cardiac difficulties.

Warm fresh water or hypochlorite solution at a temperature of 98°F is used for the less severe cases but the temperature may be raised to 100°F for spastic cases. If the temperature falls below 98°F the patient may get chilled, and pain and spasm of the affected muscles may be exaggerated quite rapidly. If the temperature is over 100°F the patient may complain of dizziness and vertigo.

Hydrotherapy should be started as soon as possible and usually begins within seven to twenty-one days from the onset of the illness. The warmth and buoyancy of the water is comforting to the patient with muscle tenderness and stiff joints and encourages him to make an effort for himself. Thus the mental relaxation banishes apprehension and inhibitory reactions such as spasm. In short, the whole nervous system is relaxed, the peripheral vessels are dilated, and there is a marked sedative effect on the peripheral sensory nerves.

The patient must be transferred carefully to the Hubbard's tank or treatment pool. He should be placed in the water slowly and should be allowed to rest for a short period before treatment is commenced. This allows him to adjust himself and is particularily important if he appears anxious or if he has respiratory weakness. He should be supported comfortably so that relaxation can be obtained and he may require to retain the splints throughout treatment in cases which have been operated upon, or when the limb is extensively paralysed. In other cases the paralysed parts may be supported by rubber rings or corks, but in the majority of cases it is quite safe to remove all splints and supports during immersion. As friction and gravity are now virtually eliminated, the exercises are mainly effortless and active assisted exercises can be given to all the paralysed muscle groups. As a guide to early treatment, observers suggest that each active assisted movement should be directed three times in the beginning increasing the number of each specific movement according to the condition of the patient, the physiotherapist watching carefully for signs of fatigue.

Patients with flail limbs can be taught balance and walking exercises in a treatment pool by firmly supporting the knee joint with light metal or perspex gutter splints bandaged to the limbs. For balance and walking exercises it is necessary for the water level to reach the costal margin if the patient is to receive adequate support from the water. Buoyancy can be used for the

assisted movements in the early muscle training and later it can be used as a resistance in the progressively stronger exercises. A simple toe-raising sling attached by an elastic strap below the knee joint is adequate for the purpose of supporting a dropped foot.

A careful watch must be kept so as not to over-work the patient and any diminution of power due to fatigue of the affected muscles must be appreciated and treated appropriately. It is therefore wise to limit the period of time in the bath from five to ten minutes in the beginning and later to increase it to a maximum of thirty minutes.

The treatment time in the pool should consist of a part for definitive exercise for specific muscle weaknesses and time should be allowed for co-ordinated exercises.

By increasing the activity of a weakened muscle without strain in a medium of warm water, it is found that its blood supply and strength are increased, disuse atrophy is prevented and the products of metabolism are more freely eliminated. It is also found that the general health improves, the patient sleeps better and his appetite is stimulated. Improved skin action also helps to relieve any atrain on the kidneys.

Hansson (1939) states that patients who are not completely paralysed should be encouraged to learn to swim as it affords both physical and mental relaxation. It is important that it should be of the symmetrical type and that only strokes that are corrective should be used. It is also interesting to note that any patient with normal arm muscles can learn to swim, but in the early stages, life jackets should always be provided. It is most important to stress that in spite of a marked initial paralysis, paralysed muscles constantly relaxed will regenerate when water therapy is carried out even years after the acute stag

From the physiotherapists' point of view, it is important to stress that they are greatly encouraged in their work when they observe that a muscle such as the deltoid can be trained in a very short time to abduct the arm in its full range under water, or the quadriceps to extend the knee fully when neither of these muscles can work against gravity out of water. This shows the great value of a treatment pool for poliomyelitis patients. Sitting up, standing and walking can take place at a very much earlier stage than when attempted on dry land.

It would not be permissable to leave this subject without mentioning certain precautions:

1. The bath must not be too cold at the beginning or allowed to become so during treatment.
2. Great care must be taken to avoid chilling of the skin, which may lead to pneumonia, nephritis etc.,
3. The affected parts and painful joints must be protected whilst the patient is undressed, dried and dressed again.
4. Equally important is the careful handling to and from the ward and the treatment room.

Passive movements.

Passive movements are essential for maintaining mobility of joints and for the prevention of contractures. The importance of early movement cannot be overstressed, but it should not be given until the body temperature has been normal for at least twenty-four hours.

Passive movements usually commence seven to ten days after the onset of the paralysis, and if the pain persists beyond this time, or is very severe, the treatment should be preceded by the application of heat. These movements may be initiated with reasonable safety before the commencement of active muscle re-education. A full range of movement of the joint should then be carried out through the "pain-free" arc once or twice daily. If regular mobilization of the joints is with-held until

pain has subsided, contractures and stiff joints may result.

Areas where contractures are particularily noticeable are the plantar fascia, tendo-achilles, hamstrings, fascial layers in the lumbo-sacral region and the peri-articular structures of the spine. The contractures which are most commonly overlooked are those of the rectus femoris and the ilio-tibial band. They are most difficult to correct and cause the patient unnecessary suffering.

It is most important to recognise the earliest evidence of the beginning of contractures and to initiate mobility by passive and active assistance by stretching to minimize the deformity. Contractures should be evaluated in terms of their deformity potential and then mobilized only enough to obtain a range of movement necessary to initiate muscle re-education of the adjacent musculature. These contractures should be recognised and stretched within three to five months of their onset.

If contractures and stiff joints are already present when the patient first comes under skilled supervision, the shortened tissues should be gently but thoroughly stretched at frequent intervals. Warm baths or irradiation with luminous heat can be of great assistance in softening the contracted tissues before stretching is begun. All movements should be very gentle and the limb must be completely supported throughout the exercises. The limb should be grasped firmly but gently over the joints. The arm is supported at the wrist and elbow, and the leg at the knee and ankle. In this way, pressure on tender muscles is avoided. Gentle passive exercises should be given twice daily. Particular care is taken with joints which have asymmetrical muscle weakness. It is most important to note that the movement must oppose the deformity positions. Constant supervision must be given to patients with a tendency to develop a particular deformity. Certain observers have stated that if an abnormal range of movement is permitted,

of if an extremely paralysed limb is not supported, stretching or weakening of the joint capsules and ligaments will result.

Vigorous irresponsible stretching may result in muscular tears. This leads to the formation of scar tissue and adhesions which further limit movement.

Severely weakened bodily segments may result in instability of these areas, and permit deformities to occur which may be far more disabling than the contractures.

My experience has shown that movements of the inter-phalangeal and metacarpophalangeal joints of the hands and the feet and the full range of movement of the spine tend to be the most difficult to correct once these joints have become stiff. Full extension of the elbow and dorsiflexion of the wrist and ankle are also most important.

It is hardly necessary to mention that it is definitely harmful to retain a muscle in an over-stretched position, which may retard its recovery or make complete recovery impossible.

Passive stretching should be retained throughout early treatment in order to correct any tendency to deformity, and it is advisable to keep a record of the range of passive movements at least until after the full range of joint movement has been obtained.

Active movements.

Recovery of muscle strength can be measured by the use of a simple spring balance and is useful in comparing the relative merits of the different forms of treatment. In this centre I have found that the manual method gave us quite satisfactory results.

1. Assisted movements.

Assisted movements are necessary when a voluntary contraction of any particular muscle or muscle group is charted as 0, 1 or 2, that is to say when the active effort is unable to overcome gravity.

Assisted movements can be obtained in a treatment pool where the buoyancy of the water provides helpful assistance in early movement. Sling suspension in which the limb is supported in slings also eliminates gravity. In some hospitals, a flat powdered board is used to obtain gravity-assisted exercises, the limb to be exercised resting comfortably upon the flat surface.

In the early stages, manual assistance is the best method, as the physiotherapist can immediately and accurately control the amount of assistance given to correspond with the effort of the patient.

The patient is instructed carefully in the movement required, either on his sound limb or by a personal demonstration by the physiotherapist. The movement is then carried out on the paralysed limb, being assisted by gravity or by the physiotherapist. The limb is gently and slowly carried through the motion which represents its function whilst the patient at the same time makes every effort to use the muscles.

In the early convalescent stage, the primary action of the individual muscles or single muscle groups should be the first to be re-educated.

Assisted movements should be continued until the patient can voluntarily and without assistance hold the limb in the desired position by full contraction of the affected muscles.

2. Free movements.

In this form of treatment, no assistance is given by the physiotherapist. The movement may be carried out in the early stages in the treatment pool or in total suspension slings. Voluntary exercises may also be performed with the limb placed in supporting slings. By using these methods, friction and the force of gravity are eliminated and so various planes and axes of movement can be achieved. The dynamic splintage given by spring suspension simulates the water treatment and encourages normal muscle movement and rhythm.

Free movements are of great value in the later stages when co-ordination exercises are given in the lying, sitting and standing positions.

Self correction of movements and of positioning of the shoulders can be greatly aided by the use of a mirror.

3. Resisted movements.

This is undoubtedly the most important part of muscle re-education as the primary function is to strengthen the muscles. The remaining innervated muscle fibres appear to respond to progressive resistance exercises by an increase in their strength and work capacity in practically the same manner as normal muscles. In the early stages as in manual assistance, manual resistance is the best method of giving resistance exercises, because minute variations can be given, and the resistance carefully graduated to the returning power of the muscle.

Resisted movements may also be given by spring suspension in which the limb to be treated is supported comfortably in slings suspended from an overhead frame and a spring of suitable tension introduced into the circuit. Later as the muscle function improves, more effective and progressive resisted exercises can be planned with the aid of springs of varying tension or pulleys and weights. The advantage of the latter method is that the patient's progress is obvious to him thus affording him great encouragement.

With the frequent muscle charting previously advocated it can easily be assessed when any muscle group has improved sufficiently for the treatment to be "progressed", and an attempt should be made to get the muscle to work at its maximum strength.

Simple and extremely useful finger exercoses can be performed by the patient attempting to squeeze a rubber sponge or soft rubber ball of a suitable and convenient size.

Resisted exercises should be of a specific nature, because the more complex an exercise is, the greater the possibility for trick action. After six months from the onset of the disease, resisted exercises should be instituted for all muscles that can overcome gravity. The patient should undertake practise exercise before assessing the maximum load of resistanc the patient is capable of tackling. He must understand how essential is his contribution of mental and physical effort. The physiotherapist can aid by constant encouragement and by the judicious use of varied exercises to avoid monotony. There appears to be little doubt that progressive resistance exercises should be given to prime movers and the best results have been obtained by simple movements.

If the exercises produce fatigue and a diminished range of movement, they must be reduced in number and range. It is important to note however that the strength of a muscle may vary from day to day owing to central factors such as mental fatigue, or to local factors such as alteration in the temperature of a limb.

In this unit, active exercises with springs and slings, and active exercises in the Hubbard's tank are the two most important methods used.

Trick movements.

All muscles do not recover at the same rate so that inco-ordinated movements can develop at any stage in the recovery. If exercises are not expertly controlled, the patient tends to use and develop the normal muscles, thus increasing the muscular imbalance and the tendency to deformity. The reaction of a weak muscle may entirely disappear when there is overactivity of stronger muscles in the area. The weakened muscle ceases to function because it tires readily, and if the patient continues to use the limb, a new pattern of movement develops. If the functioning muscles can be kept at

their normal length, and if these inco-ordinated movements are not established, deformities can be prevented.

Commoner types of trick movements.

1. The thumb movement is frequently limited by paralysis of the abductor pollicis brevis and opponens pollicis. Opposition of the fingers and thumb to form the normal round O can often be performed by the combined action of abductor pollicis longus and flexor pollicis brevis.
2. Similarily, when flexor pollicis longus is paralysed, trick flexion of the terminal phalanx can be performed by extending the wrist and abducting the thumb.
3. In weakness of the interossei, extensor digitorum longus, extensor indicis and minimi digiti may abduct the fingers, and the fingers and hand should be exercised on a flat smooth surface when any weakness can readily be detected.
4. The fingers can be flexed by the wrist extensors, if there is some shortening of the long flexors.
5. The lateral abdominals will cause "hitching" of the pelvis on abduction, if there is weakness of the hip abductors on that side.
6. Weak hamstrings can be substituted in knee flexion, by flexion of the hip if lying in the supine position.
7. Paralysis of the quadriceps can be substituted by the gluteus maximus contracting in extension of the knee.

If after a reasonable period of time, severe paresis or paralysis persists and recovery seems to have ceased, trick movements should be encouraged and the surviving muscles developed.

Fatigue.

Great care must be taken by the physiotherapist throughout the treatment to appreciate the early onset of fatigue in any muscle or muscle group and adequate periods of rest must be given.

Fatigue can be recognised in a muscle if the last contraction does not show the same degree of power as the first or by a diminished range of movement during the immediate treatment or on the following day. Rest is then usually prescribed for two or three days.

Fatigue is assumed to arise from the accumulation of lactic acid, due to an incomplete metabolism and a defective circulation. Rapid fatigue of recovered muscles appears to be a permanent feature and may be due to axonal branching with overloading of the individual neurones. In the more complex activities, fatigue promotes asynergy and inco-ordination which interfere with the restoration of the normal patterns of activity. Early activity is often curtailed because of the danger of fatigue producing further weakness but the danger of inco-ordination produced by too strenuous exercises is probably a more important factor.

Short wave diathermy.

Electrical stimulation of muscle is in my opinion, of little value, but electrical stimulation of the phrenic nerve might prove the treatment of choice in respiratory disturbance of bulbar origin.

Massage.

The value of massage in paralytic cases is to stimulate the circulation of blood and lymph particularly in the distal parts of the limbs which are so often badly affected and so deprived of the muscular pump. If given gently, it can be very comforting to a patient with muscle and joint pain and it helps to increase the muscle tone by improving the local nutrition. It also acts by retarding and counter-acting muscle atrophy. If contractures are present, deep stretching frictions around the contracted tendons can also bring relief to the patient.

Careful massage to the weakened or paralysed muscles has also been advised after the temperature has been normal

for one week. When the muscular pain and tenderness have disappeared massage in the form of effluge with very gentle kneading and friction around the joints may be given and later increased. This helps to maintain the blood supply to the paralysed limbs.

Muscle and joint pain can be relieved, and deep friction around contracted tendons will aid treatment when contractures are present.

Local fatigue due to excessive reflex effort must be avoided, and the treatment periods should be of short duration - five minutes only, but they may be repeated two or three times a day. Picking up or friction to the antagonist muscles may also be given.

It is only right to point out that local hyperaemia is more easily and safely attained by the use of warm baths and infra-red rays, whilst contrast bathing may also be useful.

When staffing a large unit for the treatment of poliomyelitis, it must be borne in mind that each patient requires a great amount of individual attention from the physiotherapist. It is obviously impossible for each physiotherapist to devote her whole time to a single patient, and therefore in determining which type of treatment to employ, preference must be given to that which in the opinion of the surgeon gives equally good results even if administered for a shorter time.

Intermediate stage.

This stage commences with the disappearance of the muscle pain and tenderness and may last for two to three years, according to the severity and the extent of the paralysis, i.e. until the recovery of muscle power ceases, and this may be complete or only partial.

The aims of treatment must be to keep the joints of the paralysed limbs mobile and to produce hypertrophy in the remaining muscle fibres. In some cases, this will allow the

patient to discard an appliance or give a better prognosis when muscle transplantation is indicated.

The affected limbs should be supported so that every joint is in the optimum position for the function of the limb. Splints may be required as follows:

1. To keep the limb at maximum rest.
2. To oppose the influence of gravity.
3. To avoid undue stretching of a paralysed muscle beyond its normal resting length and therefore protect it.
4. To avoid shortening of healthy antagonists of the paralysed muscles.
5. To keep the joints in alignment.
6. To prevent deformities.

The simplest and lightest possible apparatus should be used at all times, as heavy appliances may do more harm than good. The splints may be made of aluminium or one of its alloys, one of the modern plastics or of light removable plaster of Pari They should be well padded and care should be taken to prevent pressure sores over the bony prominences. The splints should be removed at least twice a day to allow the physiotherapist to carry out the treatment.

Rigid immobilization is harmful because it has been shown that controlled activity is necessary to keep the paralysed muscles in good condition. If the paralysed limb was encased in a rigid splint, the following complications would be liable to occur:

1. Atrophy and muscular weakness.
2. Loss of tension of the muscle.
3. Interference with the circulation of the muscle and the limb as a whole.
4. Peri-articular adhesions.
5. Atrophy of bone.
6. Loss of active and passive movement.
7. Inability to exercise the muscles effectively.

The type and degree of protection however, depends on the following factors:

1. The degree and location of the muscular weakness.
2. The amount of pain or muscle spasm.
3. The condition of the ligaments supporting the joints and the stability of the joints.
4. The relative strength of the opposing groups of muscles.
5. The duration of the disease since its onset.

I found that individual prescribing was essential to determine whether a splint is required.

When activity is increased, care should be taken to use appliances to hold the weakened and paralysed part in a position so as to encourage co-ordinated action.

The protection by means of splints and appliances is usually necessary up to and during the intermediate period or until the patient has made the maximum recovery.

In the later intermediate stage, further indications for the use of appliances are :

1. To mobilize the patient by supporting unstable limbs or other parts of the body.
2. To correct and prevent deformities and so avoid surgical procedures.
3. To protect, assist or substitute for weakened muscles.
4. To immobilize, mobilize or limit movements of joints.
5. To balance muscles and therefore maintain neutrality of muscle pull.

Re-education of posture and walking.

Everything possible should be done to help recovery of the muscles essential for balancing, standing and walking. If the patient has to use crutches, particular attention should be paid to the triceps, flexors and extensors of the wrist and the movements of the fingers. Even with extensive

flaccid paralysis of the leg, abdominal and back muscles, without flexion contraction deformities of the hip, knee or ankle, or if these have first been corrected, and the patient's arms are capable of using crutches, he can be taught to balance correctly and later to walk.

Postural balance is maintained by the postural reflexes. These are activated through the proprioceptive system in the muscles and joints, the eyes and the labyrinth. These reflexes are outside the control of volition and keep the body in the erect position.

The erect position is maintained by every joint being balanced tonically. The supporting muscles which are working against gravity pull harder than those of the opposite side. Thus the tonic contraction of muscles controlling joints is increased when balance is altered by gravity. A good posture is one in which the mimimum of muscle activity is required in controlling the erect position. A poor posture requires increased effort to regain a good position and in doing so, fatigue quickly results.

Balance, both local and general, is disturbed when there is paralysis of a muscle, disability being caused more often from deformity through the disturbed balance than from paralysis. Both contractures and paralysis of muscles may cause ligaments to stretch, these being subject to continual strain. The stretching of the ligaments may cause subluxation. Unexpected deformities occur due to the action of gravity, e.g. hyperextension of the knee due to attempts to lock it. Once deformities are established, they progress rapidly even though they commence insidiously.

Principles of re-education for posture.

1. Restore and maintain mobility of the joints.
2. Correct bad habits.
3. Develop muscle sense by relaxation.
4. Re-establish co-ordination and control.

Treatment.

In poliomyelitis, the continual effort of adjustment in balancing against gravity induces fatigue and in a programme of treatment, rest must have an essential place.

Posture training must begin in the lying position and only progress to sitting and standing when the postural sense is achieved. Corrective posture exercises of breathing and balancing must be taught in the lying position, progressing to sitting and finally standing.

Joints which have a tendency to stiffness should be treated by manipulation and passive movements. If this is done from an early stage in the disease, a full range of movement can be obtained. The small joints of the hands, feet and spine are very difficult to free once stiffness has been established.

Structural modifications of the ligaments and capsules of joints and in the spine, changes in the inter-vertebral discs may become permanent.

Mobility exercises must not be given to the spine if there is a likelihood of scoliosis because the presence of a degree of tightness can minimize deformity.

Faulty attitudes brought about by disability must be corrected. These can occur at an early stage in the disease whilst the patient is still recumbent. A child with paralysis of the lower limbs may be allowed to sit up too frequently and this may cause contraction of the hip flexors and stretching of the gluteus maximus.

The position of a child's bed if not frequently altered, may be the cause of scoliosis. If the bed is against a wall, the child will turn in one direction, and constantly lifting the head may lead to an aggravated cervical curve.

Relaxation forms an essential part in corrective training; also breathing exercises with the trunk held habitually in good posture. Natural breathing with full use of the diaphragm must be the aim.

Exercises directed towards re-educating the postural reflexes in co-ordination and control must be commenced in the horizontal position. The gravity-resisting muscles such as the intrinsics of the foot, quadriceps, glutei, trapezius and pre-vertebral cervicals must be exercised individually.

When the patient has learned control of these muscles, exercises must be given to co-ordinate action by the glutei and the abdominals working together to flatten the lumbar spine. The mastering of co-ordination can only be successful if gravity is eliminated.

Every patient should have postural correction, because any weakness of the muscles of the upper limb, lower limb or pelvis may affect the position of the head, the upper part of the trunk or the whole body posture. The correct posture should be taught at the earliest possible moment, even whilst the patient is in the supine position and he must attempt to maintain the corrected position voluntarily.

In cases with isolated upper limb paralysis, the patient can be placed in the sitting position at an early date, but no definite time can be given because here as in other cases to be described later, this will depend upon the rate of progress and the response to treatment. If the trunk muscles escape, he is allowed to stand for a short time each day after a period of two months. If the trunk muscles only are affected, sitting up may be allowed after a few months of complete recumbency. The best guide to the appropriate time is when the muscle chart shows no further improvement.

If the spine, neck, trunk and shoulder girdle muscles are markedly involved, sitting up may not be possible for twelve to eighteen months. The patient may even then require the aid of a spinal support, otherwise scoliosis and kyphosis may develop rapidly and become permanent. Sitting posture is assumed very gradually and in the beginning it may be allowed for only a few seconds once or twice a week. When the patient

is accustomed, he should begin balance exercises by the use of movement of the head, arms and legs. Later flexion, extension, lateral flexion and rotation of the trunk are introduced and any tendency to scoliosis is watched for very carefully. If this is observed, mobilizing and hyperextension exercises should be given.

If the back, gluteal and leg muscles are extensively involved, weightbearing was formerly postponed for about eighteen months, but it may be begun much earlier in a treatment pool which is fitted with parallel bars. Provided the patient's arms are strong enough, it is possible to teach balance and weightbearing in water approximately four to six months from the time of the initial attack.

When the patient is first allowed out of bed, great care must be taken to avoid general fatigue. First of all he must concentrate on co-ordination of his muscles and on balance. Standing balance should be practised and well developed before walking is attempted, otherwise a most unstable gait may result. Careful observation is required to detect any tendency to deformity particularly of the back and pelvis. A patient wearing an arm abduction splint must be watched carefully and if necessary postural exercises are prescribed.

When the patient starts to walk, instruction is given in foot-placing, co-ordination of the arms and legs, shifting of the body wieght and such control of various regions of the body as the individual patient requires. Parallel bars and walking machines are excellent for this purpose, but again any erroneous deflection of the body weight during walking must be prevented. Crutches and walking sticks can also be used when required and they may always be necessary to allow the patient to get about. The patient should be taught to take steps of appropriate length and he may be instructed in "four-point" walking or in the use of tripod gait.

Experience has shown that quite a number of poliomyelitis patients have been unable to master the technique of the tripod gait and this is possibly due to the weakness of the muscles of the trunk and upper extremities.

The patient should be taught to get in and out of bed unaided and he may even learn with patience to sit down on, and get up from a chair or lavatory seat. Even a very severely paralysed patient should be able to ascend and descend stairs.

Although the patient may have shown no apparent improvement for several months, he may after assuming the erect position progress to a remarkable degree. On the other hand, loss of the power of the muscles may occur. This is due to overfatigue or from a too early removal of the supports. Unfortunately in a few instances, this damage may be permanent.

Summary of postural training.

General activity exercises must not be given because they increase the faulty posture through fatigue.

The patient must be taught correct posture with the aid of a wall mirror.

Posture and balance must be taught in the lying position.

Individual muscle action must first be controlled and then co-ordinated movements. When this is achieved, gravity can be introduced with the sitting position and finally the standing position.

Essential muscles for function.

The two factors which require immediate attention are:

1. Recognition of the most important muscle for recovery.
2. The techniques of treatment which will aid recovery.

The following list of muscles may act as a guide to the physiotherapist considering priority of their importance:

Upper limb.

The importance of the muscles diminishes from the fingers to the shoulders.

Opponens - paralysis may occur with loss of opposition to the thumb and loss of skill in hand movements.

Flexors of the elbow - paralysis or weakness in the elbow flexors is a greater disability than loss of triceps. It must however be remembered that the triceps is an important muscle if the patient has to walk with the aid of crutches. A triceps grip added to a crutch can support the elbow if the triceps is weak.

Deltoid - the importance of aiding recovery to the deltoid for abduction of the arm is obvious.

The abdominals - the abdominal muscles function in respiration, control obliquity and function in movements of the spine.

Lower limb.

The importance of the muscles diminishes from the hip to the toes.

Extensors of the hip - these are essential for walking. If the gluteus maximus is completely paralysed or is very weak, it is sometimes necessary to train the adductor magnus which is a weak extensor of the hip. This can be achieved with the leg internally rotated.

Quadriceps - if the quadriceps muscle is paralysed, the patient usually has to wear a caliper. Its true function is as a fixator of the knee and extension is secondary to this.

Calf - if both plantar flexors and dorsiflexors are involved, one should aim for the recovery of plantar flexion.

Class exercises and games.

A number of patients can be given a class of free movements and light games. By this means the patients improve in general health and morale. They are not allowed however to joing class

exercises for the first six months of the disease as fatigue may prove detrimental to recovery. Exercises aim at developing powers of endurance whilst the selection of exercises is carefully planned and consideration is given to the varying degree of paralysis in each patient.

The class is best conducted in a gymnasium with each patient lying on a mattress so that adequate rest can be obtained between exercises. Special class exercises should be given for the trunk, back and limbs and competitive games are introduced for co-ordinated movements.

There are three class grades:

1. Mat exercises - patient lying.
2. Exercises mainly in the sitting position.
3. Activity in the erect position.

Head exercises, trunk flexion, extension, lateral flexion and rotation, and limb and breathing exercises with frequent short rest periods are given.

The simplest apparatus may be used, e.g. balls of varying weight.

Organised games in the form of sitting P.T., basket ball and volley ball are also useful forms of exercise. Archery has been found to be very beneficial for cases with upper limb palsies, and wheel chair polo for paraplegic cases.

Occupational therapy.

A skilled occupational therapist is an invaluable asset to the poliomyelitis team.

Remedial and diversional therapy should play a large part in the convalescent treatment, as the "will to do" must be stimulated and any inherent ability discovered and encouraged.

During the period of convalescence, productive work of an interesting nature should be carried out, e.g. modelling, weaving, wood-turning and wood-work. For younger children,

brick-building and other forms of play requiring muscle co-ordination are useful for upper limb paralysis, whilst the riding of scooters is useful in lower limb paralysis.

Finally, it is necessary to stress that conservative treatment should not be prolonged beyond a reasonable recovery period, as this will delay final adjustment and postpone any necessary orthopaedic treatment.

Residual stage.

When this stage has been reached in about eighteen to twenty-four months from the onset of the disease, and provided that the patient has had correct treatment from the beginning, it may be found that protection is required indefinitely for the lower extremities or the back to prevent a progressive stretching of the ligaments and joint instability. Although 70% of all cases make a complete recovery without treatment, it is the seriously paralysed cases with imbalance of the opposing groups of muscles which require this protection for several years if not for the duration of their lives.

In untreated cases, an attempt should first of all be made to correct the deformities by a period of conservative treatment with physiotherapy and mechanical fixation, before any operative treatment is undertaken.

Treatment of circulatory disturbances of the extremities.

The peripheral circulation can be improved by the employment of general and local measures.

Rest in bed with adequate sleep is essential in the early part of the treatment as vasodilatation occurs during sleep. The feet are kept warm with bedsocks or well-protected hot water bottles, and they should be washed daily and thoroughly dried. Methylated spirits are applied and allowed to dry and then lanoline is rubbed in to keep the skin supple and free from scales.

Active and passive exercises are encouraged and heat may be applied in the form of dry heat or hot baths.

When the patient starts to walk, he must wear a fresh pair of socks daily, and his shoes should be of soft leather and loose fitting. Great care should be taken in cutting the toenails and corns, and if a blister appears, it should be snipped, the skin removed and a dry dressing applied.

Regular exercise, short of fatigue should be encouraged, and throughout treatment smoking and alcoholic drinks are forbidden.

In the case of the lower limb, 2% novocaine is injected into the lumbar ganglia and in the case of the upper limb, the stellate ganglia. One side only at a time should be done, in order to be able to compare the two sides. After the first injection, signs of sympathetic paralysis appear usually within ten minutes and improvement lasts for approximately three days, this period being longer with each subsequent injection.

Pain, muscle spasm and muscle tenderness may be completely relieved by this method of treatment and it may be used in the acute and convalescent phases even in children.

Cyanosis and discolouration of the skin with mild to severe oedema and hyperhidrosis are replaced by a flushed, dry and warm skin and the gradual disappearance of the oedema. The mobility of the parts is also improved and deformity tends to be diminished.

Psychological treatment.

This most important part of the treatment should begin as early as possible after the onset of the disease in an attempt to counteract the mental strain which some patients must undergo when they realise suddenly that in the matter of a few hours they have been transformed from normal healthy persons into seemingly helpless cripples.

If possible, patients should be admitted to a ward especially

reserved for this type of case, because the patients will then see the progress made by their fellows and this is a great help to their morale.

The younger patients especially must be guided in the early stages so that they can adjust themselves to a prolonged convalescence.

The excellent psychological effects of pool therapy must be remembered. Patients who lie completely immobile in bed even for weeks, find that they can seemingly move their paralysed limbs in water and are thus encouraged to make further efforts. Also the upright position and walking can be commenced much earlier if pool therapy is adopted.

As the period of treatment of poliomyelitis is necessarily prolonged over several months if not years, the importance of schooling must not be forgotten and all special centres should have full teaching facilities.

Amongst the adult patients, there is a need for education and instruction to enable them to support themselves financially and to lead an independent existence when discharged from hospital.

The patient is taught to accept his handicap and to acquire poise, self-assurance and dexterity. He thus obtains a proper valuation of his physical self and to a large extent is able to lose his selfconsciousness.

Severely paralysed patients often become very depressed at the thought of leaving hospital, because they feel incapable of standing up to the stress and strain of everyday life. This difficulty can to a large extent be overcome by allowing patients to go to their homes on week-end leave whenever they become ambulant and their condition permits. This helps them to become more self-reliant as there they can mix with their friends and relatives and gradually come to realise that they are not nearly so handicapped as they had at first imagined.

Poliomyelitis complicating pregnancy.

There seems to be little doubt that the incidence of poliomyelitis and polioencephalitis associated with pregnancy appears to be on the increase.

In the 1947 epidemic, 9.3% of women in the childbearing age group were affected but there was not an unduly high incidence of the disease in pregnant women. Taylor and Simmons (1948) found that the incidence of poliomyelitis in pregnancy was twice as great in pregnant as in non-pregnant women of the same age group.

Horstmann et al (1946) found that 32% of female patients admitted were pregnant and 20 out of the 64 cases were aged between 20 and 34 years.

Fox and Gennett (1945) reported that they had been able to trace eighty-five cases of pregnancy complicated by poliomyelitis and they concluded that pregnancy increased the susceptibility to poliomyelitis due to endocrine upsets of the ovarian, pituitary and foetal hormones.

Taylor and Simmons have suggested that this may be due to the increased congestion and permeability of the upper respiratory and digestive tracts which allow the virus an easier portal of entry during pregnancy.

In Taylor and Simmons' cases, 76% occurred in the first six months of pregnancy. Of the 24% who developed poliomyelitis in the last three months of pregnancy three died, and the other three had severe residual paralysis.

Aycock (1946) feels that there is a tendency for the disease to occur in the first trimester of pregnancy if the foetus is a male and in the third trimester if the foetus is a female, but judgment on this statement should be reserved.

Aycock and Ingall (1946) reported on six cases of abortion which occurred in the first three months of pregnancy.

It is now generally held that age, number of previous

pregnancies and the stage of the pregnancy do not appear to be factors in the susceptibility of the pregnant woman to poliomyelitis. It is generally accepted that there is a higher incidence of abortion in women who contract poliomyelitis during pregnancy than in the normal pregnancy. This however is quite usual in several of the other infectious diseases. The incidence of bulbar involvement in definitely increased in the last trimester of pregnancy and may be as high as 23%. Bulbar poliomyelitis shows no tendency to result in abortion, premature labour or to cause precipitate labour.

Gillespie (1941) recprded a successful case of Caesarian section in a young mother of 18 years, but this operation is indicated only in very severe cases, particularly if respiratory paralysis is present as it is usually the only means available to obtain a viable child when the death of the mother is imminent. Normally poliomyelitis in the mother does not interfere with the normal process of labour or spontaneous delivery and the only indications for interference are for those complications which are liable to occur in any pregnant woman. Grelland (1947) states that of 35 patients who went into labour, 32 were delivered normally. The lack of voluntary muscular effort does not contra-indicate vaginal delivery and the degree of maternal spinal paralysis has no serious effect on labour. Poliomyelitis in my experience did not appear to have any effect on uterine activity.

Statistics tend to show that no advantage is to be gained in interrupting pregnancy at any stage in these cases unless the poliomyelitis has occurred several years previously, causing pelvic deformity due to unilateral dislocation of the hip or paralysis of one leg. Pregnancy may however increase the severity of the poliomyelitis or its complications such as cystitis or diaphragmatic paralysis.

In our series of 600 cases, 19 were found to be pregnant.

On close examination of their signs and symptoms, there does not appear to be any marked difference between the pregnant and the non-pregnant patients, e.g. 55% were feverish, 38% had headache, 40% had loss of appetite, whilst 60%, 55% and 45% had pain in the back, pain in the limbs and pain in the neck respectively.

The time between the onset of the illness and the first appearance of the paralysis varied from one to ten days, and it was sudden in onset in 68% of cases.

The distribution of the paralysis was widespread, but in only four cases were the cranial nerves involved. These included the right facial and glossopharyngeal.

The duration of pregnancy at the onset of poliomyelitis was as follows:

at 3 months	5 patients
at 5 months	1 patient
at 6 months	5 patients
at 7 months	2 patients
at 8 months	3 patients
at 9 months	3 patients

The trimester did not appear to have any significance. The termination of pregnancy did not appear to depend upon the extent of the paralysis but upon the effect of the disease upon the general condition of the patient. It would therefore appear that the effect of poliomyelitis on pregnancy does not differ a great deal from any other acute infective lesion.

In the absense of respiratory embarrassment, pregnancy, labour and delivery were carried out on strictly orthodox lines and there did not appear to be any undue obstetrical hazard.

Poliomyelitis and pregnancy did not appear to cause any congenital malformations nor did it appear to retard the general development of the foetus or its birth weight.

The residual paralysis did not appear to be influenced by the fact that the patient was, or had been pregnant.

At least 80% of the maternal mortality seems to occur in the last trimester or in the immediate puerperium and it is noted that the mortality is higher amongst pregnant than in the non-pregnant patients.

Non-paralytic and abortive forms are suspected largely on a basis of symptomatology.

Bowers and Hanforth 1955. pointed out that seriously ill patients died because of the poliomyelitis and polioencephalitis and not because of the associated pregnancy.

Having now given my general impressions and conclusions as derived from the study of over 600 cases, I will now divide my series into two groups which were observed in the earlier epidemics of 1947 and 1949, and see in what respect they differ from my series as a whole.

As stated in the introductory paragraphs, the symptoms vary greatly from epidemic to epidemic and from individual to individual. I have attempted to demonstrate this by dividing my series of cases into:

1. 215 cases seen during the 1947 epidemic.
2. 144 cases seen during the 1949 epidemic.

The total 600 cases as already described in the text were seen between 1947 and the end of 1953. A book is at present in the hands of the publishers incorporating all the facts which I have observed during that time.

Seasonal incidence.

The epidemics in this country appear to reach their peak during the months of July, August and September, and this was confirmed in my series of cases. The lowest incidence occurred during the month of January but it must however be stated that poliomyelitis does occur in an epidemic form during all seasons of the year, although the number of paralytic cases is much higher during the summer months. In my series of cases, the fall in the number of cases declined slowly during the winter months.

Age.

In the epidemics which I have observed, it was obvious that the disease is certainly not confined to infants and the most severely paralysed cases were in the over 15 years age group. Trueta (1953) found that in his series 50% of patients were in the over 15 age group and of these 69% were paralytic. In children especially, an early diagnosis is most important and examination of all children in an infected area should be carried out by a competent paediatrician. Patients should be

warned that children should avoid fatigue and where possible, prevent coming in contact with sick children.

Tonsillectomy during an epidemic should be avoided if possible and injury, operations and strenuous exercise should be prevented. Six of our cases in the 1947 epidemic gave a history of strenuous exercises during the early abortive stage and the result in each case was a very extensive and severe paralysis.

Sex.

In none of the series mentioned above was there any marked difference in the sex incidence.

Date of onset of disease and notification.

In the 1947 and 1949 epidemics, the time between the onset of the disease and its notification was usually one week. The average time in each case was approximately three to seven days, whilst in the later cases, the average time was two to five days. This improvement could be accounted for by the fact that following the 1947 and 1949 epidemics, the medical profession as a whole, were much more alert to the possibility of poliomyelitis. It was also observed that the number of days between the onset of the illness and the first appearance of paralysis varied greatly and this appeared to be shorter if the patient was young.

Temperature.

In the 1947 epidemic, the temperature was raised to 100° F in 69%, 68% and 64% in the 0-5, 5-15 and over 15 age groups respectively, whilst in the 1949 epidemic, the percentage in these groups was 66%, 74% and 57%. Rigors occurred in 18%, 27% and 34% in the 1947 epidemic, and in 21%, 17% and 22% in the 1949 epidemic. This elevation of temperature usually persisted for two to six days in the earlier series. Moderate elevation of temperature or a long duration of fever appeared to me to be of grave diagnostic significance.

Catarrh.

Catarrh.

In the 1947 epidemic, catarrh in the chest was present in 13%, 14% and 7% of the 0-5, 5-15 and over 15 age groups respectively, whilst in the 1949 epidemic, these percentages had altered to 15%, 8% and 34%. In the 1947 epidemic, catarrh in the nose was present in 18%, 22% and 12% in the three age groups and 24%, 22% and 33% in the 1949 epidemic. Catarrh in the throat occurred in 13%, 14% and 16% in the 0-5, 5-15 and over 15 age groups but this had increased to 22%, 38% and 30% respectively in the 1949 epidemic.

Headache.

Headache was a fairly common symptom in the 1947 epidemic. It had a frontal distribution in 45%, 58% and 72% in the three age groups but by 1949, these figures were 32%, 92% and 60%. The headache was occipital in its distribution in 1947 in 15%, 22% and 25% but this had increased to 16%, 26% and 35% by 1949.

Vomiting.

In 1947 this occurred in 50%, 62% and 60% in the three age groups but in 1949, it was 45%, 60% and 42%.

Loss of appetite.

In 1947 this was noted in 88%, 80% and 97% in the three age groups whilst in t he 1949 epidemic, the distribution was 62%, 70% and 52%.

Sore throat.

In t he 1947 epidemic, sore throat was complained of in 46%, 52% and 51% in the three age groups but in 1949, the percentages were only 21%, 40% and 42%.

Insomnia.

In 1947, this was noted in 32%, 33% and 48% in the three age groups, but in 1949, this had changed to 42%, 56% and44%, respectively.

Pain in the limbs.

In the 0-5, 5-15 and over 15 age groups, pains in the limbs

were present in 61%, 46% and 75% in 1947, but by 1949, this had altered to 67%, 78% and 70%.

Pains in the joints.

Although never a very marked feature, in 1947 it occurred in 36%, 28% and 48% in the three age groups and by 1949 the percentages were 35%, 55% and 38%.

Tenderness of muscles.

Although stated by certain observers to be relatively uncommon in this country, I found that the percentages in the three age groups in 1947 were 48%, 40% and 74% whilst in 1949, the percentages were 48%, 74% and 66%.

Pain the the neck.

In 1947 this occurred in 58%, 54% and 74% in the three age groups, but was 43%, 62% and 63% in 1949.

Pain in back.

In 1947, the percentages were 50%, 58% and 88% in the three age groups but was 43%, 71% and 82% in 1949.

Stiffness of back.

The percentages in the three age groups in 1947 were 53%, 50% and 60% respectively whilst in 1949 it was 55%, 85% and 65%.

Head retraction.

This occurred in 23%, 20% and 17% of cases in the three age groups in 1947, and in 15%, 14% and 12% in 1949.

Nuchal rigidity.

This was present in 53%, 71% and 56% of cases in the three age groups in 1947, and in 26%, 40% and 26% in 1949.

Kernig's sign.

In 1947, this was present in 45%, 66% and 61% of cases but in 1949 it was only present in 21%, 14% and 17% of cases.

Jerky movements of limbs.

This was not a common finding and whilst occurring in 32%, 28% and 20% of cases in 1947, it occurred in only 13%, 33% and 7% of cases in 1949.

Twitching of individual muscles.

This was present in 16%, 2% and 17% of cases in the three age groups in 1947, and in 5%, 14% and 17% of cases in 1949.

Retention of urine.

This relatively uncommon finding was present in 10%, 3% and 10% in the three age groups in 1947, but in 1949 this had increased to 23%, 26% and 26% of cases.

Incontinence of urine.

This was relatively uncommon and was only observed in 5%, 10% and 3% in the three age groups in 1947, but by 1949, this had fallen to nil, nil and 1%.

Constipation.

In 1947, this was present in 52%, 41% and 50% of cases in the three age groups, and in 1949 in 50%, 66% and 63% of cases.

Diarrhoea.

In 1947, this occurred in 13%, 10% and 7% of cases in the three age groups, and in 1949, the percentages were 3%, 7% and 12%.

Dizziness.

In 1947, this occurred in 14%, 25% and 30% of cases in the three age groups, whilst in 1949, the percentages were 2%, 33% and 24%.

Diplopia.

This was complained of in 2%, 6% and 10% of cases in the three age groups in 1947, and in 2%, 7% and 10% of cases in 1949.

Unequal pupils.

This was observed in 6%, 2% and 4% of cases in the three

age groups in 1947, and in nil, nil and 2% of cases in 1949.

Nystagmus.

This was noted in 14%, nil and 4% of cases in 1947, and in 2%, 4% and 2% of cases in 1949.

Photophobia.

In 1947, this occurred in 32%, 19% and 21% of cases, and in 1949 it was present in 13%, 11% and 19%.

Generalised convulsions.

In 1947, these occurred in 7%, 6% and 4% of cases in the three age groups whilst in 1949 the percentages were 2%, nil and nil.

Stupor.

In 1947, this was noted in 10%, 6% and 12% in the three age groups but it was not noted at all in 1949.

Coma.

This was also only observed in the 1947 epidemic and occurred in 2%, nil and 2% in the three age groups.

Hemiplegia.

During 1947, this occurred in 2% nil and 4% in the three age groups and in 1949 it occurred in 5%, 1% and 1%.

Cerebellar ataxia.

In 1947, this was present in 1%, 4% and 1% of cases in the three age groups but in 1949 it was present in 1%, 3% and nil.

Paralysis of the respiratory centre.

In 1947, this was present in nil, 3% and 12% of cases in the three age groups whilst in 1949 it was present in nil, 3% and 5% of cases.

Spinal type of respiratory failure.

In 1947 this occurred in 10%, 4% and 17% of cases in the three age groups and in 2%, 15% and 4% of cases in 1949.

Paralysis of facial nerve.

In 1947 this occurred in 6%, 3% and 4% of cases and in 1949, in nil, nil and 8% of cases.

Paralysis of the soft palate.

In 1947, this occurred in 1%, 8% and 8% of cases in the three age groups and in 1949 it occurred only in 1%, nil and 2%. 1947 epidemic.

Onset of paralysis.

In the 0-5 age group during the 1947 epidemic, the onset of paralysis was sudden in 34 cases and gradual in the remainder. The paralysis increased during the first twenty four hours of it being noticed in 55% of cases and the extent of the paralysis remained constant in the remaining 45%. In no case was the extent of the paralysis seen to diminish during this time. The direction of spread of the paralysis was ascending in50% of cases and descending in 50%.

In the 5-15 age group, the paralysis usually appeared between the first and the fifth day from the onset of the illness. There did not seem to be any difference in the percentage of cases whether the paralysis was of sudden or gradual onset. The paralysis increased in extent during the first twenty-four hours in 25 cases, and remained constant during that time in 5 cases. The direction of spread was ascending in type in 50% of cases and descending in 50%.

In the 15+ age group, the onset of the paralysis was sudden in 55% of cases and gradual in 45%. In 90% of cases the paralysis was seen to increase in extent during the first twenty-four hours, but again the direction of spread was 50% ascending and 50% descending.

Muscles affected during height of paralysis.

Leg.

Gluteus maximus	37 cases
Gluteus medius	34 cases
Quadriceps	29 cases
Psoas	26 cases
Hamstrings	27 cases
Tibialis posticus	24 cases
Tibialis anticus	24 cases
Peronei	19 cases.

Small muscles of foot 20 cases.

Arm.

Serratus magnus	3 cases.
Deltoid	12 cases
Biceps	8 cases
Triceps	7 cases
Pronators	5 cases
Supinators	5 cases
Flexors of wrist	5 cases
Extensors of wrist	5 cases.
Small muscles of hand	6 cases.

In the 0-5 age group, there were ten children under the age of one and the distribution of paralysis was as follows:

1947	F. 5months.	Left leg.
	F 7 "	Both legs.
	M 7 "	Left arm and right leg.
	F 8 "	Both legs,
	F 9 "	Both legs.
	M 9 "	Right deltoid only.
	F 10 "	Left hemiplegia.
	F 10 "	Left arm and left 7th cranial nerve.
	F 11 "	Right leg.
	M 11 "	Left leg.

In the 0-5 age group, the muscles affected during the height of the paralysis were as follows:

Both legs	11 cases.
Left leg	7 "
Right leg	5 "
Right arm	2 "
Left arm	1 case
Left arm and leg	1 "
Right " " "	1 "
Left arm " right leg	1 case
Both arms	1 "

Also in this age group, there was a loss of sensation in the soles of the feet and along the lateral aspect of both legs. In one case, the plantar aspect of the foot and leg were involved another case showed loss of sensation of the left arm and leg, whilst two cases showed evidence of loss of sensation of the lateral aspect of the left leg.

Some distribution of muscle tenderness was noted in four cases which showed subsequent paralysis. In one case there was equal tenderness in all affected muscles but there was an unequal degree of paralysis. In another case, the paralysed muscles were most painful especially the deltoid which subsequently showed no recovery. It was also noted that there was frequently no tenderness in muscles which subsequently became paralysed. Another important point noticed was that there could be no paralysis or paresis of the limbs although there was a loss of knee jerks. In one case, the limb became stiff and swollen two days before the onset of the paralysis. In one case, there was paralysis of the right deltoid and swelling of the arm around the right elbow. Four cases were admitted with poliomyelitis who had a history of contact with a case of measles five week previously. The facial nerve was involved in two cases, one on the left side and one on the right

In the 5-15 age group, there was loss of sensation of the left leg and foot followed by a paralysis of the whole of the left leg in one case. In another case there was loss of sensation over both arms and legs and this was followed by paralysis of both legs. In yet another case, there was loss of pain and temperature sense on the opposite side below the lesion. In the vast majority of cases, the pain and tenderness of the muscles lasted approximately one week but odd cases persisted up to one month. In this group, it was observed that the degree of muscle tendness bore no relationship to the extent of the resultant paralysis. The time between the onset of the illness and the first appearance of paralysis was usually one to five days. The ratio of gradual onset to sudden onset was 1:1. The paralysis was noted to increase in extent within twenty-hours in all but 5 cases and in these no increase was noted.

The ratio of ascending to descending type of spread was again 1:1.

Muscles affected during the height of paralysis.

Gluteus maximus	21 cases.
" Medius	20 "
Quadriceps	17 "
Psoas	17 "
Hamstrings	17 "
Tibialis posticus	13 "
" anticus	13 "
Peronei	13 "
Small muscles of foot	13 "

Arm.

Serratus magnus	5 cases.
Deltoid	5 "
Biceps	5 "
Triceps	5 "
Pronators	4 "
Supinators	4 "
Flexors of wrist	3 "
Extensors of wrist	3 "
Small muscles of hand	3 cases

Reflexes.

In all cases where it was noted, it was found that the reflexes were diminished.

Cranial nerves.

In one case paralysis of the 4th cranial nerve was noted, the patient suffered from diplopia and the eye looked downwards and outwards.

In the 15+ age group, in one case there was loss of sensation in both arms and hands for two to three days and this was followed by paralysis of both upper limbs. In another case, there was numbness of both legs followed by paralysis of both lower limbs. Another patient complained of hyper-aesthesia around the waist and this was followed by paralysis of the lower abdominal muscles and both legs. In this group, the tenderness of the muscles usually disappeared after the second day and the pain after three weeks. Also in this

group, the degree of muscle tenderness did not appear to bear any relationship to the severity of the resultant paralysis. Again, the time between the onset of the illness and the first appearance of paralysis was usually one to five days. Rarely it appeared up to the end of the second week but in no instance did it occur later than that. The ratio of sudden to gradual onset of paralysis was 1:1.

In all but three cases, the paralysis increased in extent within twenty-four hours of it first being noticed. In three cases there was no increase of the primary paralysis.

Again, the direction of spread was in the ratio of ascending : descending :: 1:1.

Reflexes.

In all cases where the reflexes were noted, they were found to be diminished:in three cases they were found to be increased.

Cranial nerves.

In one case there was a lesion of the 3rd cranial nerve and this was shown by ptosis of the uppereyelid, a fixed dilatation of the pupil and the eyeball turned outwards and downwards.

1949 epidemic.

The youngest patient was five months old and he suffered from paralysis of the shoulder girdle and forearm on the left side. There was also weakness of the rectus abdominis. Another patient of six years suffered from epistaxis, and this was later followed by convulsive attacks.

In the 5-15 age group, there was a definite history of injury preceeding the paralysis. Another patient complained of pain in the feet during the night but this was not present during the day.

In the 15+ age group, one patient showed marked emotional upset and there was delirium in one patient.

Three cases had marked dysphagia and there was paralysis of the right half of the tongue in one case.

Loss of pain and temperature sense.

This occurred in ten cases but none in the 0-5 age group. In the 5-15 age group, there were two cases in which the loss of temperature sense lasted for three days, and one case in which it lasted for five days. The loss of pain and temperature sense was bilateral in two cases one of which died. In the surviving case the loss of pain and temperature sense lasted for a period of nine days. In the 15+ age group, loss of temperature was observed in two cases and persisted for three days, whilst in one case, loss of temperature sense lasted four days. In two cases, there was a bilateral loss of pain and temperature sense which persisted for two days.

Pain and tenderness of muscles.

In the three age groups, this pain and tenderness of the muscles lasted for approximately seven days, but in occasional cases, it persisted for anything up to six weeks. The distribution was as follows:

0-5	age group	-	38 cases
5-15	" "	-	34 "
15+	" "	-	24 "

The degree of muscle tenderness appeared in this series to bear some relationship to the severity of the resultant paralysis, although in some cases there was no pain and tenderness before the onset of the paralysis. In 43 cases there was slight tenderness of the muscles which was followed by severe paralysis. In four cases, the muscles of the legs and arms were very tender, but there was no resultant paralysis. In the 5-15 age group, there were twelve cases with moderately severe muscular pain but the paralysis was slight. In the 15+ age group, the pain and tenderness of the muscles was slight, but the resultant paralysis was slight in eleven cases.

In the 15+ age group, one patient complained of severe, dull pain at the base of the spine before the onset of paralysis ten days later. Six cases in the 0-5 age group gave a history of tonsillectomy within two months of the onset of the attack. In two cases a petechial rash appeared within forty-eight hours after the onset during which the temperature rose to 102°F. In the 0-5 age group, two cases gave a history of a severe attack of measles before the onset of poliomyelitis. In the 5-15 age group, operative interference was noted in five cases. In two cases tonsillectomy had been performed recently, in two cases appendicectomy and in one circumcision. In the 15+ age group, two cases gave a recent history of acute tonsillitis and one had had an operation for rectal fistula which was followed by paralysis of the lower limbs. In all the age groups, the time between the onset of the illness and the first appearance of paralysis was approximately one to five days. In 70% of cases the onset of paralysis was sudden and in 30% it was gradual. In 55% the paralysis increased in extent within twenty-four hours of it first being noticed, whilst in 45% there was no appreciable change. This applied to all the age groups. Also in all the age groups, the direction of spread was 50% ascending, and 50% descending.

Muscles affected during the height of paralysis.

Right arm	- 8	cases
" leg	- 10	"
" arm and leg	- 4	"
Shoulder girdle	- 1	case
Legs, arms and diaphragm	- 2	cases
Both legs and back	- 18	"
Face, neck, back and legs	- 2	"

These all occurred in the 0-5 age group.

5-15 age group.

Left arm	-	2 cases
" leg	-	12 "
L.arm and l.leg	-	2 "
Diaphragm	-	6 "
L.leg and back	-	8 "
Erector spinae	-	1 case
" " and l.arm.	-	2 cases
Facial palsy and arms	-	2 "

15+ age group.

Both arms	-	2 cases
" legs	-	12 "
Neck muscles	-	2 "
Both legs and arms	-	2 "
" arms, abdominals and sternomastoid	-	1 case
Muscles of shoulder girdle	-	16 cases
All 4 limbs, erector spinae	-	3 "

In all the above groups, the proximal limb muscles were affected twice as often as the distal ones.

Reflexes.

Where these were recorded, the reflexes were normal in 40% of cases, diminished in 40% and were absent in 10%.

Cranial nerves involved.

In all the age groups, three cases complained of dysphagia, thus indicating involvement of the 9th cranial nerve. In all these cases, there was also invovlement of the 7th cranial nerve. In one case there was involvement of the 3rd and 10th cranial nerves and one case had involvement of the 9th, 10th and 11th. In one case, there was involvement of the 6th left cranial nerve whilst in another there was involvement of the 3rd and 5th. Lastly one case showed involvement of the 9th, 10th, 11th and 12th.

I will now turn my attention to the muscle charts prepared during the patient's stay in hospital. During the 1947 epidemic, the results were as follows.

0-5 age group.	On admission			1mth		2mths		3mths		6mths		9mths	
	Total	0-3	4	0-3	4	0-3	4	0-3	4	0-3	4	0-3	4
Sternomastoid	54	4	-	3	-	3	-	-	-	-	-	-	-
Abd. upper	54	14	5	20	7	8	4	6	4	-	1	-	1
lower	56	13	7	9	9	8	4	6	4	-	1	-	1
lat.	49	10	6	8	7	6	4	5	5	-	1	-	1
Arm - prox.	52	2	2	3	1	2	1	2	1	-	-	-	-
distal	51	-	-	-	2	-	2	-	1	-	-	-	-
deltoid	53	2	2	3	2	2	2	1	1	-	-	-	-
Leg prox.	60	23	5	18	8	16	6	14	6	5	2	4	4
distal	48	28	4	15	2	19	3	16	4	7	2	7	-
Erector spinae - cerv.	48	10	7	8	5	3	4	1	2	-	-	-	-
dors.	47	11	5	9	3	9	3	3	4	-	4	-	-
lumb.	47	10	6	9	7	10	4	7	2	4	-	-	-

5-15 age group.	On admission			1mth.		2mths		3mths		6mths		9mths.	
	Total	0-3	4	0-3	4	0-3	4	0-3	4	0-3	4	0-3	4
Sternomastoid	28	3	14	4	8	3	9	2	4	-	6	-	6
Abd. upper	27	11	6	9	1	10	2	6	-	4	-	4	-
lower	28	10	5	10	-	9	-	4	-	2	-	2	-
lat.	27	13	4	7	1	9	1	4	-	3	1	2	1
Arm- prox.	28	6	7	6	8	5	7	5	2	1	2	1	1
distal	28	5	5	2	5	2	3	1	1	1	1	1	1
deltoid	28	11	10	6	9	9	8	6	-	3	2	2	3
Leg- prox.	29	16	8	6	8	8	6	3	2	5	-	4	-
distal	27	12	4	7	3	4	4	5	2	4	-	3	1
Erector Spinae - cerv.	28	10	4	6	3	6	4	3	1	1	-	1	-
dorsal	28	12	2	10	-	10	-	5	-	7	-	4	-
lumbar.	28	8	6	7	5	4	6	3	2	3	1	2	1

0-3 = no strength of contraction up to contraction against gravity only.

4 = strength of contraction against gravity and resistance.

15+ age group.	On admission			1mth		2mths		3mths		6mths		9mths	
	Total	0-3	4	0-3	4	0-3	4	0-3	4	0-3	4	0-3	4
Sternomastoid	29	4	18	4	10	3	7	2	7	-	9	-	6
Abd. upper	36	22	4	21	4	17	2	17	1	16	1	10	-
lower	39	20	8	19	5	18	3	17	2	15	-	10	-
lat.	36	28	8	24	5	14	7	12	7	9	5	5	4
Arm -prox.	32	16	14	14	12	9	6	7	6	7	4	1	1
dist.	23	10	5	9	3	5	5	5	4	2	2	1	1
deltoid	31	17	13	17	8	11	5	7	6	6	5	3	3
Leg.-prox,	36	29	6	22	11	22	8	15	10	14	8	10	3
distal	29	18	8	13	12	10	6	9	8	8	6	5	-
Erector Spinae-													
cerv.	39	20	8	18	5	10	5	8	6	5	3	1	1
dorsal	36	26	6	23	5	12	2	11	2	8	4	7	5
lumbar	37	29	4	27	4	16	4	15	3	9	3	8	2

During the 1949 epidemic, the following results were obtained.

0-5 age group.	On admission			1mth		2mths		3mths		6mths		9mths.	
	Total	0-3	4	0-3	4	0-3	4	0-3	4	0-3	4	0-3	4
Sternomastoid	34	10	7	8	5	8	5	7	2	1	2	-	1
Abd. upper	31	11	7	3	10	3	5	2	4	2	2	-	2
lower	32	8	10	4	10	5	6	3	3	2	2	1	2
lat.	30	7	10	8	8	8	8	3	6	3	3	2	2
Arm - prox.	24	8	8	8	4	4	4	3	2	3	2	2	2
distal	25	11	4	5	1	2	3	1	3	-	-	-	-
deltoid	24	5	4	4	3	4	3	3	2	3	1	-	2
Leg.-prox.	30	10	7	8	7	8	6	3	5	2	5	2	2
distal	30	9	4	8	4	7	5	5	4	4	1	-	3
Erector spinae -													
cerv.	33	6	13	2	5	2	4	2	-	-	2	-	2
dorsal	34	4	13	4	10	2	5	2	-	2	-	2	-
lumbar	33	4	12	3	10	2	3	2	2	2	-	2	-

0-3= no strength of contraction up to contraction against gravity only.

4= strength of contraction against gravity and resistance.

5-15 age group	On admission		1mth		2mths		3mths		6mths		9mths.	
	Total	0-3	4	0-3	4	0-3	4	0-3	4	0-3	4	0-3	4
Sterno-mastoid	16	9	3	7	2	6	4	4	5	2	7	-	8
Abd. upper	18	9	4	9	4	7	5	4	4	2	4	1	2
lower	18	9	3	9	3	6	-	6	-	5	1	4	-
lat.	19	8	6	4	5	2	4	2	3	-	3	-	2
Arm-prox.	19	6	4	4	2	4	2	3	2	2	2	2	2
dist.	19	4	2	4	2	3	1	1	2	1	1	1	1
deltoid	19	6	2	6	1	5	1	4	1	4	1	4	-
Leg-prox.	18	10	3	7	3	6	2	5	4	2	5	1	3
distal	18	2	4	2	2	2	2	1	1	-	-	-	-
Erector spinae-cerv.	18	-	7	-	4	-	4	-	3	-	3	-	2
dorsal	19	6	4	5	4	4	3	3	3	1	3	-	6
lumbar	18	6	6	3	2	2	2	1	1	-	1	-	-

15+ age group	On admission			1mth		2mths		3mths		6mths		9mths	
	Total	0-3	4	0-3	4	0-3	4	0-3	4	0-3	4	0-3	4
Sterno-mastoid	21	12	3	9	6	6	5	4	5	4	1	-	2
Abd. upper	25	24	4	19	5	10	1	9	2	8	6	-	2
lower	30	24	6	15	5	12	6	8	6	7	9	-	2
lat.	30	19	8	15	9	11	4	4	8	4	3	-	-
Arm-prox.	20	14	8	9	9	5	7	2	2	2	2	2	2
dist.	21	8	8	8	5	6	3	4	3	4	2	2	2
delt.	25	13	10	6	10	7	4	6	5	4	5	2	4
Leg-prox.	25	19	3	17	4	9	6	5	3	1	1	1	1
dist.	25	18	3	10	5	9	1	3	2	2	2	1	1
Erector sp inae-cerv.	21	11	9	7	12	-	4	-	4	-	4	-	-
dorsal	30	15	5	14	6	4	5	3	4	1	2	-	-
lumbar	31	19	5	16	4	8	2	5	4	4	-	-	-

0-3 = no strength of contraction up to contraction against gravity only.

4 = strength of contraction against gravity and resistance

Another method which I have used recently to determine the results of treatment in paralytic cases is as follows. The strength of contraction was determined at the onset of treatment and again at periods of three months up to one year. With this in mind the following tables were compiled.

0-5 age group.

Right leg					Left leg.			
Proximal		Distal			Proximal		Distal	
M	R	M	R	3mths.	M	R	M	R
2	2	1	3		-	2	2	4
2	3	2	3		1	2	-	3
3	4	3	4		3	4	4	5
4	5	3	5		2	5	4	5
5	5	5	5		-	3	-	-
3	5	4	5		2	4	1	2
1	1	3	4		-	-	-	-
				6mths.				
3	5	4	5		2	5	5	5
3	3	4	5		4	5	5	5
-	3	5	5		3	5	5	5
3	4	3	5		3	3	3	3
-	3	-	2		3	5	-	1
5	5	5	5		1	3	-	6
3	5	5	5		3	4	5	5
				9mths.				
3	5	-	5		5	5	5	5
3	4	3	5		3	5	5	5
4	5	4	5		4	5	3	4
3	4	3	5		-	1	-	3
3	5	5	5		2	2	5	5
3	3	-	-		4	5	5	5
				12mths.				
-	3	-	-		3	4	5	5
3	4	3	5		-	1	-	3
3	4	3	5		3	4	4	5
-	3	1	3		5	5	5	5
2	4	3	4		-	2	5	5
5	5	5	5		3	5	-	3

0-5 age group.

Right arm					Left arm			
Proximal		Distal			Proximal		Distal	
M	R	M	R	3mths.	M	R	M	R
3	5	3	5		3	5	3	5
4	4	5	5		4	4	5	5
3	5	4	5		3	5	4	5
-	3	5	5		-	3	5	5
2	3	-	-		5	5	5	5
3	5	3	5		3	5	3	5
				6mths.				
4	5	5	5		3	5	3	5
5	5	5	5		2	3	4	5
2	3	-	-		5	5	5	5
3	5	3	5		3	5	3	5
-	3	5	5		-	3	5	5
5	5	5	5		3	4	-	3
				9mths.				
2	3	-	-		5	5	5	5
3	5	4	5		4	5	5	5
2	4	-	2		5	5	5	5
3	5	2	4		-	2	4	5
-	-	2	3		5	5	5	5
5	5	3	5		5	5	4	5
				12mths.				
-	2	-	2		5	5	5	5
3	5	5	5		4	5	5	5
3	4	4	5		5	5	5	5
-	2	3	5		-	3	5	5
5	5	5	5		3	4	-	3
-	2	-	2		3	4	-	3

0-5 age group.

Abdomen.

Right							Left.					
Upper		Lower		Lateral			Upper		Lower		Lateral.	
M	R	M	R	M	R	3mths.	M	R	M	R	M	R
3	4	3	4	3	4		5	5	5	5	5	5
3	3	3	3	3	3		3	3	3	3	3	3
2	3	2	3	2	3		2	3	2	3	2	3
						6mths.						
3	4	4	4	4	4		3	4	3	4	4	4
2	2	3	3	3	5		2	2	2	3	3	5
2	3	2	3	3	4		2	3	2	3	3	3
						9mths.						
3	5	3	5	3	5		3	5	3	5	3	5
2	3	2	2	2	4		2	3	2	2	3	3
3	5	3	5	3	5		3	5	3	5	3	5
						12mths.						
5	5	2	4	2	4		5	5	2	4	2	4
3	5	3	5	3	5		3	5	3	5	3	5
3	4	2	3	4	5		3	4	2	3	3	5

Erector spinae.

Right							Left					
Cerv.		Dorsal		Lumbar			Cervical		Dorsal		Lumbar	
M	R	M	R	M	R	3mths.	M	R	M	R	M	R
3	5	3	4	3	3		5	5	4	4	3	3
4	5	4	5	4	5		4	5	4	5	4	5
4	4	2	4	2	4		4	4	2	4	2	4
						6mths.						
4	5	4	5	4	5		5	5	5	5	5	5
3	4	3	3	3	4		3	4	3	3	3	4
5	5	3	5	5	5		5	5	4	5	5	5
						9mths.						
4	5	5	5	4	5		4	5	4	5	4	5
3	5	3	4	3	3		5	5	4	4	3	3
4	4	2	4	2	4		3	4	2	4	2	4

5-15 age group.

Right leg				Left leg			
Proximal		Distal		Proximal		Distal	
M	R	M	R	M	R	M	R
			3mths.				
3	5	5	5	3	5	5	5
-	-	-	-	-	-	-	-
3	3	5	5	2	4	3	4
2	4	-	-	5	5	5	5
1	3	3	4	3	3	5	5
3	4	5	5	3	3	5	5
			6mths.				
-	-	-	3	-	-	-	-
-	-	-	-	-	-	-	-
2	3	3	5	4	4	4	5
2	2	-	2	2	2	-	2
5	5	5	5	4	5	5	5
4	5	5	5	4	5	5	5
			9mths.				
2	4	5	5	2	2	5	5
4	4	4	4	5	5	5	5
4	5	5	5	-	4	-	-
2	5	5	5	3	5	5	5
4	5	5	5	3	4	2	2
2	3	3	5	4	4	5	5
			12mths.				
4	5	5	5	2	4	5	5
-	2	-	2	-	4	3	4
-	1	1	2	2	3	4	4

5-15 age group.

Right arm					Left arm			
Proximal		Distal			Proximal		Distal	
M	R	M	R	3mths.	M	R	M	R
4	5	5	5		4	5	5	5
4	5	5	5		4	5	5	5
3	5	5	5		3	5	5	5
3	4	3	5		2	4	3	5
3	5	4	5		3	4	3	5
				6mths.				
3	5	3	5		4	5	3	5
5	5	5	5		-	2	2	3
3	3	5	5		1	4	4	4
				9mths.				
3	5	3	5		4	5	3	5
4	5	5	5		4	5	5	5
2	4	5	5		3	3	5	5
				12mths.				
5	5	5	5		3	5	5	5
-	2	3	4		3	4	4	5

Abdomen.

Right							Left					
Upper		Lower		Lateral			Upper		Lower		Lateral	
M	R	M	R	M	R	3mths.	M	R	M	R	M	R
5	5	3	4	3	4		5	5	3	4	3	4
4	4	3	4	5	5		4	4	3	4	3	5
5	5	2	4	2	4		4	5	4	5	4	5
						6mths.						
2	4	2	4	5	5		2	4	2	4	5	5
-	5	2	5	3	3		2	5	2	5	3	4
3	4	2	3	4	5		3	4	2	3	3	5
						9mths.						
3	3	2	2	-	3		2	4	2	4	5	5
2	5	2	5	3	5		-	5	2	5	2	5
3	5	3	5	4	5		3	5	3	5	3	5
						12mths.						
4	5	5	5	5	5		4	4	3	4	4	5
3	5	2	5	3	5		3	5	2	5	3	5
-	2	-	-	-	3		-	1	-	-	-	3

5-15 age group.

Erector spinae.

Right							Left					
Cervical		Dorsal		Lumbar			Cervical		Dorsal		Lumbar	
M	R	M	R	M	R	3mths.	M	R	M	R	M	R
3	5	5	5	5	5		3	5	5	5	5	5
5	5	3	3	3	3		5	5	3	3	3	4
3	4	1	2	3	4		3	4	1	2	3	4
						6mths.						
4	5	3	4	3	4		4	5	3	4	3	4
5	5	2	4	3	5		5	5	2	4	2	5
5	5	2	5	2	5		5	5	2	5	2	5
						9mths.						
4	5	2	4	2	5		4	5	2	4	2	5
4	5	2	4	2	3		4	4	2	4	2	3
5	5	1	4	5	5		5	5	1	4	4	5

15+ age group.

Right Leg					Left leg.			
Proximal		Distal			Proximal		Distal.	
M	R	M	R	3mths.	M	R	M	R
-	-	-	-		-	-	4	4
2	2	3	5		2	4	5	5
-	2	3	4		-	2	4	4
-	-	-	-		-	-	-	-
2	4	2	3		1	3	5	5
				6mths.				
-	-	-	-		-	-	-	-
3	3	2	4		-	2	-	-
1	3	4	5		1	2	3	3
2	5	3	5		3	4	4	5
3	4	5	5		4	5	5	5
				9mths.				
3	5	1	4		2	3	-	2
1	3	2	5		3	5	2	4
1	5	-	3		3	5	5	5
1	3	5	5		1	3	3	5
2	2	4	5		-	-	-	-
				12mths.				
3	5	4	5		3	5	4	5
-	2	5	5		2	2	3	5
3	3	3	3		2	3	2	3
-	2	2	2		-	1	2	3
4	4	4	4		3	4	3	4

15+ age group.

Right arm					Left arm			
Proximal		Distal			Proximal		Distal	
M	R	M	R	3mths.	M	R	M	R
3	5	3	3		4	5	5	5
-	1	2	2		3	3	2	2
-	1	-	1		-	1	2	3
-	-	-	3		2	3	1	3
4	4	4	4		4	5	5	5
				6mths.				
3	4	3	5		2	5	5	5
3	5	5	5		3	5	3	5
4	5	4	5		4	5	4	5
1	4	3	4		5	5	5	5
3	5	3	5		4	5	5	5
				9mths.				
4	5	5	5		3	4	2	2
3	5	5	5		3	5	5	5
3	5	2	3		-	2	4	5
-	3	5	5		1	4	5	5
-	1	3	4		1	3	3	5
				12mths.				
3	4	4	4		3	4	3	5
2	5	5	5		4	4	5	5
4	5	4	5		3	4	4	5
4	5	4	5		4	5	4	5
4	5	4	5		3	5	4	5

15+ age group.

Abdomen.

Right							Left					
Upper		Lower		Lateral			Upper		Lower		Lateral	
M	R	M	R	M	R	3mths.	M	R	M	R	M	R
-	-	-	-	-	-		-	-	-	-	-	-
2	2	2	2	4	5		2	2	2	3	4	4
2	2	2	2	3	3		2	2	2	2	3	3
3	3	3	3	3	3		1	1	1	1	2	2
2	2	2	2	3	4		1	1	1	1	2	2
						6mths.						
3	5	3	5	3	5		3	5	3	5	3	5
2	2	2	2	3	3		3	3	2	3	4	4
-	1	-	1	-	1		-	1	-	1	-	1
2	2	-	2	2	4		3	3	2	3	2	5
3	5	3	5	2	5		3	5	3	5	2	5
						9mths.						
3	3	2	3	2	4		3	3	2	3	2	4
2	2	2	2	1	5		2	2	2	4	3	5
2	3	2	2	3	5		2	3	1	2	3	5
2	3	-	3	1	4		2	3	-	3	-	3
-	2	-	1	-	1		-	1	-	-	-	1
						12mths.						
2	3	2	3	3	5		2	3	2	3	2	5
3	5	4	5	5	5		4	5	4	5	2	5
2	2	2	2	2	4		2	2	2	2	2	2
2	2	2	2	3	4		5	5	5	5	5	5
2	2	1	2	-	2		1	2	1	2	-	3

Erector spinae.

Right							Left					
Cervical		Dorsal		Lumbar			Cervical		Dorsal		Lumbar	
M	R	M	R	M	R	3mths.	M	R	M	R	M	R
3	5	4	5	4	5		3	5	4	5	4	5
5	5	4	5	4	4		5	5	4	5	4	4
2	2	-	2	2	2		3	3	-	-	2	2
2	4	2	4	2	4		3	4	3	4	3	4
2	4	1	2	1	2		2	4	1	2	1	2
						6mths.						
4	5	1	3	2	3		4	5	1	3	2	3
3	3	2	2	2	3		3	3	2	2	2	3
5	5	1	2	1	2		5	5	1	2	1	2
3	5	2	3	2	4		3	5	2	3	2	4
5	5	2	2	2	2		5	5	2	2	2	2
						9mths.						
5	5	1	3	2	2		5	5	2	5	4	4
3	5	1	5	2	5		3	5	1	5	2	5
-	4	-	2	-	2		-	4	-	1	-	2
4	5	2	3	3	3		3	5	2	3	4	4
3	5	1	3	2	3		2	5	2	5	2	5

In the above tables, the following contractions were used:
P - proximal group of muscles.
D - distal group of muscles.
M - maximal muscle power at onset of treatment.
R - resultant muscle power after treatment.
O - no contraction.
1 - flicker.
2 - contraction but not against gravity.
3 - contraction against gravity only.
4 - contraction against gravity and resistance.
5 - normal contraction.

The figures O-5 therefore indicate the strength of muscle contraction.

In the above tables, the following muscles were involved in order of frequency:

Leg.
Gluteus maximus
Gluteus medius
Quadriceps
Psoas
Hamstrings
Tibialis posticus
" anticus
Peronei
Small muscles of foot.

Arm.
Serratus magnus
Deltoid
Biceps
Triceps
Supinators
Pronators
Extensors of wrist
Flexors of wrist.

It was also noticed that the proximal group of muscles in the leg were affected twice as often as the distal ones. In the arm the ratio was 3:1.

In my total series, the left leg was affected in a ratio of 2:1 as compared with the right leg in the O-5 age group, whilst in this group the right arm was affected in the ratio of 4:3, as compared with the left arm. In the 5-15 age group, the right arm and the left arm were affected in the ratio of 3:2 and the right leg and left leg were affected in the ratio of 1:1.

In the 15+ age group, the right arm and left arm were affected in the ratio of 1:1 whilst the right leg and left leg were affected in the ratio of 2:1.

In the leg, it was also noted that the extensor muscles were more often affected than the flexors. This also was true for the abductors and evertors when compared with the adductors and invertors of the foot.

It was also noted that where the abdominal muscles and erector spinae were involved, this was usually bilateral and to an equal extent.

On the question of recovery, careful examination of the original muscle charts have led me to the conclusion that the distal muscles recovered more quickly than the proximal ones.

The muscles of some patients showed permanent paralysis and these muscles finally degenerated . The amount of degeneration depended upon the degree of damage to the controlling motor nerve.

In other cases, the affected muscles undergo disuse atrophy, the amount depending upon its controlling motor nerve being temporarily out of action.

Detailed study of the patients' muscle charts have led me to conclude that muscles which are classified in the 0, 1 or 2 stages of contraction very seldom if ever recover any useful function, even after skilled muscle re-education.

Illustrative cases.

Male, aged 21 years. First complained of pains in his legs and these were vigorously massaged. Received treatment for malaria even after he became paralysed. He was feverish and had headache and had vomited for several days. Pain in legs and joints and tenderness of muscles which lasted two months. He was incontinent of faeces for seven days. He had obstructive collapse of the right lung and severe dyspnoea, after removal from the iron lung in which he received treatment for two weeks. The latter condition was treated by postural drainage and breathing exercises. After four months his muscle chart was as follows:

Muscles	Right	Left
Sternomastoid	3 - 4	3 - 4
Abdomen - upper	0 - 0	0 - 0
lower	0 - 0	0 - 0
lateral	0 - 0	0 - 0
Arm proxomal	3 - 5	4 - 4
Distal	5 - 5	5 - 5
Erector spinae.		
cervical	2 - 4	2 - 4
dorsal	0 - 2	0 - 2
lumbar	0- 0	0 - 0
Lower limbs	0 - 0	0 - 0

Female aged 28 years.

Pain in back which lasted four weeks. Retention of urine lasted five days followed by incontinence. General hyperaesthesia in the acute stage, most marked in back. Feeling of constriction in upper part of back even after four months. Paralysis of diaphragm and respirations maintained only by accessory muscles of respiration. On examination, there was marked contraction of the fingers and thumb of t he right arm and no active extension was present. In the right arm there was also flexion contraction of the elbow, pectoralis major and latissimus dorsi. In the left arm, there was flexion contraction of the fingers and thumb and a marked ulnar deviation of the left wrist. There was also an equinus deformity of both feet.

Her vital capacity on lying was 350 ccs. but on sitting, this was increased to 400 ccs. The muscle chart four months after the onset was:

	Right	Left
Abdomen - upper	2 - 2	2 - 2
lower	2 - 2	2 - 2
lateral	3 - 3	3 - 3
Arm - shoulder girdle	0 - 0	0 - 1
proximal muscles	0 - 0	2 - 3
distal "	0 - 0	1 - 3
Flexorsof fingers	0 - 2	0 - 2
Extensors of fingers	3 - 3	0 - 2
Erector spinae - cervical	2 - 2	3 - 3
dordal	0 - 2	0 - 0
lumbar	2 - 2	2 - 2
Leg - proximal	3 - 4	3 - 3
distal	3 - 5	3 - 5

Female aged 25 years.

On admission she had marked spasm of the left leg which made it impossible to perform the muscle tests. One month after admission she developed acute appendicitis and appendicectomy was performed. Convalescence was uneventful. Two months after admission, the spasm of the left leg persisted and there was a marked equinus deformity. At this time a parasympathetic block was done, and there was an immediate increase in the temperature of the left leg. An hour after the injection, the whole limb was relaxed and passive movements were given throughout the full range. The next day, the whole limb was spastic in the morning but after a few manipulations, it became relaxed again and a full range of passive movements was possible. One month later there was full movement of the ankle, knee and hip when moved passively.

Muscle chart after one year.

	Right	Left
Sternomastoid	4 - 4	4 - 4
Abdomen- upper	2 - 2	2 - 2
lower	2 - 2	2 - 2
lateral	2- 2	2 - 2
Upper limbs	4 - 5	4 - 5
Erector spinae- cervical	3 - 4	3 - 4
dorsal	0 - 2	0 - 2
lumbar	2 - 2	2 - 2
Leg - proximal	2 - 3	5 - 5
distal	3 - 3	2 - 3

Complications.

I wish to point out that quite a number of patients had had previous treatment before admission to a recognised poliomyelitis centre. The following complications were noted;

In the 0-5 age group.

Genu valgum	-	15 cases
Genu recurvatum	-	3 "
Scoliosis	-	3 "
Lordosis	-	1 case
Talipes equinus	-	4 cases
Abdominal herniation	-	3 "
Pes planus	-	1 case

In the 5-15 age group.

Dorsi-lumbar scoliosis	-	6 cases
Subluxation of hip joint	-	3 "
Pes cavus	-	1 case

In the 15+ age group.

Scoliosis	-	5 cases
Dorsi-lumbar kyphosis	-	2 "
Subluxation of hip joint	-	5 "
Talipes equino-varus	-	3 "
Kyphosis	-	4 "
Genu recurvatum	-	1 case
Talipes equino-valgus	-	2 cases
Abdominal herniation	-	2 "

Poliomyelitis and pregnancy.

In my series of 13 cases observed during the epidemics of 1947 and 1949, 55% suffered from fever which however was present for only two to five days. Rigors were present in 20% of cases whilst catarrh of the chest, nose and throat occurred in 10% of cases. Frontal and occipital headache occurred in 20% of cases each but it usually only lasted for twenty-four hours. Vomiting was present in 30% of cases and it too only lasted for twenty-four hours. In 10% of cases the patient was irritable and difficult to manage and in 40% of cases, the patient complained of loss of appetite. Sore throat occurred in 25% of cases and insomnia in 40%. Pain the limbs was a symptom in 55% of cases but in only 15% and 25% was there pain in the joints and tenderness of muscles. Forty-five per cent complained of pain in the neck and pain in the back occurred in 60% . The 'knee-kissing' test was positive in 40% of cases. Head retraction only occurred in 10% of cases, nuchal rigidity in 20% of cases and the Kernig's sign was positive in 10% of cases. 15% had jerky movements of the limbs, and twitching of individual muscles occurred in 20% of cases. 25% of cases suffered from retention of urine and 30% had constipation and only 15% had diarrhoea.

Other signs of polioencephalitis were only present in isolated cases and these included diplopia, nystagmus, photophobia, generalised convulsions, stupor and coma. Hemiplegia only occurred in one case.

Paralysis of the respiratory centre and diaphragm occurred in two cases whilst paralysis of the facial muscles and soft palate appeared in three and two cases respectively.

The pain and tenderness of the muscles lasted for periods of time varying from four days to two weeks.

The time between the onset of the illness and the first appearance of paralysis varied from one to ten days, the average time being 3.5 days.

In six recorded cases the paralysis was sudden in onset, whilst in only four cases was the onset gradual.

In five cases, the paralysis increased in extent within twenty-four hours of it first being noticed.

The direction of spread of the paralysis was particularily noted in six cases being ascending in three cases and descending in three cases.

The muscles principally affected during the height of the paralysis were the shoulder muscles, right and left arm, deltoid, right and left leg and abdominal muscles.

Naturally from this relatively small number of cases, no definite conclusions should be drawn, but it is hoped that they will eventually form the nucleus of a larger and more illustrative series.

Illustrative cases.

Patient aged 36 years. First symptom was pain and tenderness in the lower ribs, back and hips. During the first twenty-four hours the patient was extremely irritable and short-tempered. 'Knee-kissing' test was positive and she complained of pain in the neck and back. There was definite nuchal rigidity. Retention of urine was present for 120 hours. Paralysis did not appear until three days after the onset which was gradual. Muscles of both hips and erector spinae were affected during the height of the paralysis. The patient was seven months ~~preg~~ pregnant and she was delivered normally at full term.

Patient aged 22 years. She complained of pain in the back of three days duration and also pain in the neck. The 'knee-kissing' test was positive. The time between the onset of illness and the first appearance of paralysis was ten days.

The paralysis was sudden in onset and was descending in type. The patient was eight months pregnant and was delivered normally at full term.

Patient aged 35 years. Time between the onset of the illness and first appearance of the paralysis was one day. It was of sudden onset and ascending in type. The muscles affected were the cervical and dorsal erector spinae, the shoulders and upper arms. She was six months pregnant on admission and aborted twenty-four hours later.

Patient aged 23 years. Pain in limbs and joints and tenderness of muscles. Pain in neck and back. 'Knee-kissing' test positive. Head retraction, nuchal rigidity and Kernig's sign positive. There was inequality of the size of the pupils, generalised convulsions and coma. Paralysis of the diaphragm occurred and she was placed in an iron lung. She died six days later.

Patient aged 32 years. 'Dromedary' type of temperature. Nuchal rigidity, twitching of facial muscles, generalised convulsions followed by coma of short duration. She had some difficulty in breathing and swallowing was disturbed. She had left facial paralysis and paralysis of the palate and diaphragm. The convulsive attack was diagnosed as ?eclamptic. She was 38 weeks pregnant but delivered normally at full term.

Patient aged 27 years. Admitted with paralysis of abdominals and dorsal and lumbar erector spinae and total paralysis of both legs. The child was delivered by Caesarian section, but it was premature and died one week later.

Duration of pregnancy.

In 1947 and 1949 epidemics, the duration of pregnancy was as follows; two at three months of which one died; one at five months; three at six months of which two died; two at seven months of which one died; three at eight months of which two died; and two at nine months. In the whole series, the maternal mortality was 8% and the foetal mortality was 40%.

Cerebrospinal fluid.

I wish to make it quite clear that in my opinion there is no indication for doing a lumbar puncture during an epidemic of poliomyelitis unless there are definite signs of increased intracranial pressure and a differential diagnosis from meningitis is in doubt. I reached this conclusion in 1949 after a detailed study of over a hundred specimens of cerebrospinal fluid taken from paralytic poliomyelitis patients of various ages and at various times after the onset of the disease. Changes in the cerebrospinal fluid are not necessarily specific in the early stages of the disease although occasionally a slight increase in the number of cells may be noted. In approximately 25% of the cases which I examined, the pressure of the cerebrospinal fluid was slightly increased, and in 95% of the cases the fluid was clear. It was however noted that the cell count appeared to be slightly higher in the 0-5 age group than in the 15+ age group where the duration of the disease was equal.

The cellular reaction was mainly mononuclear, especially in the first few days, but it usually dropped to normal by the end of the first week. As the disease progressed, there was a definite increase in the number of mononuclear cells but the total cell count was diminished. Also it was noted, that there was quite a marked increase in the number of white cells without there being any clinical symptoms which could be diagnosed as evidence of meningitis. In most cases, there was a definite preponderance of polymorphonuclear cells in the first few days and this was followed later by a marked lymphocytosis and a consequently diminishing polymorphonuclear cell count usually by the end of the second or beginning of the third week after the onset.

The concentration of albumen was also seen to rise and reach its maximum by the end of the third week. In a few cases, it did not return to its normal value for anything up to three months.

The serum globulin showed a slight rise in a few cases but this was by no means constant.

Careful study failed to reveal any increase in the chloride content although the sugar content did show a slight increase in a few cases. It was however noted that in the 0-5 age group, the chloride content was somewhat lower while the protein content was higher.

Taking into account all the clinical findings and the results of the examination of the cerebrospinal fluid, I was forced to the conclusion that there definitely appeared to be no relationship between the composition of the cerebrospinal fluid and the severity of the disease. I also showed that the prognosis could not be based on the findings of the cerebrospinal fluid. I will give the following examples to illustrate the above points. (see over) . In all cases examined, the culture was sterile and the W.R. and Khan tests were negative.

Age	Time since onset	Pressure	Clear or cloudy	Cells Poly's	Cells Lymphs.	Protein	Globulin.	Chl.	Glu.
						mgm%		mgm%	
3yrs	2days	+	Clear	2	-	50	Neg.	715	N.
4 "	5 "	N	"	-	13	55	Trace.	720	N.
4 "	14 "	N	"	5	225	75	+	700	N.
7 "	2 "	N	"	50	5	40	+	740	N.
12 "	8 "	N	"	-	13	55	Trace	720	N.
8 "	15 "	N	"	45	150	30	+	705	N.
25 "	1 "	Sl. +	"	5	10	50	Trace	720	N.
24 "	7 "	N	"	13	87	50	Neg.	725	N.
28 "	14 "	N	"	5	210	75	+	700	N.
*18 "	2 "	Sl. +	"	2	-	50	Neg	710	N.

*This patient was pregnant but apart from the slight increase in pressure and protein content, the cerebrospinal fluid was normal.

Conclusions.

After perusal of the patients case sheets, it would appear that the earlier the diagnosis is made and the sooner the patient undergoes a period of complete rest, then the better will be the prognosis. The possibility of permanent damage to the neuromuscular units is thereby lessened.

Careful study of the patients muscle charts makes it clear that there is a tendency for severe paralysis to occur and increase in extent the greater the age of the patient.

In the case of poliomyelitis, I have found that the system of grading muscles recommended by the Peripheral Nerve Injuries Committee of the Medical Research Council did not appear to give quite sufficient information and I therefore tentatively suggest the following:

Strength of contraction denoted by:

0 = no contraction.
1 = flicker of contraction.
2 = contraction but only when gravity eliminated.
3 = contract ion against gravity only.
4 = contraction against gravity and slight resistance.
4+= contraction against gravity and moderate resistance.
5 = normal contraction against powerful resistance but muscle tires quickly.
5+= apparently normal muscle contraction.

In addition, I would suggest that the letters (T), (P) and (S) be added where applicable in the muscle chart when the muscles concerned show muscle tenderness, muscle pain or pain when the muscle is stretched.

It was also obvious that each case required individual consideration.

There appears to be little doubt that patients who have had tonsillectomy are much more liable to polioencephalitis than those who have their tonsils still in situ. In my patients I found that this was true regardless of the age of the patient or the time that had elapsed since the operation.

It was also noted that rest was essential for recovery, but that recovery could not take place without activity. Inexpertly prescribed activity may cause deformity, insufficient muscular reaction, delayed recovery and even loss of strength.

Death in poliomyelitis is usually due to inhalation of secretions, blood or vomit into the lungs. This results in collapse of one or more lobe and possibly necrosis of the lung tissue. Circulatory collapse may result with death in a few hours.

Atelectasis of the lung resulting from aspirated secretions or food is treated immediately by bronchoscopy and antibiotic treatment with the patient in the prone position.

Visible paralysis of the lower cranial nerves results in dysphagia and the regurgitation of food through the nose. The airway is obstructed from the pooling of secretions in the oro-pharynx, aspirations of vomitus, food, or reflex spasm of the glottis. This is best treated by postural drainage where the pillows are removed and the patient placed in the prone position with his head to one side. He should be prevented from turning back into the supine position except under supervision or the ability to swallow returns. Mechanical suction may also be necessary, or the chest squeezed manually during expirations to assist the expulsion of inhaled secretions. Secretions from the mouth are swabbed away, and some assistance may be obtained by raising the foot of the bed eighteen inches.

Impairment of the muscles of respiration can be detected by watching the rate and depth of respirations. The patient should be treated in a respirator. If pulmonary oedema results atropine should not be used to reduce the profuse watery secretions, as they then become extremely sticky. If in a respirator, there should be short periods of positive pressure breathing to facilitate care of the skin, sphincters and limbs.

Abductor paralysis of the vocal cords should be treated by immediate intubation or tracheotomy.

Sedatives are definitely contra-indicated in all cases with involvement of the bulbar centre. Restlessness should not be controlled by sedatives until the patient is in the respirator.

Gastric distension may occur during convalescence and produce nausea, abdominal distension, respiratory embarrassment and occasionally death.

If postural drainage, frequent changing of posture and bronchoscopy fail to maintain an airway or to remove the bronchial secretions, then tracheotomy is indicated.

Tracheotomy should be done in every patient who shows evidence of severe and rapidly progressing paralysis where the vital capacity is diminishing toward a marginal level (25% of the patient's normal vital capacity). It should be stated that laboratory tests cannot replace careful clinical observations as a guide in determining when a mechanical respirator should be used.

I must reluctantly conclude that to my knowledge there is no efficient method of curing the disease, preventing the onset of paralysis or controlling an epidemic. All we can do at present is to try and repair the damage, and whilst admitting that deformities cannot be completely prevented, we can keep them within reasonable limits by known methods.

QUESTIONNAIRE TO PATIENT'S DOCTOR.

PATIENT'S NAME ... AGE ... SEX....

1. Date of onset of illness ...

2. How did illness start ? ...
   1. Feverish? ...
   2. Temperature at onset and for how many days was it raised? ....
   3. Rigor? ...
   4. Catarrh in chest ...
      in nose ...
      in throat ...
   5. Headache - frontal or occipital? ....
   6. Vomiting? ...

3. Was the patient irritable and difficult to manage? ...

4. Did the patient have:-
   1. Loss of appetite? ...
   2. Sore throat? ...
   3. Insomnia? ...
   4. Pain in the limbs? ...
   5. Pain in the joints? ...
   6. Tenderness of muscles? ...
   7. Pain in the neck? ...
   8. Pain in the back? ...
   9. Stiffness of back and reluctance to stoop, e.g. knee-kissing test? ....
   10. Head retraction? ...
   11. Nuchal rigidity? ...
   12. Kernig's Sign present? ...
   13. Jerky movements of limbs? ...
   14. Twitching of individual muscles? ...
   15. Retention or incontinence of urine? ... (Duration in hours)
   16. Constipation? ...
   17. Diarrhoea? ...
   18. Dizziness? ...
   19. Diplopia? ...
   20. Inequality of size of pupils? ...
   21. Nystagmus? ...
   22. Photophobia? ...
   23. Generalised convulsions? ...
   24. Stupor? ...
   25. Coma? ...
   26. Hemiplegia? ...
   27. Acute cerebellar ataxia? ...
   28. Paralysis of respiratory centre?....
   29. Paralysis of facial muscles? ...
   30. Paralysis of diaphragm? ...
   31. Paralysis of soft palate? ...
   32. Loss of sensation? Give distribution. ...

33. Loss of pain and temperature sense on opposite side below lesion? .....

5. How long did pain and tenderness of muscles last? ....

6. Did the degree of muscle tenderness bear any relationship to severity of resultant paralysis? Please give details.

7. Time between onset of illness and first appearance of paralysis .....

8. Was paralysis of gradual or sudden onset? ...

9. Did the paralysis increase or decrease in extent within 24 hours of it first being noticed? Please give details.

10. Give direction of spread
e.g. legs to back and arms (ascending) ...
arms to back to legs (descending) ...

11. Muscles affected during height of paralysis. ....

12. Reflexes.

	Right	Left
Supinator		
Biceps		
Triceps		
Abdominal reflexes		
upper		
lower		
Knee jerk		
Ankle jerk		
Plantar responses		

+ = normal
++ = exaggerated
- = diminished
-- = absent

13. Cranial nerves involved.

14. Date and report of examination of C.S.F.

15. Date and report of examination of blood.

16. Give approximate time after onset when paralysis first showed signs of recovery.

17. Any recent operations and date. e.g. removal of tonsils.

18. Any other member of the family or near relatives who had Infantile Paralysis or a feverish illness at the same time.

19. Any other information which may be of interest and relevant to this investigation.

20. Hospital to which patient was first admitted.

QUESTIONNAIRE TO PATIENT.

"INFANTILE PARALYSIS."

PATIENT'S NAME .... AGE ...

1. Date of onset of illness? ....
2. How did the illness start? ....
   1. Feverish ....
   2. Temperature at onset and for how many days it was raised? .....
   3. Rigor (shivering) ...
   4. Catarrh in chest....
      in nose ....
      in throat ....
   5. Headache ....
   6. Vomiting ....
3. Was the patient irritable and difficult to manage? .....
4. Did the patient have
   1. Sore throat? ....
   2. Pain in the limbs? ....
   3. Tenderness of muscles? ...
   4. Pain in the neck? ....
   5. Pain in the back? ...
   6. Stiffness of back and reluctance to stoop?....
   7. Dizziness?....
   8. Double vision?....
   9. Unequal pupils?....
   10. Jerking of eyeballs from side to side?....
   11. Inability to pass urine or loss of control of the bladder? ....
   12. Constipation?....
   13. Diarrhoea? ...
   14. Jerky movements of limbs? ...
   15. Twitching of individual muscles? ...
   16. Paralysis of muscles of face? ....
   17. Difficulty in breathing (Paralysis of diaphragm) ? ....

18. Insomnia (Sleeplessness)? ...
19. On attempt to swallow, did fluid return through the nose? ....
20. Intolerance to light in eyes? ....
21. Generalised convulsions? ...
22. Loss of appetite? ....
23. Pains in the joints? ....

5. Time between onset of illness and first appearance of symptoms? .....

6. Was paralysis of sudden or gradual onset? ...

7. Did the paralysis increase or decrease in extent within 24 hours of it first being noticed? .....

8. Give direction of spread
e.g. ascending - legs to body and arms ....
descending - back to legs ...

9. Any recent operations with date? ....
e.g. removal of tonsils.

10. Any other member of the family or near relative who had infantile paralysis or a feverish illness at the same time ? .....

11. Any other information which may be of interest and relevant to this investigation ..

12. Name and address of doctor who saw patient .....

13. Hospital to which patient was first admitted .....

NAME AGE

DATE OF ONSET

MUSCLES OF HEAD AND NECK, AND ABDOMEN.	Right	Left
Masseter		
Temporal		
Pterygoid externus		
Pterygoid internus		
Trapezius		
Sternomastoid		
Supra-hyoid		
Longus cervicis		
Longus capitis		
Rectus capitis anterior		
Rectus capitis lateralis		
Scelenus anterior		
Splenius capitis		
Intercostals		
Diaphragm		
Abdominal - upper		
- lower		
- lateral		
Rectus abdominis		
Erector spinae -cervical		
-dorsal		
-lumbar		
Quadratus lumborum		

Strength of contraction.

0 - no contraction
1 - flicker
2 - contraction but not against gravity
3 - contraction against gravity only
4 - contraction against gravity and resistance
5 - normal contraction

NAME AGE.

DATE OF ONSET

MUSCLES OF THE LOWER LIMB.	Right							Left.						
Psoas and iliacus														
Sartorius														
Quadriceps femoris														
Gracilis														
Pectineus														
Adductors of the hip														
Abductors of the hip														
Gluteus maximus														
Gluteus medius														
Medial hamstrings														
Lateral hamstrings														
Tibialis anticus														
Extensor hallucis longus														
Extensor digitorum longus														
Peroneus tertius														
Flexor hallucis longus														
Flexor digitorum longus														
Tibialis posticus														
Peroneus longus														
Peroneus brevis														
Abductor hallucis														
Abductos minimi digiti														
Flexor digitorum brevis														
Flexor hallucis brevis														
Flexor minimi digiti brevis														
Adductor hallucis														
Lumbriacales														
Interossei														

Strength of contraction

0 - no contraction
1 - flicker
2 - contraction but not against gravity
3 - contraction against gravity only
4 - contraction against gravity and resistance
5 - normal contraction

NAME AGE

DATE OF ONSET

MUSCLES OF THE UPPER LIMB.	Right	Left
Trapezius		
Latissimus dorsi		
Rhomboid		
Levator scapulae		
Pectorals		
Serratus magnus		
Deltoid		
Supraspinatus		
Infraspinatus		
Teres major		
Coraco-brachialis		
Biceps		
Triceps		
Brachialis		
Pronator teres		
Flexor carpi radialis		
Flexor carpi ulnaris		
Flexor digitorum sublimis		
Flexor digitorum profundis		
Flexor pollicis longus		
Brachio-radialis		
Extensor carpi radialis longus		
Extensor carpi radialis brevis		
Extensor digitorum		
Extensor minimi digiti		
Extensor carpi ulnaris		
Supinator		
Abductor pollicis longus		
Extensor pollicis longus		
Extensor pollicis brevis		
Extensor indicis		
Abductor pollicis brevis		
Opponens pollicis		
Flexor pollicis brevis		
Adductor pollicis		
Abductor minimi digiti		
Opponens minimi digiti		
Lumbricales		
Interossei		

Strength of contraction.

0 - no contraction
1 - flicker
2 - contraction but not against gravity
3 - contraction against gravity only
4 - contraction against gravity and resistance
5 - normal contraction

Respiratory failure.

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