Poliomyelitis Anterior Acuta,

with the history of a case in an adult,

a special reference to the symptoms and

pathology.

By

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Poliomyelitis Anterior Acuta.

The subject of this Thesis is one that has attracted the attention of numerous writers; it was first described by Underwood over a century ago, but only within the last forty years has it been investigated by such writers as Klein, Duchenne, and Laborte, and doubts remained as to the situation of the lesion, whether in the paralyzed muscles or in the cord, and it is virtually only within the last twenty years that the pathology has been correctly ascertained.

Most of the literature on the subject describes the disease as observed among children, but although somewhat rare, an essentially similar condition occurs in the adult, and a case affecting an adult taking place in my own practice some months ago which was correctly diagnosed during life, I took the opportunity of describing it and making it the subject of my Thesis.

Generally speaking, patients affected with this disease recover more or less completely, or if death results it is usually at some consider-
able time after the onset of the disease, and only the chronic changes of the cord could be noticed. In this case death occurred within four weeks of the onset, and the opportunity of examining the cord at this early stage of the disease was seized.

The history of the case that came under my notice was as follows:—William Colver, aged 21 years, of age, about 5 ft. 6 in. in height, and 11 stones in weight, of healthy regular habits, comfortably situated, working as a weaver in a cotton factory, married with one child, came to me on Sept. 25, 1889, complaining of headache, a numb tingling sensation of the right hand, of being generally out of sorts. Simple medical treatment was adopted, he was ordered to go home, to remain in bed. Next day on calling to see him, I found this numbness was tingling had extended to the left arm, and also to the right leg, his right foot dragged in attempting to walk. On the day following, he had complete paralysis of both arms, both legs, and also different parts of the spinal, intersosseus muscles, his respirations were mainly abdominal. This
Temperature during the two days averaged from 99.8° to 101°. He had associated constant disturbance of sensation. He also complained of pain in the limbs, dull aching pain down the region of the spine. The sensory functions were in no wise seriously affected, and in a few days appeared normal as regards sensation to touch, heat, cold, & the special senses. The organic reflexes remained good, the patient swallowing, retaining control of his powers of micturition & defecation. As regards the latter there was severe constipation for a few days due to the digestive disturbance at the onset. The skin reflex & tendon reflexes were completely lost, & also power of movement of any of the muscles of the extremities.

About the second day after the onset, I had a consultation with Dr. Custer of the Army Medical Staff, & my elicit was diagnosed, and at the request of the relatives a further consultation was held with one of my colleagues on the 11th day of my elicit of the anterior lateral portions of the gray matter of the cord decided upon as the lesion existing.
There was no tendency to the formation of bedsores. He perspired very freely during the first few days of the disease. His mental powers appeared quite unimpaired by his fever of attention, memory, speech, were normal. He became very fretful & irritable; about the 19th day was troubled with restlessness, but his anxiety to recover, his distress as to his wife's & child's future seemed to increase this. The spine was normal in shape & appearance. On deep percussion, especially over the lower dorsal, lumbar area, there was some pain. Bones & joints normal. Muscles became very flaccid, & rapid atrophy set in, especially in the regions of the deltoid, calf, & planta.

He continued in a completely paralytic state for about three weeks, when he commenced to regain some movement, firstly in the right leg, more rapidly in the right arm. On the 25th day after the first symptoms, on account of his poor circumstances & the difficulty of nursing, he was removed to the Victoria Hospital, Burnley, & put in one of my beds, whereon the following afternoon died suddenly, before the Amseion flow could be called.
Nothing of note had been observed during the earlier part of the day to indicate such a speedy demise, & I did not see him after his admission into the Hospital. Permission was obtained for a post mortem which gave no results as regards naked eye changes in the brain, thorax, or abdomen to account for the sudden death. The Spinal Cord, 3 pieces of the ulnar & sciatic nerve were removed for Examination. On section the Spinal Cord appeared normal to the naked eye with the exception of the anterior portion of grey matter of each side which looked fuller & slightly elevated, and different in colour to the rest of the grey matter. Pieces of the cord from the cervical, dorsal, & lumbnar region were placed in weak trichlorate of Potash solution, & forwarded to Dr. Williamson, Pathologist to the Manchester Infirmary to whom I am deeply indebted for kindly making sections & advising me the results of the microscopic Examination which showed inflammation & degeneration of the anterior Commiss of the Spinal Cord.
muscles they supply, as well as fulfilling voluntary and motor reflex impulses.

The case that came under my care illustrates very well the clinical features of the disease in the adult. For the purpose of description, it is useful to divide the clinical history into four periods, as described by Lister, (1) and Cover, (2), viz:  

1st. The Initial Stage.  
2nd. The Stationary Period.  
3rd. The Period of Repression.  
4th. The Period of Atrophy & Deformities.

In this case, however, death took place soon after the commencement of the 3rd or Period of Repression, consequently the Period of Atrophy was never reached.

The 1st or Initial Stage was marked in with headache, general malaise, feverish symptoms, in this case the temperature was not high, it did not exceed 101°. He was very drowsy and lay quietly in bed. There was no delirium, no convulsive twitchings, but he had a thickly fumed tongue, absence of appetite, & other febrile symptoms associated with a dyspneic state. The symptoms noticed by the
patient on the first day were sensations of numbness & tingling in the right hand & on my asking him to grasp my hands with both his, in order to test their relative muscular strength, I found the right quite weak & feeble, whilst the left at that time remained food power. The following day complete paralysis of the right arm had taken place, & the left arm was partially affected, as also the right leg, but he was able to walk dragging his foot on the floor. On the next day or the 3rd from the onset of the disease, he had complete paralysis of both upper & lower extremities. The spinal & intercostal muscles being also impaired, the sequence of the paralysis being the upper limbs first, & then the lower. The functions of the bladder, & rectum were not affected, the patient retaining complete control. There was total loss of the knee joints & ankle joints. At no period was there any anesthesia, in fact, for a few days a certain degree of hyperesthesia existed which appeared to lessen during the course of the disease, especially after the febrile stage.
The 2nd or Stationary Period lasted for about 2 weeks. He continued to be perfectly helpless and the lower limbs could be flexed, extended or rotated, in whatever position they were placed, he was powerless to resist the slightest movement, or raising either of the arms they fell by his side.

Sensation at this stage appeared normal, but both the tendon, spinal reflexes remained affected. The abdominal retained control, but he was troubled with constipation for some days.

The 3rd or Period of Depression. In this case some improvement began about eighteen days after the Paralysis took place. He found himself able to move the right toes, and also to use some of the muscles of the right arm, and this progress was daily increasing until his death, which occurred on the twenty-sixth day from the onset, three weeks after the Paralysis had fully developed.

Death was probably due to the disease extending to the medulla and a complication of the cerebral nerve centers.

The 4th Stage of Deformities was not reached.
Rheumatoid arthritis acute is comparatively
not a common disease among children.
Its synonyms are infantile paralysis, Es-
central paralysis of children, acute atrophic
spinal paralysis of children.
We may accept the same arrangement of
classification of the symptoms as adopted
in the adult variety, viz.
1st. The Initial stage or Period of Invasion.
2nd. The Stationary Period, or Period of Remission.
3rd. The Period of Remission of the paralytic phenomena.
4th. The chronic stage of Atrophy and Deformities.

The principal differences in the clinical
features of the adult, & infantile varieties in
the initial stage, is due to the fact, that in
children the brain responds more readily to the
influence of the fever, which may rise to 103° or
105°. The nervous symptoms are generally more
marked, muscular twitching, epileptiform con-
mulsions, & even delirium being sometimes pres-
cent, associated with the other pyrexia symptoms
of hot skin, flushed face, quick pulse, & feeble
respiration. In other cases, this stage is sometimes
not noticed, the pyrexia being slight, and the
general symptoms as trifling, as to be acciden
to the effects of cold or weakness, until the subsequent discovery of paralysis of some of the limbs clears up the diagnosis. The paralysis is almost always rapidly developed in children, and the essential feature of this disease is, that the total loss of motor power occurs during this stage. Any subsequent change is in the direction of improvement. The extent of the paralysis varies, and generally speaking the whole of the four extremities are not involved, in many cases there is paraplegia, in some monoplegia, & less frequently hemiplegia. It is probable that cases occur in which the nervous symptoms with convulsions occur off the child, & the death is attributed to these and the exact pathological lesion not recognised. Dechevne (3) reports that out of 70 cases collected by him in children, the disease was ushered in with convulsion in thirty. He considered these seizures were probably of the ordinary type which accompany most acute diseases in children. Sower (4) thinks it possible the convulsions are sometimes due to direct cerebral irritation. The severity of the symptoms in the initial stage appears
to have as relation to the extent, and distribution of the subsequent paralysis, which depends upon the extent & situation of the lesion, and cases have been noticed in which very extensive paralyses have occurred, and accompanied by hardly any fever.

Some writers think the inflammation of the cord is a cause of the acute fever, while others consider the fever & convulsions as well as the cord lesion are due to some unknown process in the body. Cases have also been recorded where a primary acute attack has occurred leaving no paralysis, to be succeeded by a secondary acute attack during which the paralysis becomes evident, the interval between the two varying from a few days to a few months.

The Period of Remission is that time which extends from the full development of the paralysis to the time when improvement commences, during this time the function of the muscles is stationary. Its duration is variable, and averages from a few days to a few months, generally from two weeks to six weeks, or two months. The paralyzed muscles
are relaxed and powerless, and the affected limb or limbs are quite useless. The extent of the paralysis varies, it is rare that all the muscles of a limb are affected, but it does occur in very severe cases, and there may in addition, be extensive fasciculi of the muscles of the trunk, - the spinal and intercostal, - the muscles of the neck being principally affected. It usually occurs that only some muscles or groups of muscles are affected. The functions of the rectum & bladder are rarely interfered with, but in those extensive cases where the neck muscles are involved, swallowing may be difficult. The Skin & Ponder Reflexes are almost invariably lost - the latter due to the loss of the muscle centre by the disease in the grey matter of the spinal cord. In small children it is much more difficult than in adults to judge whether there is any loss of sensation or not, but most usually sensory paralysis is absent in all stages of the disease. In very severe cases sensation may not be quite normal, if a paralyzed limb may bear without pain what would be a severe application of a faradic
current to a healthy person.
After the first week or ten days there is a
change in the condition of the muscles which
are paralyzed; they become soft, flaccid, and
undergo rapid atrophy — in those in which
permanent paralysis has taken place the
irritability to the faradic current is lost, and
in the other muscles becomes distinctly lessened.
This was first shown by Duchenne (5) who
regarded it as a rule that all the paralyzed
muscles in which the faradic irritability at
the end of the second week is only very or less
diminished, do not remain permanently par-
alysed, and that recovery of the muscles takes
place more rapidly and completely the less
the faradic irritability is lost.
During this period the muscle irritability to
the continuous current is increasing, but it
gradually lessens as the muscles waste, and
may disappear when atrophy has occurred.
The motor fibres also lose their faradic in-
ritability due to the degeneration of the nerve
fibres, the degeneration of the nerves to the affected
muscles, cause the quality of the muscle
irritability to differ from normal.
the reaction of degeneration, in which the excitability is lost, if the voluntary excitability becomes altered in character, closure contraction occurring as readily, if not more so, when the positive pole is placed on the muscles as when the negative pole is so placed, a contraction occurs more readily when the current is stronger than in the normal state.

The period of Repose does from the commencement of improvement, if may be of some months' duration. Many of the paralysed muscles improve, and in a few cases complete recovery may take place, more frequently groups of muscles become more flabby and atrophic and permanent paralysis results. According to Lewes (6) improvement usually commences in the parts last affected, and gradually spreads until all the muscles have recovered except those to be left permanently impaired, this usually taking place at the end of one to three months. Ross (7) says that when the paralysis is general, the upper half of the body most frequently shows signs of amendment first, the paralysis disappearing
rapidly from the neck, upper extremities, and trunk, and becoming restricted to the lower limbs. Laborde (6) terms this the period of first respiration. After a variable interval of time this is followed by a second period of amendment, which he terms the period of second respiration, and during this some of the muscles of the lower extremities improve progressively, and the muscular power of one limb may be fully restored, while one or more muscular joints of the other limb may be left permanently affected. The muscles that are permanently paralyzed are affected in jumps ascending as they are associated in their action, and not as regards the peripheral distribution of their motor nerves.

Chronic stage of Atrophy & Deformities.

Improvement ceases after some months, and any improvement taking place after six months from the onset is usually extremely slight. The muscles remaining paralyzed undergo rapid atrophy, which usually becomes well marked in a few weeks, and the contraction of the recovered muscles being
unnecessary, resulting deformities ensue. The most frequent deformities occur in the lower limbs, as the muscles below the knee usually suffer more complete paralysis than those of the thigh and buttock.

The two most frequent deformities occurring in infantile paralysis are talipes equinovarus and talipes equinovarus varus. This is due to the fact that the anterior and lateral groups of muscles, viz., the peronei, tibialis anticus, and extensor are most often paralyzed, and the heel is drawn up, and the foot turned inward by the unopposed action of the gastrocnemius, — the contracted muscles, as time goes on, undergoing a fibrous degeneration, with subsequent retraction of their connective tissue, so permanently shortened that the foot cannot be dorsiflexed.

Talipes equinovarus results when the anterior group of muscles and the adductors are affected, — and talipes calcaneus when the calf muscles are paralyzed. This last condition is rare; a talipes varus still rarer. In some cases both extensors & flexors of both limbs below the knee may be paralyzed.
and the patient is unable to stand, and moves about by crawling on the hands and knees dragging his legs behind him. In the thigh the anterior and internal muscles are most frequently affected, viz. the rectus, vasti, adductors, and the flexors cause a certain degree of permanent flexion whilst the thigh is abducted. Larnach (9) states that this deformity is always associated with flexion of the thigh on the body, i.e. flexes spine and legs. The external muscles are often weak, i.e., in walking the child goes way at the hips. The muscles of the spine and those of the neck, diaphragm are rarely affected permanently. Spinal curvature often results from the attitudes imposed by other deformities. Lordosis is the most frequent deformity and occurs when the sacro-iliacis is affected. The sternocostal muscles are affected. Deformities of the upper limb are much less common than those of the lower extremities. The muscles of the shoulder suffer most frequently, the deltoid being the subject of paralysis & atrophy, when the deltoid,

supra & infra. Originati, triceps and extensors are affected, whilst the other muscles of the forearm escape we have what is termed the 'upper arm type' of Remak (10) present, the lesion in the cord being on a level with the 5-6th Cervical roots. In such cases the weight of the arm causes the shoulder to drop, luxation of the head of the humerus readily takes place. The extensors and flexors of the forearm may be affected, as the interossei & thenar muscles of the hand sometimes suffer better singly or in groups.

The muscles that are paralysed always become atrophied, in some cases so much so that the bone may appear just under the skin, while in other cases an increase of the fat in the subcutaneous tissue may give a wrong idea as to the size of the muscle. The bones of the paralysed limbs also undergo an arrest of development, and grow more slowly than in the unaffected limbs, and the limb be subsequently much shorter. A deformation of the osseous structure itself may take place, the long bones becoming thinner, porous, & more easily broken.
some cases little true bone may remain, the osseous tissue being replaced by fat, the epiphyses may also be much smaller & distorted. The joints may become more mobile from relaxation and stretching of the ligaments, and from loss of the support given by the muscles in a healthy state, and also from alteration of the articular ends of the bones. It can thus be understood how easily dislocations may take place.

The skin of the paralyzed limb is also altered, its surface has a thinned mottled appearance and feels cold to the touch, owing to the circulation being slowed which is due to the arteries themselves being diminished in calibre.

Such conditions are favourable for the formation of chilblains and indolent ulcers which heal very slowly.

**Etiology.** Acute atrophic spinal paralysis occurs in both the adult and in children but with by far the greater frequency in the latter. Sower (11) asserts that it is six times more frequent in the first ten
years than in all the rest of life put together, the majority of cases occurring before three years of age. The exact causation is little known, it occurs in both the weak & strong, is most often in healthy children with a good family history. Lower(12) gives heredity as a predisposing cause, if Fraser Stewart(13) thinks it occurs most often in the children of nervous parents, whilst Heine(14) asserts that neither sex nor hereditary predisposition & racial any influence in its causation.

The most frequent & striking cause is exposure to cold after being overheated, and also the convalescence from acute febrile diseases, excitement is also a probable cause. Over exertion of children who have recently learned to walk, is also a probable factor in its causation.

Siddler(15) of Philadelphia first noted that the disease occurred most frequently in the warm months of the year and added that in his cases, four-fifths were seized during the warm months from May to September. Lower(16) confirms this, and
states that two-thirds of his cases commenced between June and September.

The case that came under my observation occurred during September, and the exciting cause appeared to be due to a chilling after bathing; he admitted remaining in the water for a long time, with feeling very cold afterwards. The interval between his last bath and the onset of the disease, was two days.

Pathology. The disease, as its name indicates, is an acute inflammation of the spinal cord affecting the anterior horns of grey matter, and it is usually limited to this area. The extent of the lesion varies, in most cases only the lumbar region of the cord is involved, and sometimes the cervical, or most rarely the dorsal.

The multipolar nerve cells, which are the essential elements of the anterior horns, become rapidly disintegrated and some destroyed, and although some think that the marked process begins in the nerve elements, while others think the nemophlia is first
affected, the undesired result is the destruction of the multipolar nerve cells, and we have following secondary atrophy of their axis cylinders, the anterior nerve roots, and motor nerves, if the muscular fibers supplied by them, — in these cases we have permanent paralysis of the muscles. In others some of the nerve cells are only disabled by the inflammatory reaction, and in these recovery may take place, and resultant recovery of the muscles supplied by them.

Acute atrophic spinal paralysis rarely proves fatal, and correspondingly, opportunities of examining the spinal cord soon after the onset of the disease are rare. Ross (17) in his ‘Diseases of the Nervous System’ says, ‘Unfortunately no observations have yet been made with respect to the disease during the first few days or weeks, owing to the fact that the disease of itself is not fatal.’ The occurrence of a fatal result is also much rarer in the adult than in children, and in the case that came under my own observation, death taking place within a month from the onset, the result of the histological...
Examination is all the more valuable. Schultze (18) has recorded a case occurring in a man forty-two years of age, in which death took place within 60 months of the onset of the paralysis, and in which the anterior horns of the spinal cord were affected; their multipolar nerve cells were atrophied, the left anterior horn in the upper segment of the cervical enlargement was smaller than the right, and reduced to about one third of its volume.

Kahler and Pick (19) have also described a case in a woman twenty-four years of age, in which the right anterior horn was atrophied in the lumbar enlargement and the central parts of multipolar cells destroyed.

Cormin's case (20) recovers a case in which death took place seven years after the onset of the paralysis, and the changes were in the motor ganglion cells of the anterior horns of the gray matter.

Drummond (21) describes a case in a child five years of age in which death took place after a few hours illness. The spinal cord in the cervical region presented a condition of congestion of the anterior horns,
and on microscopical examination, the capillaries of the plexus matter were seen
distended, and there were minute extravasations
with swelling of the neuroglia, and ganglion
cells which were peculiar with indistinct processes.
Since then he has recorded two other cases
(22) of the disease in the adult; — in one
death occurred twenty-four days after the
onset, and in the other, twenty-one weeks
after the onset. In both there was distension
of the vessels of the plexus matter with atrophy
and disappearance of the ganglion cells.
Dr. Charlewood Turner (23) also describes
a case in a child, 2½ years old, in which
death occurred six weeks after the onset.
In this the microscope showed extravasated
blood, often massed along the vessels, and
scattered through the plexus matter with de-
struction of the multipolar cells in the
anterior horn.
In the case under my care, Dr. Williamson
prepared the microscopical sections as
follows: — the specimens were hardened in
bichromate of potash solution, imbedded
in cellodioin; — transverse sections were
Diagram illustrating the area of anterior commissure involved in the lumbar region.
cut, and stained with toluene, aniline blue black, Weigert's stain, osmic acid, etc. In microscopical examination the outer part of each anterior horn of the gray matter was found affected in all three regions - cervical, dorsal, and lumbar. The changes were only slight in the dorsal region, and most marked in the lumbar. The posterior columns of the rest of the gray matter was not affected.

The pia mater was normal except at the anterior part of the cord, where the vessels were dilated, and there was a cell infiltration surrounding them.

On examination of a transverse section of the lumbar region stained with toluene, an area involving the central, anterior-lateral, and posterior-lateral groups of ganglion cells was seen infiltrated with a mass of small round cells on each side. A line drawn across the cord through the gray commissure would practically join the posterior margin of this area, although the vessels slightly beyond this could be seen distended. Similarly, a line drawn through the centre of the
Diagram of spinal cord showing the area of inflammation of the antemortem column of gray matter, its fluid nature, twisting pinwheels, and the blood vessels dilated.
anterior cornua on each side in an antero-posterior direction, would practically limit the internal margins of the infiltrated area. The blood vessels, both in the inner part of the anterior cornua, y around the margins of the infiltrated area were seen abnormally large and dilated, y their lumen was packed with red blood corpuscles. The lymphatic sheaths of the were also seen widely distended and filled with small round cells. The cells in the peri-vascular sheaths, and in the infiltrated area were nucleated, y appeared about the size of red blood corpuscles. The cells were most numerous around the margins of the infiltrated patch. The nuclei of the small round cells stained well with toluidine.

In this region of round cell infiltration involving the antero-lateral portion of the anterior cornua on each side, no nerve cells could be seen in specimens stained with aniline blue black, or according to Weigert's method. At the antero-internal, y the median portion of the anterior cornua could be seen some cells that were normal in appearance, but most were shrunken, especially those...
Rough diagram of microscopical section showing small round cells in the area of inflammation of anterior cornu of spinal cord & its vessels, distinct granular perivascular sheath which are much distended & crowded with small round cells.
immediately around the patches of infiltration
in the specimens stained according to Weigert's
method, the anterior external part of the anterior
horn was stained brownish yellow, and the
same conditions as seen in the Cöpf specimen
stained specimen observed, viz., a mass of ground
celled infiltration with dilated blood vessels,
and the perivascular sheaths of the vessels
also widely distended with the small round cells.
In this region there was a complete absence
of the fine network of nerve fibres, which in
a normal cord occupy the gray matter, and are
stained black by this method. Elsewhere
throughout the gray matter of both the anterior
and posterior columns, this fine black stained
network, showing healthy nerve fibres could
be seen distinctly. A few cells stained
dark black, could be observed scattered through
out the region of cell infiltration. The white
substance of the cord stained well, and pre-
sented a healthy appearance. In the lateral
portion of each gray commissure could be seen
dilated vessels with distended lymphatic sheaths
crowded with small round cells.
The white commissure was normal.
In the cervical region the changes were not as marked as in the lumbar region, but occupied nearly the same portion in the anterior commissure. The patch of infiltration did not extend quite so far anteriorly, but passed rather further posteriorly, reaching slightly beyond a line drawn through the preg. commissure. The cell infiltration was not as marked, and the right commissure was not affected quite as much as the left.

In the dorsal region the changes were slight. The capillaries were dilated, and their perivascular sheaths were distended with small round cells. The cell infiltration at the anterior external part of the anterior commissure was much less marked, and the multipolar cells in this area were chromatophore throughout. Elsewhere throughout the preg. matter they were normal.

There were marked changes in the anterior nerve roots. Specimens stained by Wright's method showed only a few nerve fibers which were widely separated from each other instead of lying close together. Between the
fibres was an infiltration of small round cells varying in degree, in some marked, in others quite slight in degree. Among the nerve fibres were some clear unstained circular spaces from which the nerve fibres had completely disappeared.

Pieces of the anterior roots teased out, showed the white substance of Schwann broken up into irregular masses, some round, some oval in shape, and the nerve nuclei were increased. At some spots both axis cylinder and white substance had disappeared and only the external sheath was left.

The posterior nerve roots were almost normal, only a few irregular spaces where the nerve fibres had disappeared were seen.

The Filum Terminale showed little change beyond dilatation of the vessels, of their fascicular sheaths.

The Ulnar nerve just above the wrist, and the Radial nerve showed numerous degenerated fibres, both in transverse section, when teased.

In later cases of Polymyelitis anterior acuta we have the anterior corona becoming atrophied and shrunken in the
affected area, and an increase of the connective tissue of the grey matter.
In the paralyzed muscles, the muscular fibres most affected lose their transverse strie, and become infiltrated with silk fibres, of the nuclei increased. The interstitial fibrous tissue is increased, and contracts until it becomes cicatricial. Some muscular fibres may only be slightly affected, while others are quite normal.

Diagnosis. During the febrile stage this is very difficult and almost impossible; the pyrexia may suggest one of the B-arthritides, or some other acute disease, and if convulsions or delirium are present, some cerebral disease may be suggested. The development of the paralytic gives the clue to the diagnosis - its acute onset, the rapid development of the total paralysis, symptoms pointing to the lesion being a primary system one, situated in the anterior corner, being the points to consider in clearing up any doubts. Then paralytic occurs
There may be some difficulty in distinguishing between Poliomyelitis anterior acute and Transverse Myelitis of the lumbar region. In the latter we have loss of sensation, paralysis of the sphincters, a tendency for edema to form, and as a rule, no atrophy. In central paralysis there is no loss of paralytic irritability, no muscular atrophy.

**Prognosis.** Poliomyelitis anterior acute early forms fatal. If such a result occurs it is generally due to the severity of the febrile process at the onset, or else due to the extension of the disease, and subsequent involvement of the central nerve centres or to some affection of important organs, as Pneumonia, &c.

**Treatment.** In the acute stage, the treatment is to keep the patient at rest in bed, to reduce the fever, and any urgent symptoms. Aconite, hydrochloride of potassium, and belladonna are useful at this stage. If jaundice commences, irritation of the stomach, mustard poultices to the spine, later on, beeches, &c.
After the initial stage is over, the treat-
ment should be directed to arresting the in-
flammation, & promoting absorption of the
inflammatory exudation. The patient should
be kept in bed, & at rest as much as possible
and all excitement avoided. The diet should
be light and nonstimulating, - principally
milk, butter, & those drugs capable
of producing contraction of the blood vessels,
such as Ergot, Belladonna should be given,
and later on, absorbents, such as Iodide
of Potassium, 1 tonics of Iron & Quinine
or Drops of the Iodide of Iron.
It is desirable as far as possible to
maintain the nutrition of the muscles,
and avoid atrophy, so far as we can, - to
attain this gentle massage of the limb or
limbs should be commenced early, and con-
tinued while the anterior horns are recover-
ing, or later on rotation and traction of
the muscles. The batting should be
used after a month or six weeks, beginning
with a weak current and gradually in-
creasing. Owing to the weak circulation
in the affected limbs they should be
warmly clad, and protected from cold.

In the chronic stage mechanical appliances must be utilized to correct deformities.

If desired, I shall be glad to forward microscopical sections of the spinal cord of the case recorded.
List of Works to which reference is made:
8. Ibid. P. 429.
9. Ibid. P. 432.
10. Archives für Psychiatrie 1879.
114, Westgate, Burnley.

I certify that I have been engaged in medicine since 1875, and have taken the degree of Bachelor of Medicine in 1879.

I have been engaged as a medical practitioner in this place since then, and I am currently the Superintendent of the town.

I certify the above.

John Smith

March 25, 1891.