Notes on a few Cases of Paralysis: A thesis for the degree of M.D.

Before commencing to detail the cases of paralysis which I purpose to describe individually, I think it will be advantageous if I refer to the general conditions or causes of paralysis as commonly met with.

"Paralysis" then may be defined as a loss of power in producing a contraction of the voluntary muscles. It is a sign of impaired action and is due to the imperfect conduction of motor stimuli to the muscles due to some pathological condition in certain parts of the nervous system, either the Brain, Spinal Cord, or in or acting on certain nerves through which motor stimuli are carried.

Paralysis is almost always due to some organic change in the parts concerned in the production of the lesion; but whilst most organic in origin true or one lesion in motor regions of the nervous system there are what are termed "Sensory Paralysis" which are functional in origin which are primarily due to changes in the
the sensory nerves or sensory centres.

Paralysia may be "Temporary" or "Transient" in which case recovery more or less complete occurs, or it may be "Permanent" in which the lesion or the effects of the lesion remain throughout life.

Again paralysia may be "General" i.e. affect almost the whole body, or it may be "Local" i.e. affect a part of the body e.g. a limb, a group of muscles, or even a single muscle.

As regards their origin i.e. the site of the lesion which produces them Paralyses may be said to be:

1. "Encephalic" (including Cerebral, Orbital, Bulbar)
2. "Spinal"
3. "Peripheral".

If we look at the conditions which result from lesions in these respective regions we find:

1. In "Encephalic" paralysia there is often a sudden or apoplecticiform origin. As a rule it affects only one side of the body—very often the limbs face of the same side. Sensibility may be diminished more or less (anaesthesia) on the affected side for a longer or shorter time. Due to "local" power of control over the sphincters of bladder and rectum. The superficial reflexes may
may be diminished whilst the deep may be increased on the paralyzed side. Electrical simulation may not give results differing much from normal. Such a condition is termed a "Hemiplegia."

In "Spinal Paralysis" if the lesion be complete, both sides of body are affected from brim downwards. Sensibility is diminished or altogether absent but at the upper limit of this anaesthesia there is often a zone of increased sensibility (hyperesthesia) producing a gentle sensation or constriction which is supposed to be due to irritation of the sensory fibres at the upper end of the lesion. There is generally loss of control over the sphincters. There is generally complete absence of reflex action in the affected muscles. They tend to undergo rapid atrophy. Under electrical stimulation present the "Reaction of Degeneration."

Bed sores are especially apt to follow lesions of spinal origin due to trophic changes in the skin. Such a lesion as described above is termed a "Paraplegia." If the lesion be incomplete and affect one side of the cord there is produced a "Hemiparaplegia" in which the muscles on same side are paralysed, but in which there is anaesthesia of the opposite side of the body as well as on side of lesion.
III. In "Parietal" paralysis limited to a certain muscle or group of muscles supplied by a certain nerve, if the injury is complete so that the nerve function is entirely destroyed there is rapid atrophy in the affected muscles. Under electrical stimulation there is a total loss of or great diminished irritability of the muscles, or often they present the "reaction of degeneration." As "Reaction of Degeneration" is a common phrase in connection with the behavior of paralysed muscles under electrical stimulation in certain cases, I may use it later on it may be better today what it is meant by it.

In this condition, if the nerve of supply of the muscle be stimulated either by Faradic or galvanic electricity there is no result.

(2) The Faradic current if applied directly to the muscle produces no result.

(3) The Galvanic current if applied directly to the muscle produces a contraction more readily than in a healthy muscle.

With these "Quantitative" there are also certain "Qualitative" changes—e.g.

(4) The contraction resulting from the galvanic current is very sluggish— it is slow in appearing...
appearing of diminished power whilst present, + slow in disappearing.

(5) The polar reactions of healthy muscle are reversed— the anododal resulting when we would expect a cathododal excitation.

In an incurable case there finally comes a time when the muscle itself gives no reaction to electrical stimulation in whatever form applied.

1. Cerebral Paralysis - with cases

The causes of cerebral paralysis are:

A. Acute e.g. 11 Traumatic & lacerations resulting from direct injuries to Brain or Compression due to depressed fractures of the skull; gunshot wounds &c.

B. Haemorrhage on to the surface or into the substance of the Brain as in apoplexy.

C. Embolisms & thrombosis which would destroy the functions of certain motor areas by cutting off their blood supply.

D. Congestions of Brain of severe occurring in motor areas would result in paralysis more or less complete & of longer or shorter duration.

B. Chronic e.g. 11 Summons of any description e.g. Cancer, Gummatia with which may be classed
class Anemia, Hydatid Cystic.

2. Diffuse Tumor of Cerebral Spinal Sclerosis which is a very chronic process.

In the cases I have met in private practice have all been of the acute type.

In one case I was called to see a stout, plethoric, young man who had been seized with sudden severe pain in head with a partial paralysis of the lower limb on one side, but had not become quite unconscious. I bled him from the arm to the extent of ten to twelve ounces with immediate relief. The cause in this case I ascribed to a severe local congestion which the extraction of blood immediately remedied.

Of the cases of Hemiplegia which I have had I shall notice the following as the most interesting:

Mrs. Ackroyd, Cleveley, North. Age 63 yrs.

I was called to see this woman who had been found lying across the hearth in her own house. Her left arm was below her resting on a hot coals which had become embedded in the muscles causing a burn lacerated wound. She was quite unconscious when found made no attempt to speak. On examination it was found that the
the limbs on the left side of the body were paralyzed, the arm on movement presenting slight rigidity. The left angle of the mouth was drawn upwards and outwards, whilst the right side of the face hung lax, evidently included in the paralysis. The head troops were markedly turned to the left side whilst the head could be moved to the opposite side it immediately returned when elevated. The pupils were markedly contracted and did not react to light. The temperature was taken in both axillae several times during the day always showed a difference of 1 or 2°, it being higher on the paralyzed side. She never regained consciousness, the state of coma gradually deepened, mucus began to collect in the air passages, her pulse from being full bounding became small very rapid she died fourteen hours after the seizure.

The points of interest in this case are-

(i) The paralysis of the face on the side opposite to that of the limbs.

(ii) The deviation of temperature at different times on different sides of the body.

(iii) The condition of Left Congenital Arterial Variation.

(iv) Facial Paralysis: In hemiplegia the
The face is often paralysed on the same side as the limbs but it is sometimes on the opposite side producing the condition known as "Cross Paralysis" or "Alternating Hemiplegia." The muscles affected are those of the lower part of the face viz. the orbicularis oris, Buceinator, the muscles drawing up the angle of the mouth - the muscles round the eye & upper part of the face as a rule escaping. With this there is generally some implication of the motor part of the 5th & hypoglossal nerves; this is seen in mastication when the food collects between the cheek & the gums on the affected side, in protruding the tongue it passes to the paralysed side being pushed there by the muscles of its sound side. In facial paralysis due to direct implication of Pons or 7th (Bell's Paralysis) the resulting conditions are just the reverse.

Cross Paralysis is produced by a lesion of the Pons in the lower part of one lateral half - a lesion here may implicate effectant fibres of 7th after decussating, & at the same time effectant motor fibres which decussating in the medulla pass to the opposite side of the body to produce a paralysis in the limbs opposite to that side on which the face is affected.

II. The Temperature: - If the temperature be taken
taken in the two axillae in a case of hemiplegia there will be found a difference of ½ - 2° F. The paralyzed side shows the higher temperature. In Jane's case it was 1½° higher. This is not due to a rise of temperature on the paralyzed side, but, as can be shown by taking the temperature in the mouth, there is a lowering of the temperature of the body generally, this being more marked on the non-paralyzed side, vac- motor changes on the paralyzed side allowing the free circulation of blood, thus causing an apparent rise of temperature on that side.

The rectal temperature (normally about 99.4° F.) shows variations according to the time the lesion has existed. There is (1) a lowering due to shock of the system generally, during which for from 1-2 hours after the onset the temperature is about 96° F.

(2) the temperature then becomes normal, remains so more or less.

(3) if the case tend to become fatal there occurs before death a sudden marked rise in which the rectal temperature may reach 104°-106° F. In a case in which the initial lowering has given place to a rise in temperature this in its turn giving place to a second lowering it is probable that a
a second haemorrhage has taken place producing a fresh shock to system.

III. Conjugate Deviation: This is one of the commonest "eye-symptoms" of Cerebral Haemorrhage. It is that condition in which the eyes are turned both to one side and slightly upwards as the head is markedly turned in the same direction, as if the person were trying to look over one shoulder due to more marked contraction of the sternocleidomastoid of that side. If the head be placed in the normal position it turns again to the side. By an effort of the will the person may be able to direct the eyes to the middle line but is unable to look to the opposite side for relaxing the effort; the eyes return to their former position.

Conjugate deviation may be present in lesions of

1. The Cortex, 2. The Internal Capsule, 3. The Pons.

In the two former in cases in which the paralysis is complete the deviation occurs to the side opposite to that of the paralysis therefore towards the side of the lesion. If the lesion be in the Pons the deviation then is towards the paralysed side away from the side of the lesion.

With this obtains in cases in which there is total loss of function in those cases in which spasm of muscles results instead of total paralysis the exactly opposite results ensue—so that when the lesion is irritative in the Cortex or
Internal Capsule the deviation is towards the convulsed side away from the lesion. If the lesion be irritative on the Dors the deviation is towards the lesion.

The explanation of this difference is found in the effects which the lesions produce in the muscles affected. When the muscles are paralysed the muscles of the opposite side prove stronger and pull the parts to their side.

In irritative lesions instead of a paralysis there is a spasm in the muscles affected, then their action proves stronger than that of muscles of sound side. There is a drawing of the parts towards the affected side and away from the side of the lesion.

Here is then a difference as regards the pros and cons.

Conjugate Deviation according as the lesion is in the Optic or Internal Capsule or in the Dors.

In lesions of the lower part Dors lateral half of Dors there is a "Cross paralysis" the side of the face paralysed indicates the side of the lesion due to the fact that the fibres of the Dorsal would are affected anterior to the decussation of the fibres to the opposite side i.e. it depends entirely on the relation which the lesion holds to the part of the nerve affected. In the same way in Conjugate Deviation the side paralysed according to the relation which the lesion holds to the nuclei of the Dors.
e.g. (2) In lesion above the nucleus of the 6th nerve the eye cannot be directed towards the side of the lesion beyond the middle line (b). If the lesion be in the nucleus itself the external rectus of the opposite eye is completely paralysed so that the eye is turned inwards it cannot be moved at all outwards whilst the internal rectus of the other eye is able to move the eye to the middle line but no further (c). If the fibres of 6th nerve be injured after having left their nucleus the external rectus of that side is paralysed but the internal rectus of the other eye is intact so that eye can be moved to either side. But the nucleus of the 6th nerve is intimately connected in activity with part at least of the nucleus of the 3rd nerve. This is ascribed to a special Co-ordinating nucleus or to special commineral fibres connecting the two by which they act together with them the muscles which they supply viz External Rectus (by 6th) + Internal Rectus (by 3rd) of opposite eye. The preponderance in activity in the production of Conjugate deviation seems to lie in the 6th the deviation varying just as the nucleus is involved or not in the lesion. Conjugate deviation then when present may be of great service in aiding us to arrive at a definite
definite diagnosis as to the site of the lesion more especially in those acute cases in which we find the person profusely somnolent there is difficulty in ascertaining where paralysis actually does or does not exist.

In one case the pupils being markedly contracted there being general left-sided paralysis whilst the face was affected on the right side, the Conjugate deviation being to the left - I think the lesion was located in the Pons of the right side probably towards the lower part. The external rectus of the right eye and the internal rectus of the left eye were paralyzed so that antagonistic muscles pulled the eyes to the left. The left Temporo-Mandibular pulling the head in the same direction.
A case of Bulbar Paralysis:

I was enabled to examine this case thro' the kindness of Dr. McNaughton whose professional aid was sought. I examined him first in March 1866.

Wm. J. Richardson, age 10½ yrs. Walker or lop.

This boy is rather tall for his age, very thin, with a pallid anaemic complexion, & vacant expression of countenance.

History: His mother says he has always been delicate of a nervous temperament. He had 3 or 4 fits in childhood & another about 18 months ago which was attributed to his getting into a passion with one of his brothers. He has been very deaf once.

An attack of "fever" six years ago. During the last 18 months he noticed his speech becoming affected, his voice gradually getting weaker until it was reduced to a mere whisper. Some words he can now utter pretty plainly, others he cannot. When he does attempt to speak the saliva dribbles from his mouth. His pillow is quite wet with it every morning. He was dismissed from school partly on account of his general incapacity for learning partly because he made much a mess of his books with the saliva. He has great
great difficulty in swallowing. The food lodges in
the sides of his mouth, and he has to force it
down with the aid of his fingers. Liquids
require a greater effort than solid food. He
deeps very badly his nervous and restless at night
This leaves him very drowsy in the daytime. He
usually sits "nodding" by the fireside. Sometimes
he falls asleep whilst standing before the fire,
and in consequence he has had several
narrow escapes. He gets breathless exhausted
if he walks any distance. His appetite is
good, his bowels costive, the passes but little
urine. Latterly he has lost flesh considerably.
his weight (clothing included) being only 141/2 lbs.
His father died some years ago from heart disease
and dropsy. He has 3 brothers & sister who are all
alive & healthy. There is no family history of
syphilis or nervous disease.

Present Condition: The patient intelligence is below
the average, but he quite understands the meaning
of questions that are addressed to him. His answers
are almost unintelligible owing to the loss of voice &
defective articulation. I heard him attempt to
read a passage from one of his school-books, but
but it was simply an indistinct rumble. His tongue lies a helpless mass on the floor of his mouth, presents a peculiar appearance. It is raised in the centre along the median raphe, is puckered in at the sides or deeply grooved transversely. He cannot touch the upper lip or teeth with it without the assistance of his fingers. He cannot protrude it when the mouth is open but he can do so slightly when the lips are approximated. He can neither spit nor whistle. The sensibility of the tongue to touch is unimpaired, taste remains perfect. The swallowing of a small piece of bread was a slow tedious process, she had to take several gulps before she could get down a drink of water. The soft palate, uvula, fauces appear normal. He has no pain. Cutaneous sensibility is unimpaired. The patellar tendon reflexes are slightly exaggerated & there is slight ankle-clonus. The pupils are contracted, equal, react to light.

Pulse is 124; Respiration is 22; Temperature 99.6°F.

Heart & lung sounds are normal.

Locomotor System:— On checking him the first thing noticeable is the apparent enlargement of the knee & shoulder-joints. Closer inspection
Inspection however shows that this swelling is merely apparent, not due to any affection of the joints themselves, but to the general atrophy of the surrounding muscles. The deltoids are almost completely atrophied, present the appearance of their membraneous strips or bands, by which the heads of the humeri can be distinctly felt. There is no marked wasting of the muscles of the back of the thumb, or of the interosseous muscles. The ribs are very bare of prominence owing to the atrophy of the pectoralis, latissimus, and others. In the lower extremities, the muscles are so distinctly outlined under the skin as to render the patient an interesting subject for the student of artifical anatomy.

The circumference at the middle of the arm is 5 1/2 in., of forearm 6 inches; of thigh 23 1/2 inches, of the leg 7 1/2 in., of the chest 28 1/4 in.; of the neck 8 3/4 inches.

Urinary System: Urine neutral in reaction. Specific gravity 1026. It contains neither albumen nor sugar, but on standing presents a white flocculent deposit. Phosphates were found.

This was his condition when examined at the beginning of March.

Treatment consisted in giving small doses of
Ptosis of lids, whilst he was ordered milk and a light nourishing diet.
His condition gradually grew worse, on March 15th he had become unable to hold up his head owing to the atrophy of the power in the depressor muscles of the neck. His chin consequently had dropped down to the sternum. He complained of pain over the 7th cervical vertebra, he has distinct tenderness to pressure over that region. The mouth is full of froth when opened presents appearance of being full of soap bubbles. When the chin is raised to the normal position the supra-clavicular region is retracted forming a depression about an inch in depth behind the clavicle — this is probably produced by the feeling forwards of the mammary of the clavicle owing to paralysis of the Rhomboids and Trapezius.

March 26th: Respiration is more embarrassed. With inspiration there is marked movement of the muscles of the neck and abdomen. When sitting he grasps the sides of the chair with his hands so as to keep the shoulders fixed. There is no cough.
April 15th: The face is now expressionless; the mouth hangs open; the cannot close it properly owing
owing to incipient paralysis of the orbicularis oris, the tongue is occluded, atrophies retrogressed as if there were some unseen agency tending to draw it backwards & downwards; the voice is feeble & the articulation very imperfect. The movements in mastication & deglutition are very much impeded but not more so than formerly. His weight is now 47 lbs. There is an entire absence of fat, the superficial veins standing out in bold relief all over the body, the lymphatic glands in groin, axilla are prominent. The spine is very much arched, its natural curve being much deepened. Muscular tremors & twitchings are noticeable all over the face, trunk, etc but seen after the patient has made any motion. The muscles of chest, upper arm are very much atrophied - but all the movements of the shoulder joint can still be performed in a slow manner, tho' the Bicipital & deltoid are so wasted that the shoulder girdle looks like that of an articulated skeleton so plainly can its own formation be determined. There is no flexion or other deformity of hand or fingers. He has perfect control over his thumb fingers.
his usual occupation being "whittling" sticks. He can still walk considerable distances without any co-ordination or ataxia. The breathing now seems to be entirely abdominal the intercostal muscles being almost entirely in abeyance, whilst little help was given in the performance of this function by the muscles of the neck or the extraordinary muscles of respiration.

April 30th: Pulse 130; Respiration 32; Temperature 99.2.

There is a certain degree of irregularity about the thoracic movements i.e. the two sides do not appear to keep time, one side expanding somewhat in advance of the other. The soft palate hangs down and is so slightly sensitive to touch that a finger can be swept round it feel without causing any inconvenience. The mouth hangs open with a foamy secretion between the lips.

May 1st: He complains of pain in the throat, cough; respiratory sounds very feeble; slight wasting of muscles of ball of thumb of the entire intercostal space in each hand. There is a marked depression between the pits due to increasing atrophy of intercostals.

May 15th: Pulse 108; Respiration 24 per minute.
May 19th. Can hold a glass of water steadily in both hands. Cardiac impulse very apparent in 4th and 5th intercostal spaces. The left clavicle is about 1/2 inch higher than the right.

Pulse 140: Respiration 28.

May 21st. Pulse 130: Respiration 28. Temperature 95.4.

Head inclines to the right side. Heart sounds normal. Respiratory sounds almost inaudible in front. With a finger in axilla can feel all round sternum. Joint very distinctly edge of clavomus, artery etc. In order to raise himself from the horizontal to the erect posture he first raises his head from the pillow, then catches his thighs, then slopes the legs on the thighs,腋s his hands behind him, gets up without much difficulty. Electrical reactions normal.


...
June 5th: Very prostrate. Cannot raise himself in bed. About every 10 seconds has a cough, usually the imitation of mucus collected in the throat.

Pulse 126: Resp. 22. Very much amimosed. Temp. 101.4°F.


June 6th: Pulse 128: Resp. 24. Temp. 102.6°F. Cough not quite so incessant, about every 20 seconds. Managed to stand up in bed but with difficulty. Takes no nourishment.

June 10th: Pulse 120. Temp. 101.4°F. Frequent, want to pass water, but little at a time. Palate & fauces quite insensible to touch.

June 10th: Dies of Asphyxia & Exhaustion.

A post mortem examination was allowed & portions of the brain & spinal cord obtained. Sections of these however presented no marked change so that unfortunately this aid to diagnosis was not of use.

I have given a full report of this case because of its interesting nature, & also to facilitate the diagnosis as all these is to depend upon...
in the description of the boy's symptoms.

Diagnosis: It appears to me that there are symptoms here present which cannot be accounted for by any one nervous affection. Here are undoubtedly Bulbar symptoms as evidenced in the difficulty of articulation of swallowing, the dribbling of saliva and the condition of the tongue. So what are the other symptoms to be ascribed? Amongst the conditions at all likely to account for them may be included: Bento hypertrophic paralysis, Friedreich's hereditary ataxia, Multiple Cerebro-Spinal Sclerosis, Progressive muscular atrophy.

As regards Bento hypertrophic paralysis there was never notices any apparent enlargement of muscle of calf of leg or elsewhere. The boy was able to walk without any peculiar gait until the disease was far advanced, she retains the movement of the forearm fingers till the end. There were the muscular twitchings. The patellar reflex was slightly exaggerated with slight ankle clonus. All these seem to point away from Bento hypertrophic paralysis.

The absence of myotonia, the manner in which the symptoms developed seem to exclude Multiple Sclerosis & Friedreich's disease. On the other
On the other hand, the gradual development of atrophy accompanied with fascicu-los becoming more marked as the atrophic process advanced; the fibrillary twitchings and the fact that the muscles or what was left of them still responded to the faradic current. All these are such as would be expected in Progressive Muscular Atrophy. Whether this was the original affection, with secondary Bulbar complication or whether the Bulbar symptoms were primary in appearing cannot be precisely determined as both were present when the case was first examined. I think it probable that Progressive Muscular Atrophy was the major affection in the case, that the Bulbar Paralysis was in this case "a fragment of some more widely diffused nervous disease" as Bristow describes in his book on Medicine (p. 1039). Here he also states that Bulbar Paralysis "is not uncommonly associated with Progressive Muscular Atrophy, generally coming on late but sometimes manifesting itself at an early period" as it seems to have done in this case.

10½ yr. is not a common age to find either affection, but being, almost essentialiy, diseases of adult life.
The exact origin of Progressive Muscular Atrophy has been much debated. Some observers are of opinion that it is an affection of the muscles and that the nervous system plays no part in its production. Others again, among whom are recent observers, agree that the primary change is in the anterior Cornua of the Spinal Cord and consists of an atrophy of the large motor ganglionic cells found there. The attack is not as a rule general, but attacks a certain group of these cells, so followed by atrophy in motor nerves, or muscles, as the atrophy proceeds in the muscles so the paresis develops pari passu.

The lesion seems to be found in certain groups of cells more frequently than in others. Hence certain types of the affection are commoner than others. The most frequent type, just as the most frequent lesion is in the cervical region of the Cord, is that in which the hand forearm suffer—this often begins in the ulnar, the posterior trunicae, or the anterocci when it becomes marked the fingers become flexed contracted and a "claw-hand" or "main-squeak" results.

Sometimes only some of a group of cells are affected, then certain muscles of a group escape whilst others of the same group are paralysed. This same condition
may obtain in any individual muscle some fibres escaping whilst others are included in the paralysis. The case described forms one of the rarer types in which Progressive atrophy is associated with Gluteo-Lumbo-Laryngeal paralysis. In such a case the former may precede, occur simultaneously, or follow the latter. In this case the two affections seem to have developed progressively together. The atrophy of nervous tissue in this case was evidently a very general process tho' there was no history of any hereditary fault. This has been known to exist in very many of the cases which have assumed a general type.

The strange thing about the case is that no pathological changes were visible to the naked eye or to the microscope tho' these were very carefully looked for, more especially as the symptoms during life were very marked & characteristic.

**Special Paralysies**—may be divided into (1) Extra- & Intra-Treaddullary.

Causes of Extra-Treaddullary—(1) Traumatisms (2) Hemorrhage & Palsy (3) Inflammation of hemi-vaso (4) Bone disease, e.g. Caries, causing compression (5) Infiltration of paraffin surrounding the Cord. (II) Intra-Treaddullary—

(1) Hemorrhage, Subtubousc. (2) Syphilitic: acute or chronic (3) Tumors (4) Neoplasms in the Cord.
Cases of Paralysis due to Spinal lesions:

Case of "Infantile" Paralysis:

Alice Lowther, 2 1/2 years old, 9 months, at Durham.

This child was born healthy, but, when 1 1/2 years old, commencing teething, she was taken from her cradle one morning with her left arm useless. It was sent for by her parents who thought "her shoulder was out." I found, however, that the parts presented normal relationship to each other. The left arm being helpless at the side for pinching it the child cries but was unable to draw the arm away.

The child was oligophrenic for a few days but presented no other symptom. That perfect power over her other limbs. At the present time, 9 months after the primary attack the arm is very flaccid but is only slightly wasted. Sensation remains unimpaired, but contraction under electrical stimulation occurs throughout the whole extent of the arm.

No other member of the family has had any nervous affection.

The term "Infantile Paralysis" is often used as synonymous with "Poliomyelitis Anterior Acuta."
but whilst the affection presents so commonly features as in the case described, it sometimes happens that, instead of an arm or leg being attacked in a previously healthy child, a child may be born with some muscles of those of one side of the face paralysed. Hence the term "infantile" is applied to a paralysis of childhood independent of the seat of the lesion of the nervous system. The commonest lesion is that approach to the name "Poliomyelitis Anterior Acuta" - an acute inflammation of the nervous tissue in the anterior columns of the spinal cord which if persistent is followed by softening, atrophy, of the cord with destruction of nerve cells and increase of connective tissue. This may occur generally throughout the cord, often however it is confined to the cervical or lumbar enlargement. When an arm or leg are paralysed in the paralysed muscles there is a loss of tension in the fibre with an increase of the muscle sheath surrounding connective tissue whilst beneath the position of the muscles is occupied by adipose tissue this may be present to such an extent that the limb may appear quite as large as normal. If however the condition remains permanent, the affected limb does not grow at all in proportion to
of the other ten marked cases the person may in adult life have one arm remaining of about the size it was when attacked. The difference in size will of course be more marked the affection comes on before much growth has occurred. It is more pronounced in the upper than in the lower extremities.

The muscles do not tend to become rigid but due to the fact that the healthy muscles are not antagonizes. Infantile paralysis is the real cause of many many non-antagonises deformities.

The Causes of Infantile paralysis may be described as

(1) Pathological changes during mito-uterine life.
(2) Traumatic during labour.
(3) Febrile disorders of shopping-conflict.
(4) Dehydration - this seems to be a very frequent cause so that the period of life between 6-18 months is a very common time for its occurrence.
(5) A local congestion of the nerve centres is probably the cause of those cases in which the affection is transient of short duration.

As regards Results - there is often a partial restoration of function (the muscles or 12, there may be a complete restoration of function (3), destroyed, the muscles may remain permanently paralysed.
There are certain parts of the muscular system which seem to be more frequently affected by this paralysis than others e.g. 

1. The anterior muscles of leg i.e. the flexors of the foot 

2. The extensors of the leg (both the extensors 

3. The extensors of the hand - the supinator 

4. The anterior arm - the upper arm type of paralysis e.g. with the biceps, brachialis, antecubitalis 

5. The dorsiflexors of the foot 

6. The plantar flexors of the foot 

7. The flexors of the leg 

8. The flexors of the hand 

9. The extensors of the arm 

Amongst the resulting deformities, calcaneus varus, talipes equinovarus, are common ones. 

The treatment followed was to be rigorous at first, afterward, 

10. Traction (with or without splints) 

As there is a strong natural tendency to prompt passive exercises as galvanism should be persevered 

with as this may not succeed in restoring full power to the muscles we may at least prevent antagonsistic muscles from having 

full scope to present the tendency to deformity.
which in itself is no mean consideration to the person in after life. In any case in which the muscles react to paralytic stimulation less slightly it may be that is always reason to hope for ultimate recovery.

I shall now briefly refer to a typical case.

William Applewell, aged 40 yrs. unmarried. Cloth weaver. Calvin, Yorks. Complains of weakness of both legs and left arm of difficulty in walking.

Was very strong during early youth, left fatherless at 12 yrs. had to assist largely in keeping small farm at 16 yrs. was earning a man’s wages while a man’s work.

Illness began at about 17 yrs. He noticed first that he was very constipated, had symptoms of gravel on microscopy, feeling about his rectum with frequent micturition, could not retain fluid in his bladder when nature demanded its emptying. Noticed that at night when drank his salt became unsteady, in fact was several times taken for drunk this always almost bellowed. This was present but to a much smaller extent during the day time.

He attributes his complaint to the hard overworked conditions of life. He never had shooting pains. About the same time as his walking became affected he noticed that his left arm began to lose its strength. He could not do his work.
which at that time was all hand weaving, with his accustomed ease. As he has gradually lost almost use of his left arm his right arm continues useful he can write well but after writing for some time his hand jumps about now often she cannot avoid making peculiar flourishes.

From the first also he has had marked feeling of constriction round the waist this is most marked when stripped or exposed to cold air. His condition has gradually grown worse.

Family history: Father died at 48 yrs. death attributed to medicine given by a quack. Doctor has a post mortem examination required but does not know exact cause of death. Mother alive well. Subject very at one time from melancholia. 3 brothers- one alive healthy one dead in infancy, the other dies in the asylum at 21 yrs. One half brother alive well. Sisters- 3 alive well healthy; 1 or 2 dead in infancy.

Present Condition: The body generally is much emaciated, this being more especially the case on the left side of the left forearm is 1/2 inches smaller than the right one, left upper arm is 2/4 inches smaller than the right at its middle. The left shoulder is especially emaciated of the shoulder girdle can be traced almost as plainly as in the skeleton. His is very intelligent despite the facts of this case very clearly, but presents a yellow anaemic, unhealthy
appearance, this is no doubt caused by severe cachexia.
His bowels being morbidly constipated about twice a week, rather oftener after use of an injection, whilst from the same cause he is troubled greatly with dyspepsia but has nothing resembling "gastrie crises".

Motor powers: He cannot walk in a straight line, but reels about would fall if he had no support. Cannot stand without some help even when his eyes are open. He is able to walk outside with a stick, but only short distances and with difficulty. His walk is marked by action and irregularity; he throws his legs about,ậpping them down on the heels first. He can touch anything with his left hand after great consideration, a considerable time, jumping about from side to side. He cannot raise his left arm but on asking him to touch his nose end with the fingers of his right hand he is often wide of the mark. The same happens when he is asked to touch one toe with the opposite heel. On asking him to grasp anything with his left hand there are marked tremors in the muscles which act in a very spasmodic manner, or forcibly expelling the hand. The motor formulae behave in a similar manner. On offering him he has considerable strength in his lower extremities but as he says himself he "cannot use it as he likes." On applying the electric current to
to the muscles of the left arm, the galvanic current produces contractions very readily, but the faradic causes only the faintest reaction — the "reaction of degeneration" is present. The muscles of other parts seem to act normally to both galvanic and faradic currents.

Reflexes: The knee, ankle, and wrist jerks are diminished. No ankle-sole or plantar reflexes. Sensation: He has numbness in the lower extremities sometimes feels as if his feet had no toes. Has no feeling of pain, heat, or cold but has a creeping sensation at times. He can feel perfectly in the upper extremities and can locate place definitely when touched, in the lower extremities, however, this is not so, the difference is especially marked below the knee. When touched near the ankle he refers it to the knee. Often says inner when touched on outer side of the leg rises even. In the same way he cannot distinguish between heat and cold in the lower extremity, everything is merely a touch.

Severe heat is spoken of not as a burning but as pain just as it is a pinch. On prickling him with a pin he says it feels like someone pulling with thumbs and the impression remains for a long time after.

Special Senses: Smell, taste are normal. Sight: he can see clearly, but better towards night. Has no double vision, but the retina seems very sensitive as when an object passes the window or set of eight
right he often sees a second similar one pass in the same way.
On asking him to fix his eyes on an object they move to
the side present a lateral mydriasis. The pupils
vary in size sometimes much contracted this is especially
seen in the mornings, I have seen him several times at
night in daylight pupils has always been more
dilated than the can read much better at night.
The left pupil is always smaller than the right
in daylight presents almost a "pin-point" appearance.

The Angle- Retained phenomenon is present the pupils
not reacting at all to light, whilst to very near
accommodation they react distinctly.

Diagnosis: This case I take to be one of Dissociated
Sclerosis of the Cord of an irregular very chronic
nature. The symptoms which point to the affection
are (1) the age at which the disease began (2) the
exaggerated patellar reflex (3) the mydriasis
(4) the tremors occurring in the arm on voluntary
effort. These are symptoms which are would expect
that are conspicuous by their absence of the rhizitis
which are so generally associated with this form of
sclerosis are not seen here, the absence of ankle-
dromes which usually consists with exaggeration of
patellar reflex, the absence of any mental
mental disturbance, of which if present, would have produced a typical example of the condition. Here are also present symptoms of Locomotor Ataxia as evidenced by (11) The incoordination which was first noticeable at dusk, (2) The gait-Motion which has existed from the first (3) The behaviour of the pupil which presents the angle-Robertson phenomenon which is a common symptom in Locomotor Ataxia (4) The absence of mental disturbance.

In Discminated Ataxia the lateral columns of the cord are the parts in which the change exists. It sometimes happens that other parts are also included when the symptoms vary with the part affected. In the case described the posterior columns are involved, included in the sclerosis, i.e., the marked atrophic condition of the left arm & shoulder seems to point to an affection of the anterior cornu of the side of the cord in the cervical region. The case therefore may be described as an irregular very chronic case of Inular or Discminated Ataxia in which the lateral, posterior, anterior cornu of the Spinal cord are all included at some part of their extent.
Case III. Mrs. Bennet, aged 34 yrs. Trifara. I first saw this patient who was a postlicenciate's wife on attending her during her third confinement. She had a tedious labor eventually requiring instrumental aid this being partly due to the fact that she was very weak and not materially assisted the natural efforts. She made a good recovery and told me then that she suffered a great deal from chronic phrenism that she had not much power in her arms. A few months afterwards I was sent for to see her as she had almost lost all use of her hands and could not support the weight of her head. On careful inquiry I found that she had had shooting pains in her hands and forearms for a few years that she had gradually lost power in them until she could not grasp anything tightly or long at once, so that for some time she had not dared to touch glasses or because of the numbness the let fall broke. As the last the use of her arms they began to waste, rare was much atrophied. She had been under medical treatment for some time at first for phrenism without experiencing any relief, had been told that there was no relief to obtain for chronic phrenism. Her condition has gradually grown worse so that she is unable to
to use her arms at all not even being able to feed herself, everything has to be given to her as to a child. She lies in bed with her head fixed by pillows, her neck being too weak to support its weight, or even to move it into a fresh position—so that at first sight one would think she were a subject of "a Jewel Analysis of the Dream."

Family History: The mother, whom I knew personally, has always been healthy, fairly strong. Her only younger sister, whom I attended during an attack of typhoid, was to all appearances a healthy girl. I got such a strange history concerning her father that only brother that I write to their family medical attendant asking for particulars & the following which I give verbatim.

"The father, a miner, got a slight blow on his head & since then has suffered from headache, giddiness, symptoms almost like locomotor ataxia. The son from a similar accident got enlargement of the thigh, went blind, but can see a little now." These facts, the perhaps not very elaborate, seem to point to a constitutional trait which from seemingly small causes leads on to well-marked pathological changes in the nervous system.

Present Condition: The body generally is in a bad state...
state. Of nutrition—The extremities are always cold and present a cyanotic appearance. The muscles are very much atrophied but show no fibrillary twitchings or tremors when tapped—they react readily to the galvanic but only very slightly to the Paradie current. Present appearance as in commencing reaction of degeneration. Patellar reflex can not be elicited.

Sensation to touch is unimpaired, heat, cold, pain on pinching or pricking are readily discerned. No perfect control over sphincters. There are no rigidity or contractions. Speech is thick sloping at occurring the voice is faint. She has some difficulty in deglutition. There are no irregularities in the conformation of the spine, nor is there any painful spot either on tapping or with the stethoscope.

Treatment—From the family history I came to the conclusion that there was probably some syphilitic taint on the paternal side of the family, directed my treatment in that direction. At first I gave her Jmmary 3/4 dose of Hg Hydrargyrum. Bichlor +10 gr. doses of the Iodide three daily. Afterwards, I gave her the Iodide alone gradually increasing the dose to 20-30 gr. three daily. With this treatment she
she began at once to improve in about two months. 

The gradual recovery of her habitation was able to walk 

assisted, then, household duties better than she 

had done for some time previous. I left the 

district short, after this was afterwards informed 

that after continuing well for some months the 

condition returned不久 shortly in death, made 

symptomatic of Bulbar paralysis having developed. 

Diagnosis: The case one of Progressive Muscular 

Atrophy, Diocesitis, Sclerosis, or Plane Myelitis 

anterior. These being the affection to 

which the symptoms point. 

In Progressive Muscular Atrophy the affection usually 

begins at one part of the arm or leg, then advances and spreads affecting the muscles at one part, and affecting both forearms or both legs at one time. The paralysis is not noticed until the atrophic process develops. In this case the 

paralysis was noticed as the first symptom the atrophy 

follows. Muscular tremors are found in Progressive 

Atrophy but were not visible in this case. The case 

therefore does not seem to be one of Progressive 

Muscular Atrophy. As far as these chief symptoms
symptoms. In this case we may look elsewhere for a diagnosis. It resembles somewhat in the history of the symptoms disso...neurosis but these are not the tremors on voluntary movement which are so characteristic of the affection, the patellar reflex is in abeyance instead of being exaggerated there are neither rigidity nor contraction of any sort.

On the other hand the age of the patient, the want of cerebral symptoms, the gradual onset below characteristic of the disease, the fact that paralysis began at the distal part (first hands became powerless first) that this was followed by atrophy of the muscles in the whole extent of the limbs, the total absence of tremors, spasms and rigidities the fact also that recovery occurred for some time the patient's condition actually improving not due to a mere stoppage of the course of the disease—All these symptoms lead to the rarer Polymyelitis Sitaicuta—as a diagnosis. The condition is analogous to that in Infantile paralysis, the pathological changes being of an exactly similar character producing same results the only difference being that one is an affective of childhood, is acute in character quickly reaches its full intensity—the chronic condition representing the "Infantile" in the adult. In the case described it is
likely that the aneurism has spread to the postero-lateral columns of the cord as this would account for the shooting pains which were present. The disease eventually spread upwards and reached the medulla as the decubitus ulceration were evident. Included in the paralysis, death would be due to asphyxia and gradual manitron from imperfect swallowing. The result of treatment at first the family history point strongly to syphilis as the pre-disposing cause.

Case IV. Edward Remmey, aged 14 yrs, Bipton. This boy was born healthy and remained so until about 2-3 yrs old when he could walk and talk well. Then without any apparent cause which the parents could discern he began to grow weak in his legs that difficulty in walking or standing until finally he was unable to use them at all while they gradually became wasted and deformed. There was no other perceptible change for a few years the parents cannot tell or define when his arms began to undergo the same change until he has reached his present condition.

Present Condition: The boy is apparently tall for his age and fairly intelligent with a massive head-piece, rather heavy expression of face. He talks well and is cheerful. He
He sits up in Sartorial fashion, his person generally being emaciated, all his limbs being very much atrophied. His lower extremities, so far as voluntary power of moving them is concerned, are useless; his upper extremities are in a similar state with the exception that he can flex or extend the fingers of either hand—he is unable to formate or supinate & cannot move the elbow or shoulder joint. There is no rigidity or contraction in the upper limbs, the lower limbs can be moved either in flexion or extension but the feet present both the condition of equino-varus whilst his motor power are thus nil. Sensation remains unimpaired, on tickling the sole of foot he cries out asshole but is unable to remove the foot from the cause. Sensation of heat & cold remains unimpaired. Deep reflexes are absent. Organic reflexes are perfect on setting him on to the floor or in his trying to move oscillations of head & trunk occur. No mental faculties are unimpaired, in fact he is "cute" for having often been told that he is a nuisance he thinks my services have been called into requisition to get him out of the way. He positively refuses to take anything at all from me. Speech & deglutition are normal. His sight is good.
Pupils normal to light accommodation. There is slight mydriasis occasionally.

Respiratory, Circulatory & Digestive systems are normal.

He has a good family history — Grandfather 69, mother 70 alive 70 yrs old. Both in 60's both healthy.  2 brothers, 3 sisters all exceptionally strong. One sister in brother (months) being younger than himself. There is no nervous affection in any branch of family. Symptoms of:

Diagnosis: This case is identical with Dissociated Sclerosis with the exception of the absence of rigidity, the patellar reflex being about instead of exaggerated, these being the chief distinguishing features between this affection & the one to which I attribute the symptoms viz. Friedreich's Hereditary Ataxia as it is termed—this being a disease of early life but not hereditary beyond the fact that it sometimes attacks two or three members in one family but may never have existed in the families of either father or mother.

Friedreich's Disease seems to stand no better with Motor Ataxia, Dissociated Sclerosis—partaking part of both as symptoms so. The pathology also seems to show the anterior ventral lateral columns of the cord are the portion included in the sclerosis. If the condition goes on, it spreads gradually upwards until vital nuclei
The condition of equino-varus was produced by the muscles on the outer tarsal bones, and the joint being far above the heel. These muscles, therefore, by their contractions, bring the foot towards the position of equino-varus. The calf muscle then shortened by their contractions, the foot became fixed in its abnormal position.

A Case of Bright's Disease:

Charles Adams, aged 12 yrs. Kepton. When first I saw this patient I found him feeling after an undiagnosed attack of scarlet fever. He had been playing outside during convalescece, caught a chill, I was called to see him as he complained of pain in small joint, stiffness, faintness, weakness. I ascertained that he was passing very little urine. On examining it found that it contained a large proportion of albumen. He had a considerable amount of albumin. He was suffering from Pet-Solarial Nephritis. For this I treated him "accordum arteriarum." He was recovering nicely, when during this his second convalescence he developed an attack of general acute phrenicums affecting the muscular system very severely. Every moment
or slight touch caused the unfortunate boy severe pain. His temperature never rose above 102.5. But still he did not develop any cardiac mischief was treated with soda, salicylates, diaphoretics, with quinine to relieve the pain. After such a succession of ailments the boy was left very much marinated was so weak that inability to move was not noticed. In the course of two or three weeks when the boy began to sit up it was found that he could not use his extremities at all from the position in which he had lain in bed to which he always returned when disturbed. His three joints had become fixed in the flexed position, the flexor tendons being very tense. The muscles are very much atrophied. He cannot move the lower limbs at all, he can use his fingers which is all he can do with his upper extremities. He flex so cannot be straight. The muscles contract readily but very feebly to the galvanic current.

The Paralysis in this case is due to atrophy of the muscles the result of rheumatic inflammation of their substance. The contracted state of the flexor tendons of the leg, the retention of power in the flexors but absence of the fingers seem to indicate Infantile Paralysis which is about the only other affecting
affection to which the symptoms could be attributed.

Treatment: This consisted in passive exercises, friction with stimulating liniments, electricity regularly applied, and exercise with tonics. 

Syr. Foment. C r. His recovery was an extremely slow process, finding that does not denote the time when I get him admitted into the medical wards of the Christchurch Infirmary. Here his treatment consisted of electricity, hot packs, Galvanois massage, etc. His recovery was very protracted, he regained the use of his arm more quickly than of his leg; he was able to walk in time, his strength gradually returning.

I have thus described cases of Cerebral, Spinal, or, I think correct in the last case of Peripheral Paralysis. The commonest condition found with the paralytic has been that the trophic process of the limbs has been interfered with, or in the first case there was present a certain amount of rigidity.

Trophic changes in paralyses may involve the skin, the joints, or the muscles of a limb.

In some cases of Cerebral Paralysis there is a tendency to the formation of a severe cough in the centre of
& if the gluteal region, this occurs even when pressure is avoided unless the part is kept perfectly dry so as to be purely an atrophic condition of the skin. In spinal cases there is sometimes a great tendency to the formation of an acute sycosis, or this takes the form of a gangrenous inflammation on the sacral region extending to both sides, it is ascribed to an affection of the grey matter of the posterior column of the Cord.

In the joints, there is sometimes present in a case of hemiplegia a subacute synovitis often commencing a few weeks after the condition has become established. In spinal paralyses the most marked changes in the joints are those which occur in connection with Locomotor Ataxia or sometimes called after Charcot, who has studied the condition minutely, "Charcot's Arthopathy." It is of the nature of an acute atrophy of the articular ends of the bones, attacking mostly the larger joints leading to marked deformities even in extreme cases to a dislocation of the affected joints. It is more prevalent in female than male cases of Locomotor Ataxia.

In Muscles: In a case of hemiplegia there is a gradual and slow wasting of muscles, as occurs.
in all un-nervised muscles which is termed the
"atrophy of disuse" in which the muscles continue
to react to the faradic current. The limb may
continue for a longer or shorter time in this state often
there sometimes sets in a much more rapid wasting
in this the muscles do not react so readily to the
faradic current there gradually develops the
reaction of degeneration. This is ascribed to a
secondary descending degeneration in the lateral
columns of the cord as seen in those cases in
which the pyramidal tract has been involved in
the lesion.

In Spinal paralyses atrophy of the muscles occurs in
lesions especially if the anterior columns involving the
large multipolar nerve cells when the influence of
the cells is suddenly cut off rapid atrophy is
the resultant as is seen in Infantile Paralysis - or a
slightly degree shown in its development in Subacute
Post-infectious Anterior or Progressive Muscular Atrophy.

In Cerebral paralyses in early childhood or spinal
cases, as mentioned before, in the case of infantile paralyses,
there is often marked retardation of growth in the limbs
affected. In hemiplage in adults there is a retarded
growth of the finger nails.
There is also seen sometimes atrophy to a slight extent in paralysed limbs both arms or legs. This is a trophic change, i.e., due probably to various causes, to a change in the substance of the limbs which predisposes to the occurrence of atrophy.

What is the cause of the atrophy of paralysed muscles? Disease is a cause leading to a partial atrophy of muscles, partly because of the want of activity which is essential for the maintenance of health in any organ, and also because it loses the extra blood supply which is created by that activity. But if muscles are regularly caused to contract by electricity, the latter may retard the process of wasting it cannot prevent it—so that disease is not the main factor in the process of wasting. That it is not due entirely to nerve-motor influence is clearly shown by an experiment which is described by some authors. The facial nerves receive its nerve-motor branches during its course from its origin to the muscles it is found that if the nerve is divided before or after it has received these fibers, the result to the muscle is exactly the same in either case—the nerve-motor fibers apparently not making the slightest difference. In hemiplegia also there is a nerve-motor paralysis on the affected side leading
leading to hyperaemia. Increase of temperature compared to the sound side— the hyperaemia however do not stay the process of wasting which is due to the direct trophic influence of the nerve of supply.

Rigidity is also a condition frequently seen in paralytic limbs. It may be present from the first occurrence of the palsy, or it may develop at a later period, hence it is described as being of two varieties: Early & Late.

Early Rigidity affects the arm as much as the leg. I saw an example a few months ago in a case of meningiitis of tubercular origin in which I was for some time doubtful as to the nature of the case. Rigidity of the arm was the first symptom to enable me to come to a definite diagnosis. Early rigidity is generally ascribed to irritation of the brain tissue from the lesion or to an inflammatory change round about the lesion. It is found to be very common in certain lesions of the brain, the optic thalami, the corpus striatum, etc.

Late Rigidity generally occurs much or months after the palsy is established. It also is most marked in the arm. It is held by some to be
be due to the restraining influence of the cerebrum being diminished or destroyed so that the cerebellum has its power unimpaired. It is generally associated with a descending generation in the lateral columns of the cord and often presents a greatly exaggerated patellar reflex with well-marked ankle-clonus.

It is generally not so marked in the morning after sleep as it is towards night. Shave at present a case which exemplifies this well - the man was some three months ago seized suddenly whilst smoking with total paralysis of the right side. He had no pain, was unaware of his condition until he wanted to move his arm. He recorded perfectly the suddenness in 20-30 minutes but this was followed by a second attack of paralysis which was permanent. I attribute the cause to an embolus. The temporary recovery was probably due to the clot being washed back in some way. About 6 weeks after he noticed that he could use his fingers in the morning but that they were almost useless at night. Now, possible, moving the arm there was considerable stiffness both on flexion and extension. His patellar reflex was much exaggerated. Ankle clonus was persistent if the foot was pressed near the toes. As a rule the flexors of the arm are affected, in the legs the extensors
and as occurs in primary scleritis the legs are fixed rigid in the extended position. The arm becomes fixed in the flexed condition with the hand flexed also.

In the rigidity of meningitis the legs also assumes the flexed position with the thigh flexed on the abdomen, the leg flexed on the thigh.

Rigidity attacks those muscles in which paralysis takes it never attacks the muscles of the trunk, but sometimes is seen in the muscles of the face. The face is then drawn towards the paralysed side, it looks at first sight as if the sound side were paralysed.

The escape of the trunk muscles is explained by the Breda’s hypothesis in reference to bi-laterally acting muscles viz. that commissural fibres (or special nuclei) connect the nuclei of both sides so that the cerebral influence may be cut off from one side it is transmitted from the sound side of the brain to the nuclei of the sound side by the commissural fibres to the nuclei of the other side so that the trunk muscles escape in this way paralysis, rigidity which attack the muscles of the limbs, face.

Paroxysm rigidity then is due to increased tonicity in the muscles probably from the unrestricted action of the cerebellum when marked leads to
The structure which is a result of the rigidity depends on the same causes for its production.