Clinical and Pathological Observations on Nervous Disease

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by

W. Mackay, M.B., Ch.M.
Devizes, England.

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Three Fatal Cases of Meningitis, Spinal, and Cerebral, following Influenza - with Autopsy.

Case 1. Eliza L., married, thirty-eight, no miscarriages, seven children, one stillborn or dying. One sister died of consumption at fifteen years. The patient had an attack of influenza in July, 1870. This was accompanied by intense neuralgic pains in the back of the neck. The pain extended from the seventh cervical vertebra up to the vertex, radiated downwards to the shoulders, and was sometimes so intense that the patient walked about her room all night. After the pain had continued for a fortnight, gradual loss of power appeared in the right hand; a few days later the right arm became weak; then after a few days she lost the power in both legs, and could not walk up stairs. Further symptoms, with the exception of the pain did not appear at this stage.

I saw her on Sept. 18, and made the following note. "Face pale but well nourished. Little underactivity of muscles. Her face shows no distress, probably diminished on left side of neck, expression anaemic; he,
in her back with the head fixed, can move
it laterally and antero-posteriorly, but with
great pain; this pain is referred to the back
of the neck from the fifth to the seventh cervical
Tongue pale, constipation present. No appetite;
afraid to go to sleep because "She fears she can
not continue if she does.""
On examining the spinal functions the following
condition was found on Sept. 9th.

**Note.** Hands grasp very feebly - weakness on left;
poor on legs also very feeble. Cannot raise
herself in bed. There is uniform loss ting of the
hands and arms on both side, but there is left
marked in the leg. Sclerotics & Faradise incontinently
much diminished in arm, left so in leg. There
is slight resistance to passive movements of the
arm and leg, but this does not amount to rigidity.
No spasm.

Reflexes. Plantar reflex much increased in both
leg; plantar and abdominal not elicited, nor
spinal, trochanter and scapular. Ankle clonus readily
obtained on both legs. Knee jerk exaggerated in
both and obtained above patella, no foot jerk.
Contractures. Muscles totally normal. Pupils
of medium size - light, and accommodation reflexes normal. Coordination and muscular tone apparently unaffected.

**Knee:** Pain is present in the region of the knee mentioned, liable to exacerbation when at rest, and increases on movement in pinnel pain. There is numbness in the tips of the fingers of both hands, and in the outer extemities of the shoulders. She has had tingling in the calves of the legs, but not at present. Sensibility to touch good, except in tips of fingers. There are scattered patches of hyperaemia on the upper and outer surfaces of the arm, and on the flexor surfaces of both thighs. Also some hyperaemia to pin-prick in the fingers, tips, and partially in the palms of the hands. Hot sponges test gave no reliable reaction.

The skin is harsh and dry, and desquamates freely, this being most marked on the left arm. Reaction of degeneration doubt put on Sept. 26th (but on Sept. 24th anal and cutaneous closing appeared simultaneously).

**Vital:** normal. She is four months pregnant.

Patient was admitted into the Droit College Hospital, where, under treatment, the
pain subsided, and the muscles of the arm improved. Obstructive morning sickness however, proved a formidable complication, and this may have been the cause of a recurrence of the spinal symptoms. Oct 22nd Neuralgic pains have returned in former locality. There is a swollen gland at the anterior upper border of the sternum, tender on the right side. Temperature varied from 98° to 101°, but never touched normal."

The patient became gradually weaker and on Dec 17th complained of difficulty in breathing. When it was found that the respirations were chiefly upper thoracic in type.

The following day the diaphragm was paralyzed and the breathing laboured, most of the accessory muscles being in action. The left arm was now completely paralyzed, the right very feeble and the leg also feeble. A consultation was held to consider the advisability of evacuating the uterus, but it was decided that her condition forbade the operation. The following day at 7 A.M. she was examined, and breathing by pneumothoracic farps. She became comatose, and died of asphyxia at noon.
A post mortem examination was made twenty hours later, but permission was not given to examine the cervical portion of the spine. The brain, removed, was the cord, and vertebral in situ. On laying open the bony canal, the cord was seen to be distinctly swollen. The dura mater was injected, and towards the upper part of the preparation, thickened, and opaque. There were no signs of tubercle in the membranes, nor of caries or softening in the vertebral. On lifting up the dura there was found considerable injection of the pial vessels on the posterior aspect of the cord.

On the anterior aspect, the dura was firmly adherent to the vertebral bodies, from the level of the sixth cervical upwards, while on the posterior aspect it was slightly adherent - apparently by fibrous scarring - from the level of the fourth cervical up to upwards. This scarring at the level of the third and second bodies took the form of a tough layer, one eighth of an inch thick, between the dura and the cord, to both of which it was adherent. Still higher, opposite the atlanto-occipital articulation, the deposit reached nearly half an inch in thickness, and the cord was here completely
Air integrated by softening. At the lower level, the cross section was soft and pulpy. There was considerable atrophy of all the left nerve trunks as compared with those on the right side.

It was found impossible by a prolonged use of numerous hardening agents to harden the cord sufficiently to allow of good section, being cut at twice and compared. Some are acid and glycerine preparation however showed numerous examples of disorganized nerve fibre, some shrunken, others with swollen and distorted axon cylinders, medullary sheaths in all stages of disintegration, and abundant darkly stained myelin globules. There were also present greatly swollen, and granular multipolar cells, compound granular corpuscles, and the usual constituents from cubic cells.
Case II. Frank V., single, aged nineteen, had a typical attack of influenza during the third week of November 1891. There had been personal care in the same house before patient was attacked. The acute primary symptoms, headache, backache, sore throat, hoarseness, and constipation, were present and coughed up a bloody expectoration, and he was able to leave his room.

On Decr. 5, the twelfth day, after the onset of the illness, he became delirious, complaining of intense pain in the head. The following day he had hyperesthesia of the left side of the face, flushing of the cheeks, widely dilated pupils, which were equal and reacted to light, and an early obtained tactile cheiroall. There was an occasional transient deep flush over the left cheek.

On Dec. 6, he was admitted to the Driggs Cottage Hospital, and subsequently developed clear symptoms of meningitis of the contrecoup. He died on Decr. 11.

An autopsy was made four hours later at which I was kindly assisted by Dr. Steiner of the Will's County Asylum. The usual appearance of acute meningitis were found, with abundant lymphoid condensation. Careful search was made for typhoid, but without result.
The pons, and medulla were removed for more careful examination. The pia was seen to be intensely congested over both organs, and it was distinctly thickened. The medulla was sliced transversely at intervals, of three sections. There were found on a level with the middle and upper part of the olivary body, three foci of subpial and softening. These occurred at the site of the emergence of the pneumogastric root on each side, and of the hypoglossal roots on the right side (Fig. 1).

The largest was that involving the roots of the right pneumogastric which passed through the softening patch. This measured 6 mm. in its greatest length, was oval in shape, and in its center could be made out in cross section, (not sufficiently clearly indicated in the engraving) the radicular branch of the vertebral artery, associated with the vagus root. The focus of softening on the left side was of similar shape and measured 4 mm. in its greatest diameter. The third patch of softening...
occupying the groove between the olivary and pyramidal bodies, involves the root of the right hypoglossal nerve and was about three mm. in its greatest diameter.

On making similar sections of the Pons in addition to the extreme thickening of the meninges (pia-arachnoid) there was a small blood extravasation which stained the sheath of the Vth nerve on the right side, where it emerged from the meninges.

Microscopical Examination. The patches of softening could not be included in the sections as they invariably disappeared in the process of cutting and mounting, leaving a frayed edge of medulla tissue. This edge took on a deeper Carmine stain than did the surrounding parts.

At the region of the right pneumogastric root there was a complete disappearance in the site of the softening, of the fibers of the stratum zonale, and a disappearance of the axon cylinders from the outer edge of the ascending root of the Vth nerve which here abutted on the margin of the softening patch. The most striking feature in the part of the section, however, was the great prominence both in numbers and size of the small arterioles,
paping inward toward the nuclei in the floor of the IV ventricle. Notably prominent were the two reefs, which run from the anterior median fissure backward, parallel to and on each side of the median raphe. Extreme engorgement was present in all these reefs, with occasional corporeal extravasation into the prevascular spaces; and these conditions were also found in many of the reefs supplying the ventricular nuclei, and in the pons as high up as the pigmented cells of the locus cerebellus; but the engorgement was nowhere so marked as in the reefs in relation with the pulmonary arteries.

Case III. H.H. aged eighteen, unmarried, belonging to a family which was probably pathological, had influenza of the ordinary type early in Feb. 1892.

He returned to work for a week, but went to bed again for headache on Feb. 27th. He was found moribund on the next day in a state of great excitement, with
a flushed face, photophobia, and slight pyrexia.
He was removed to hospital, where his disease ran a course very similar to that of case 2, the chief feature being the greater prominence of motor symptoms, and the meningeal delirium.

Death occurred on March 8th, and the autopsy was made twenty hours later. Dr. Strohm again kindly assisting me.

The thickened and adherent dura, meningitis, exudation in the arachnoid, and injected pia present the usual appearance, and distribution forms in acute meningitis. The pia mater, as in the former case, was found healthy, and the basal ganglia and brain substance exhibited superficial congestion, but were of normal consistence. No tubercular deposit could be detected.

Medulla. The pia here was instantly injected, the surface everywhere being of a brilliant scarlet colour. The injection of the pia mater could be seen extending some distance down the cervical cord, and it was well marked in the medulla of the cranial nerves where these emerged from the membranes, e.g. in the hypoglossal, vagus, accessory, auditory, facial,
and fifth. The medulla on slicing showed no focus of softening, but at nearly all the planes of section were seen the minute striae of engorged vessels, running backward in the direction of the ventricular nuclei. These were especially prominent, as in the former case, in the vicinity of the vagus root. On the ventricular surface of this slice of the medulla, which, four mm. in thickness, included at its lower portion the apex of the calamus scriptorius, there was a small hemorrhagic focus of the size of a dorsum millepedae, in the gray matter, in the locality corresponding to the vago-accessory nuclei. This was visible as a dull spot through the ventricular floor.

The Pons showed considerable congestion of its arteries, in some planes of section, but by no means as much as the medulla.

Microscopically—no change constituted in the appearance of the typical elements of the medulla, but as in the former case, the arteries, in the vago-accessory root-zone, were at times ramifying among the ventricular nuclei, were distended with corpuscular material which in several places had escaped as minute effusions.
One of the largest of these was that in the neighborhood of the vagus nucleus, to which attention has been made. The encephalic roots in several cases attained a calibre of 0.40. No change could be made out in the appearance of the large cells of the nuclei. As in the former case, portions of pia from the orbital lobule, the tips of the temporal parietal lobes, and from the pia of Sylvius were examined by Comil and Baroix's methods for tubercle but with negative results.

Remarks.

As a contribution to the pathology of influenza these cases are open to the obvious objection that the lesions they record are those of menigitis, and not necessarily of influenza. In reply to this I can only state that I can nowhere find a sufficiently detailed account of the appearance of the medulla and pons in cases of ordinary meningitis to enable me to judge how far the conditions I have described correspond with the usual morbid appearances of that disease.

In view however of the opinions held by some observers (e.g., Althea) as to the probability that the chief incidence of the toxic effect of influenza virus is upon the bulbar centers the condition found
in the two latter cases, from the words preceding.
In this connection I am permitted by the courtesy of Dr. Bowes, superintendent of the Welli County
Asylum, to record that similar appearances, viz., cutaneous congestion of the pontic and
medullary pia, with engorgement of the vessels at
the base of the brain, were found in three out of
six cases, in which deaths occurred from
influenza or its complications, without meningitis
being present.

Post-mortem conditions similar to those I have
described have also been recorded, since the above
note were made, by Maidment of Granta, 1 Mackenzi 2, Drabbles 3, and others.

It ought to be borne in mind also, in view
of the conflicting statements which have been
made relative to this point, that hypovasoma
which stops short of actual inflammation is
a vital condition which does not bear transmission
to the dead brain, and that, as Ziegler remarks, 4
"A membrane which during life was hypovasoma
died, may after death show no sign of its former
condition."

1 Maidment, loc. cit. p. 556. 2 id., p. 606. 3 id., p. 723.
4 Pathology, Macalister's translation. Vol. I., p. 44.
Whether the empyema of the bulbus ultimately
show to be a fairly constant concomitant of
influenza or not, it must be allowed that the
clinical symptoms in many cases of the disease -
the rare motor disturbances, genital, pulmonary,
and cardiac complications, the facial neuralgia,
neuroparalytic ophthalmia, etc., which have been
frequently recorded - do suggest peripheral and
medullary disturbance, manifested chiefly in
alterations functions of the vagus, and of the fifth nerve.
Indeed affection of every cranial nerve have
been reported, in connection with the disease,
with the exception, so far as my knowledge goes,
of the fourth, ninth, and tenth.
Two Cases of Tetanus, with an Autopsy.

Case I. Mr. R., a pit. slogger, a rude developed healthy man of sober habits, and with no marked heredity. Complained of inability to open his mouth, and of pain on trying to do so. Also of stiffness and pain in the neck when he tried to move his head, and of some stiffness and pain in the lower part of the face. The symptoms developed on coming home from work the day previous. He had been exposed in his work to a cold east wind at his back - the mouth being opened, and had felt chilled and shivering on coming home.

On examining him, the jaws could be separated voluntarily to about three quarters of an inch. On attempting to depress the jaw further there was pain in the temporal and masseter muscles. The speech was thick and indistinct. The teeth were apparently due to impaired mobility of the lip, as well as to the rigidity of the lower jaw. He could swallow only with difficulty. He would come be any while his corne.

On the following day the teeth could not be at all separated. There was drawing down of the angles
of the mouth, with flattening of the upper lip. The jaws had several times come together with a forcible snap, and the tongue had been lacerated in consequence. There was aching pain in the abdomen and chest, and the lumbar muscles, when he felt them and bent on each side of the spine. He was fed by a tube through a gap in the molar teeth.

Two days later he was in a condition of opisthotonos. The teeth were firmly clenched, and he had frequent attacks of painful little spasm in the dorsal and lumbar muscles.

He was now put on hypertonie injection of curara 50%. Every four hours. After twelve hours, the spasm had relaxed slightly, and he therefore made an uninterrupted recovery, being able to walk out ten days later. In the early stages, he has been treated with large doses of chloral and bromide, but without relief.

Case 24. Mrs. H., aged 59, was always healthy till she had an attack of influenza in 1891. She occasionally suffers from rheumatic pain in the joints. Menopause six years previous.

On Dec. 27th, 1893, she complained of sore throat, stiffness in the jaws, and inability to open the mouth - pain in the right temporo-mandibular
joint, in the elbows, shoulders, and in the right umbilical region, midway between the umbilicus and the anterior superior spine. She had headache, and backache, but had not shiver.

T. 98°. P. 100. R. 40.

On examining her nothing beyond the symptom, complained of ones he made out, except that the umbilicus was displaced slightly to the right of the middle line.

On the following day she was walking with pain in the spot already mentioned in the right abdomen. The pain came on in paroxysms, lasting half a minute, with three or four minutes interval. She was perspiring freely.

On the 29th she was quite unable to open her mouth, and it was aceremoned by palpating through the mucous membrane of the cheek, that this was not due to persistent spasm of the muscles. She complained of stiffness in the leg, but doe not keep them extended and is able to flex them and the thigh, but the latter movements increase the abdominal pain. There is no pain, cardiac nor piti, thorax. She has vomited every day, once or twice. No symptoms. The abdominal walls are hard and firm, the bowel, rest with an Erhoma...
The diarrhea of the umbilicus still continued. 

She was now diagnosed and she was moved to the Royal Hospital. 

On Dec. 31, there was rigidity of the legs, and the back was stiff but not arched. She moved her arms freely. The head was fixed in goniocleisis, kinesic and isometric. The right arm was separated, preferring to admit a spoon. 

There were frequent attacks of tonic spasm with force pain. 

On Jan. 1st, her temperature at 8 a.m. was 63.8°F, pulse and breathing as before; at 8.40 she had a Grove spasm, clutched the mattress, turned suddenly pale, and died. The post-mortem temperature was not taken. The nurse tells that she was taken to a chair immediately that preceded death, persisted long after her removal from the ward. 

An autopsy was made twenty-four hours after death, at which I was kindly assisted by Dr. Wilson of the Well County Ambulance. 

Agon morti, at first intense, had now passed. 

There was much hyperemic congestion on the front of the skin over the anterior tibial muscles on each side, with black discoloration.
Evidently from effused dark coloured blood. Similar marks occur on the exterior aspect of both thighs. On the anterior aspect of the right thigh, below the femoral opening is a raw surface, the tip of a corn plant surrounded by bullae. No other breach of surface is anywhere visible. Thoracic organs healthy. Ventricle of the heart contracted and empty. Auricles semi-dilated, containing dark blood clot. The umbilicus is now in its normal place. On cutting through the sheath of the right rectus abdominis, a mass of dark blood clot weighing four ounces is found at the middle of the muscle. The muscle fibres in the neighbourhood of the clot is softened, and friable. On the posterior surface of the abdominal wall, there is a similar blood clot visible beneath the transversalis fascia on the right side, from which, on incising, a dark clot weighing two ounces was struck out. This occupied a cavity formed by a longitudinal rent, two and a half inches long, by one and a half broad, in the substance of the transversalis muscle, which also is softened and friable.

There is no appearance of hemorrhage anywhere.
in the left abdominal walls.

The intestines were healthy. On removing them a large glistening black discoloration was seen occupying the middle two-fifths of the right Psoas muscle, and an incision into the same sowed a dark colored blood clot.

The clot in the substance of the right transversalis occupies the site of pain complained of in the right inguinal region during life. The in the right rectus is the site of pain complained of in the right umbilical region. The 3 areas, rupture and clot, no doubt account for the pain complained of in fleeting the right thigh.

An examination of the cord was not permitted.

The brain showed marked venous congestion over the convexity and best, most marked over the medulla andpons. The consistence of each hemisphere as a whole was softer than normal. The upper half of each Cerebellar hemisphere was much softer than the lower half.

On section the Cerebral hemispheres showed extreme softening of the central medullary matter; apparently of the nature of white inflammatory softening. The Ventricles contained an excess of turbid fluid. The softening extended
to the basal ganglia (Corpus striatum and optic thalamus), and the ventricular walls, septum lucidum, corpus callosum, fornix, and subcommissural, were all of normal or soft, cream-like consistence. The cortical gray matter, however, preserved a firm的积极性, nearly approaching the normal consistence.

The brain shows a focus of softening in the substance, oval in shape, measuring eight millimeters in its longest diameter, located in the fibers of the pons, to the right of the midline, and a little above the upper pole of the \( V \) nerve. Both pons and medulla were much congested, dilated vessels being everywhere apparent in their substance.

Coronary preparations were made from blood, cerebro-spinal fluid, and softened nerve-pulp, and were stained in a search for bacilli but none of any characteristic form could be detected. Owing to lack of time cultivation experiments could not be made taken.

Remarks. Both cases were examples of so-called idiopathic thalamic, there being no dis corrosive breach of skin surface in either case.
The blister on the anterior surface of the thigh in Case I was found on inquiry to have been caused by a too liberal application of mustard, at the patient's own request to ease the pain, perhaps, at the ilio-psoas insertion.

In the first case the effect of a current of air playing upon the back of a man who was in his shirt sleeves, and perspiring freely might, according to some authorities, be credited with the inception of the disease. But it is noteworthy that both patients had direct association with soil, thefarmer frequently working in a pit, up the sides of which he occasionally clambered; while the woman, a crofter's wife, worked on her own land with the spade, and had done so within a week before the sentence fell in.

The diagnosis in the first case was easy, in the early stage of the second difficult.

The pain, spasm, ought certainly to have put one on the right track, but the sharp and urgent symptom in the case, and that which overshadowed all others, was the abdominal pain. The autopsy revealed abundant cause for her complaints. The cause however in the
Early stages, in its appendicular course, intermittent spasm, and occasional rigors with vomiting, are not early separated from one of renal colic, and the question of volvulus has also to be entertained. I have been able to find only one reference to a similar case, one recorded by Fagge, in which pain in the right hypochondrium, attributed by the patient to wind, was the main feature in the course of the disease.

The unilateral distribution of the abdominal lesion is also noteworthy: the limbs being symmetrically affected.

The direction of the umbilicus to the side of the pain might in a similar case be a sign of diagnostic value, as pointing to true spasm of the transversalis abdomen.

\[\text{Practice of Medicine, Vol I. p. 776, footnote}\]
Case I. Ellen W., aged twenty one, unmarried, was admitted into Down Cottage Hospital on March 24, 1871, for weakness of the lower limb. This had been coming on since she was eight years old, before which time she had been able to walk long distances. She had measles at eleven. She remembers that at the first onset she used to stagger in walking. As the weakness increased she would sometimes fall to the ground. For the last three or four years she has been able to get about the house only by supporting herself by the table and chairs. She complains much of giddiness. She complains too that her hands are more clumsy and weak than they used to be, and that this has come on within the last twelve months. She is the youngest of a family of eight. Four brothers are quite healthy. One sister died of "paralysis" (see Ann W. infra), at the age of nineteen. Another brother, the third youngest of the family, now suffers from paralysis (see Horatio Thomas W. infra.), and
I was bedridden at home. The father is a healthy man of sixty-two, with no appearance of syphilis, or alcoholic excess. The mother, age sixty-four, is blind from double cataract, and for the last two years has been bedridden from rheumatoid arthritis. Nothing in patient's habits or surroundings has any bearing on her case.

PRESENT CONDITION

She is an acemic girl of middle height, well nourished, with a cheerful but vacant expression. She is rarely moved to laughter of a silly uncontrollable kind. She gets out of bed with difficulty and cannot stand except with assistance. When erect she stands with the legs extended and apart, and may so that she would fall if unsupported. In standing, the sole of the feet are inverted, in the position of talipes varus. On trying to walk there is extreme incoordination in her efforts. The feet are drawn forward, raised with difficulty, high from the ground, and planted anywhere, with a jerk which threatens to throw her down. The toes are overextended in the effort to stand, and all the extensor tendons stand out strongly on the dorsa of the feet. There is a tendency to
the hands then at rest, to extend, in at the
metacarpal, p. phalangial, and flexion at the
inter-phalangial joints.

**Nervous System.** There is fair power of resistance
to passive movement; in both legs, the right being
the weaker of the two. Much better is the motor
power of the muscle of the trunk, neck, and arms.
Motor ability to Galvanic and Faradic Currents;
not unaffected, and there is no R.D. There is talpa,
equino-varus of both feet. The phalangia of the
feet are strongly flexed (See Figs. 14, 15, Appendix.)
The foot is shortened, and the metatarsal more
arched than normal. There is no rigidity or spasm
on passive movement. The plantar reflex is
excitable in both limbs. No ankle clonus, nor
knee-jerk. Rectal, abdominal, and
sacralspinal reflexes are present. Deep, equal,
active to light and accommodation. Bladder
reflex normal, but there appears to be slight
impairment of the sphincter of the bowel.
Coordination is markedly impaired in the
left, and arm. She cannot write or read, though
formerly she could do both. An attempt to
draw a circle with either foot Causing a strain
of traumatic jacks. The muscular tone is also
unpaired. She cannot, with the eyes shut bring the tip of the forefinger within three inches of the tip of the nose.

She has never had pain, numbness, nor tingling in her limbs. Cutaneous sensitivity is everywhere normal.

Her special parts are normally acute.

There is considerable lividity of the remainder of the lip, and there are distinctly cold to touch. There are no trophic signs. The optic discs are healthy.

There is a well marked lateral curvature, with convexity to the left, but no pain on movement of the spine. There is considerable twitching of the angle of the mouth when she is articulating, and this become more marked when she is slightly excited. She attempt to depress the angle of the mouth and lower lip, exaggerates this twitching to a clonic spasm. There is frequent bilateral, horizontal nystagmus and no choked movements of the limbs. The speech is imperfect, being nasal in tone - (no palatal paralysis), frequently "chipped" and the words brought out with effort accompanied by a facial contortion. This is evidently due to incoordination of the
articulatory muscles.

Her memory is good, and her mental state clear but emotional. Menstruation is deficient, bowels sluggish. She is much troubled with palpitation. The other systems are normal.

Case II. Henry Thomas, an married, aged 26, is the brother of the preceding, and the third youngest in the family.

Patient was a healthy child till his sixth year, when he had a bad attack of measles, and afterwards of scarlet fever. Within a month from leaving hospital after the latter complaint he began to complain of weakness in his leg, and to stagger in walking.

He could sit about however, becoming gradually worse until his twelfth year, and about this time his mother noticed that he staggered about, stumbling, with his head carried well in front of his body. After his twelfth year the lower limbs became helpless, so that he was unable to move about. Not years & later the hands and arms became weaker, and since his twenty-second year he has been almost entirely helpless in them too. His voice
unable to dress himself, and though he can roll over when in bed, and raise the trunk on his elbows he is quite unable to get out of bed unassisted, and is carried about by his father.

Preliminary Condition: Patient sits in a chair, with his hand and arm, resting in his lap, and his chin depressed on his sternum. He can still partly raise and rotate the head. His depression would be cheerful but for the constant grimace produced by the contraction of the occipito-frontalis, (the two halves of which contract separately) the orbiculares oculi, st. oculi, levatores anguli oris, and zygomatici. The hands are feeble, but not in mora, hyper extended at the meta carpo-phalangeal, and flexed at the inter-phalangeal joints. Any attempt to straighten the fingers by passive extrinsic caus, a co. extrinsic flexum at the wrist.

The spine shows a well marked lateral curvature with the convexity to the left, and there is a compensatory rotation of the pelvis. The recti-abdomini muscles are strongly contracted, and stand out as prominent ridges in the middle line anteriorly, and he is quite unable to stand up the spine.
The thighs are flexed on the pelvis, and the leg on the thigh, and externally, as these joints is impossible. Both knees are adducted and in contact, being fixed in a condition of genu valgum, the left overlapping the right. From the approximated knees the legs diverge to either side. (See Fig. 3. Appendix) so that knees are widely apart. The latter, are in a condition of intertine talipes, equino-varus. There was still some grasping power in the hands, and a fair amount of power in the muscle of the shoulder and elbow joint, but abstraction at the shoulder and external at the elbow joint were extremely difficult in range, owing to muscular fixation. All the limbs are much shortened in size, but what muscle remains i firm in consistence. There is no R.O. present, but irritability is diminished to both curricula.

All reflex, exhalent, and deep, are abolished but the pupil reacts to light and accommodation, and control over bladder and bowel is perfect. The speech is so affected as to make him frequently unintelligible. The words are brought out with an effort and occasionally interrupted as if by a hiccup. The effort to speak brings
m spasmodic twitching in nearly all the facial muscles, some of the mouth, the orbiculars of the eyes, and the mental muscles being most affected. With these jerks there is frequent oscillation of the eyeballs, but this is slower in time, and wider in range than one is accustomed to associate with nystagmus. These oscillations are sometimes bilateral, (conjugate), sometimes unilateral. They are generally horizontal in direction. Sometimes, oblique, but never vertical. Like his sister, he is easily provoked to prolonged laughter.

Coordination in the upper limbs is much impaired. His muscular power is also impaired, but sensation to touch, pain, and temperature are normal. There have never been lightning pains, nor depauperation of any sort. The spieral nerves are acute.

The extremities of the upper and lower limbs are cold and livid; the skin of the hands is thin and smooth.

By indirect examination the discs are seen to be healthy.

He complains that his left eye is weaker than his right, but this seems to be due to the fact
that he has lost control over its movements. Thus, when told to "fixate" on a finger, he is unable for some time to bring his line of vision to bear upon the object, and in the effort, internal strabismus of the left eye occurs.

When both eyes are fixed upon the object, if the right is suddenly occluded, deviation of the left takes place, and this may be either inward or outward. He then loses the finger temporarily, but is able after a few moments to fixate on the object again. This difficulty is chiefly with the left eye, but occurs slightly with the right eye. The ocular movements are normal in range, but jerky, in all directions.

His memory for both recent and remote events is good, and his mental capacity is above the average. Every physical effort brings on an attack of palpitation, and he suffers from persistent headache, which, however, never goes on to nausea or vomiting.

He has occasional boils, and attacks of bronchial asthma during the cold months. His general health is good.
Case III. Ann W., sister of the preceding, died at the age of nineteen years. She was a healthy child at birth, and until her fourth year, when she had measles. Very soon after recovering from this she became weakly and began to stagger in her walk. Three years later she developed a festinating gait. She could move about with difficulty and frequent falls till her tenth year after which she had to be carried. At this date she could see and use her arms, but six years later, in her sixteenth year, the upper limbs began to get weak, and this increased steadily until her death. Before her death she was absolutely helpless as regard movement of limb, head, or trunk. Three days before death she became unable to speak, though able to swallow to the last.

She always had control of her bladder and bowels, and her senses, and mind remained unaffected throughout. Her face and hands in the later stages had the same deformity that her brothers now have.
An Isolated Case

Mabel B., aged twelve, was first seen in Nov. 1893. She had lumbago in her lower back and ankles, with slight pyrexia. Two days later she complained of precordial pain, and a soft systolic internal murmur appeared. For this, which remained after the joint symptoms had cleared up, she was detained for some weeks in bed. Towards the end of her stay in bed, some jerking of the facial muscles, of the head, and of the arm was noticed, which, with the antecedent rheumatism, suggested a suspicion of chorea. The twitching movements however differed from those of chorea in being slighter — shorter in time, and of continuation in range — than choreic movements generally. On getting out of bed, later, marked incoordination of gait was noticed, and ataxia diagnosed.

Family History. She has one brother, ten years of age, and two sisters, one four, and one three. All are quite healthy and have normal height.

The mothers child-bearing history include, a still-birth child nine months after marriage, and a still-born child seven years later. The other four children were healthy.
at birth. The mother's family are said to be all healthy, but one cousin of the patient died at nine years of age from "heart disease, chorea, and convulsions." The father shows no signs of syphilis, is a temperate man, and his family exhibit no history of a neuritis. The patient has always been a delicate child, had pneumonia at six years, but no illness thereafter until the choreatic attacks mentioned above. She has never complained of pain anywhere. She has not menstruated.

On admission into Drury Cottage Hospital in February 1874 the following was her condition: a pale thin child, with a somewhat anemic countenance, but cheerful, and more or less emotional. She lies in bed with the leg extended, the toes pointed, and the dorsum very prominent, the pontine Warmbier's Talipes Equinus (Fig. 4). There is a slight form of the corrugator, which is persistent, and occasional twitchings of the angles of the mouth, lips, and side of the nose. There is an occasional slight tremor of the head, and of the arms, in the direction of a unilateral shrugging of the shoulder, or extension of a forearm. On making her follow the finger with her eyes,
sharply to the right or left there is slight but distinct lateral nystagmus. There is no lateral curvature, but there is exaggeration of the normal anteroposterior curvature in the dorso lumbar region.

Sensation is everywhere normal to touch, pain, and temperature. There is a doubtful patch of impaired sensation to touch on the outer side of the right knee. There has been no subjective pain at any time. The special senses are normal, and so is the muscular tone.

Motor power in the leg and arm is good, a thorn by resistance to pinch is movement. The muscular nutrition is everywhere good, and the tonicity is normal.

The plantar reflex is absent in both legs; the abdominal and epigastic are present. The knee jerks are absent. There is no ankle clonus.

Coordination is markedly impaired in the legs. She walks with the head well forward, and the legs widely separated. There is frequent staggering in her walk, and this is so marked when she tries to turn around that she would fall if unsupported. She has an ataxic, but not the high-stepping gait of locomotor ataxia.
Romberg's sign is present. She executes fine movements fairly well with the arms, e.g. knitting and sewing, but on making her write, there is a termn, and a lack of coordination in the formation of the letters, which is absent from her writing of a year ago.

Electrical irritability to both currents is normal. There is some hesitation about her speech intermmed to read, but it does not amount to "stuttering speech."

The pupils are equal and active both to light and accommodation. The dures are normal, and there is no contraction of the visual fields. There is no ocular pain.

She complains of tinnitus occasionally.

She has a visceral "toxic" murmur.

Remarks.

Friedrich's disease, though much more frequently recognized of late years, is still sufficiently rare to warrant being recorded, more particularly so in the case of this isolated type of the disease.

Of the family type the three cases first recorded are typical examples, and a reference to the
genealogical table illustrates the character of the disease, which in this instance is consanguineous rather than hereditary. Thus the family exhibits through five generations a clean bill of health in every main branch, but one. The disease stocked in the family is traceable to one marriage that of the paternal grandfather, Ralph W., with his second wife, a blood relative, though the exact degree of kinship cannot now be ascertained. It is unwarrantable however, to conclude that the actual consanguinity of the contracting parties constituted the "four and signify male," because the disease also appears in a great nephew of the wife viz. in John, the third grandson of her sister (name unknown). Thus it will be seen that of the seventeen grandchildren who are the issue of Ralph's second wife, and her anonymous sister, actual ataxia probably appears in four of the grand cousins, ocular paralysis, with "clumsiness" i.e. incoordination - in two, and acute atrophic paralysis in one. The probability therefore is that the paternal grandmother of the patient has the Channel by which the taint was introduced into their
family, though it is possible that a tendency, otherwise latent, may have been stimulated by the blood relationship existing between the parties to this union.

The occurrence of anterior polio myelitis in the case of Young W. the second of the anonymous sisters' grandchildren is of interest, as the association of this disease with ataxic families has been observed before. Thus in the case of the Blattner family published by Buttinseg (Virch. Arch. 1883) it will be noticed that Karl, the ataxic in the Blattner family branch, had a maternal uncle paralyzed in both legs from anterior poliomyelitis.

Another point to be noticed in this family is the sequence of the disease, in two of the cases immediate, to attacks of measles.

Acute febrile attacks have, of course, long been recognized as a factor in the etiology of the disease. An anterior scol. fever has been recorded in one case by Buttinseg, in two by Rigid, and in one by Arness. Measles in a similar relationship has been reported by Cazenave, and in a recent case by Macrauzie (American Journal of Med. Sc. p. 74). The role of measles as a neurotic, producing the
received considerable attention during recent years, ménigitis (1), meningitis, cerebral (2) and spinal (3), various forms (4), acute ascending paralysis (5), peripheral neuritis (6) and other nervous sequelae having been observed to follow it, and it would not be surprising if the cro-lain of Friedrich's disease were occasionally found to have a similar antecedence.

The condition of the extremities in these cases resembled, in the hand, the mass in puffs of progressive muscular atrophy, or opulna, ulnar, and in the foot the talipes, which frequently follows Infantile Paralysis. It is probable that in each extremity it is paralysis of the interossei which leads directly to the deformity of the fingers and toes.

The final distortion produced by the perpendicular action of the flexors of the head, trunk, and limbs, as seen in the case of Henry W., was at a degree which I think is seldom equalled by other paralyzing diseases.

In the Isolated Case of the Disease rendered normal points of considerable interest occur.
One is the rapid onset and progress of the disease, and the early stage at which it had arrived.

(2) I have notes of four cases of meningitis, non-tubercle, and apart from carcinoma, occurring during an epidemic measles epidemic.


(4) Current. ib. and Coleott. ib. i. 87. p. 71.


(6) Moura. ib. 1884. p. q. 19. who gives reference to rose and rum, and to hop.
When a diagnosis was possible. To the point I will presently recur.

Another is the history of acute xanthomatosis. This association also has been noted before. Notable exaggeration of the signs caused by acute xanthomatosis occurred in a case of Lasancei ("Britt.; 1890); and in the interview of Friedrich's original "case of acute ataxia, commencing at 13, was aggravated by acute xanthomatosis occurring at 14.

Internal xerostomia has also been previously recorded in connection with hereditary ataxia.

Another item of interest in this case is the absence of the plantar reflex, this being noted as increased in most of the reported cases.

Of greater clinical importance however is the question of diagnosis, in the early stages of the disease from Chorea. Of the desirability of this being made it is unnecessary to speak, for though Lasancei is of opinion that Chorea should be considered as belonging to the first stage of Friedrich's disease yet the importance from the stand point of prognosis, of this being kept in mind, is supreme. That it is a real difficulty
appears from the fact that observers such as Kasme, Mowen, Ormsby and others have extended their mistake in this direction. And
still more confusing does the distinction become when, as in this case, the condition of the patient was complicated by a mitral murmur and a history of Rheumatism.
In the above case, chorea, ataxia, Cerebellar
laceration, and Multiple Sclerosis being already
excluded, I relied on the following points for
making the diagnosis from Chorea.
1. Gait. Motor co-ordination is the

disturbing cause in both diseases. In Chorea
however the inco-ordination takes the form of
jerk, or spams which prevents the intended
movement being carried out by introducing
involuntary dyskinesia. Thus the equilibrium
of the body when in proper form is disturbed.
In the case of early ataxia described, however
the balance of the body was impossible quite
independently of any muscular jerk, taking
place. The swaying was manifestly due
to an interference, a steady and persistent
interference, with the motor impulse to
those muscles, the tone of which is necessary
to equipoise, and this interference took place in a quiet, if, so to speak, uninterrupted manner which was distinct from the active jerking which prevents equilibrium in Chorea. In the later stages of Friedrich’s disease the unsteadiness to Chorio spams is, I admit, much more than I have described, but in the early stages, in which the diagnosis is both most difficult and most desirable, the distinction I have mentioned can, I think, be upheld. One of the so-called ‘equilibrium under the condition, described above, the characteristic gait of the patient — the legs broad to obtain a lower base, the balancing arm, held apart from the body, the stooping back, and protruding head, the hurry, as if to bring momentum to the aid of equilibrium, and the frequent reel and stagger as if sick or intoxicated. All these features are distinct from the shuffling, irregular, jerky, and frequently slow, gait of Chorea.

2. Kempter’s sign. I have never seen this typically present in Chorea. The chief static equilibrium may be disturbed by a sudden lurch, due to muscular spasm, but in the
intervals between them it is able to balance. In the early stage of Friedreich's disease, which precedes a fall, it seems to occur without any muscular movement disturbing the equilibrium.

3. The twitchings. These were left suddenly in occurrence, slower in duration, and less wide in range than choreic spasms generally are. They more resembled a muscular tremor. Thus, some fibres at the angle of the mouth would twitch, or the anterior fibres of the trapezius would contract, but this never affected a complete muscle in its whole extent, still less a group of co-ordinated muscles. The hand was never supinated, or the arm abducted or rotated, as in Chorea. Moreover, the movements were not increased by excitement nor by voluntary effort. I am aware that in the later stages of Friedreich's disease this latter distinction does not hold good.

4. The Reflexes. Although diminished excitability of reflexes has been pointed out by Broadbent as occurring in Chorea (Brit. Med. Journal. 1869), I have never seen it recorded that this may reach the extent...
of complete abolition of the deep reflexes of the lower limb. The absence of the plantar reflex in this case was, of course, exceptional.

5. The nyctagmus. Irregular ocular spasm may, and do occur in ataxia, but I have never seen in the latter typical, bilateral, conjugate, symmetrical, nyctagmus.

6. The distribution of the spams, and of the incoordination. The hand, arm, space are most frequently the seat of chronic spasm and it is in the upper extremity that incoordination is most often absent. Chorea seldom assumes either a regard, spasm, or incoordination, what might be called a "paraplegic" distribution that is tending, affecting the leg more than the arm. The contrary, however, was the case in the ataxia. The amount of incoordination was inversely as the amount of spasm, being most pronounced in the motility of the legs. The hands, moreover, were practically free both from spasm, and from incoordination, and the distribution of the latter was of a "paraplegic" type.

7. The condition of the feet. The presence of muscular deformity, with incoordination,
A prenatal cause.

In a child, would always raise the suspicion of Friedrich's disease; pointing an \( \text{...} \) to the probability of structural cond-m-change. It seems probable that some atypical cases of Charcot's disease which have been described, have been instances of Friedrich's disease.

Mr. D. Uristone has rendered a case (Page "Practice of medicine", Vol 7, p. 734) in which there was a degree of rigid flexion of the hip and knee joints, with overlapping of the knee, from preponderant action of the adductors of the thighs, and a tending to talipse, equino-varus. In the absence of further data, it is not possible to form an opinion, but if their discussion be compared with the photographs of Case 2 in the Appendix, one may be pardoned, with all deference to a great physician, for doubting whether a similar process was not at work in both cases.
Paraplegia following
Sensory Irritation.

Mr. M., an anaemic woman of 23, consulted me in Feb. 1887. I found she had a firm, smooth, the size of a large coconut, attached by a short pedicle to the left labium majus, and ulcerated by chafing. This was removed by thermo-cautery, on Feb. 3rd under an anaesthetic. The patient did well for a week afterward, papered, walked freely, and had, for the first few days only, slight pain in the site of the wound. On Feb. 11th she had symptoms of cystitis, and on the 12th the urine was ammoniacal. On the 15th she had in addition, constipation and piles.

On the 16th, retention. On the 17th, she complained weakness in the lower limbs, and could not turn easily in bed.

On the 18th, the following note was made:

"June 12. Neuros. 18. There is weakness of both feet and ankles, extending up the legs, but stopping short of the knee. There is sensory and motor paralysis of both legs, and a tract and pain is complete over the lower two thirds of the leg, and is partial to a
Short distance above the knee. There is no zone of hypesthesia where the anaesthetic is in contact. Knee jerk is absent in both legs. There is no plantar response. The retromedial are pale and cold. There is complete retraction of urine, and the patient says she has no control over the bowels, the apparent of the process entirely having caused an involuntary evacuation."

On the 29th the skin over the pectorum became discoloured, but under treatment this disappeared without a lesion forming. On testing her electrical irritability it was found that the paralysed muscle of the leg reacted to both forms of current equally with the arm, which were unaffected.

It is noteworthy that the patient at no time complained of pain in the back or the limbs, that she had no muscular spasms nor twichings, and that with the exception of a slight rise in the appearance of the eye, her temperature remained normal throughout.

On March 17th her condition was as follows: "Sensation has gradually returned to the legs, but it is still unimpaired. She can move the limbs freely, and with a little assistance..."
can walk about the room. Plantar reflex and knee jerk are now present, but the latter is diminished. The limbs are wasted, but there has been no rapid atrophy. She can evacuate the bladder, but cannot voluntarily retain the urinal. The stomach is in the same condition.

On June 14th, 1890, three years after the attack, she wrote: "I have improved much since I came out of hospital." (She had spent ten weeks in a London hospital for medical treatment of the bladder) "but still I cannot hold myself properly either way." Her last report, dated June 1892, she has at last acquired complete control, was married, and has passed through a normal labour.

Somewhat similar to the foregoing, is the case of a child E.O., aged 6. The mother's complaint was that he was unable to keep himself clean in either way, being "taken short" at all hours and however engaged. His condition has lasted for four years. From birth the child has suffered from bleeding at the hand and at two years of age was admitted into a hospital for operation.
The bleeding was checked by acupressure and ligature, but on returning home a fortnight later it was found he could not walk without difficulty and had incontinence of bladder and bowel. The chills had experienced emetic and local pain, and had developed the incontinence and fever within the day after the operation. Details of the temperature, and of the urine state during his stay in hospital could not be obtained.

Under the long continued administration of strychnine in full doses he made a good recovery.

Remarks. The diagnosis which at first suggests itself in both cases is, probably, that of a focal meningitis at the upper end of the lumbar enlargement, involving, in the first case, a considerable area of the cord. In each case it is probable that the cord-change was consequent upon blurring irritation—the apparent nerve being in the first case the lateral tracts of the dis-innervated, and in the second either a cutaneous branch of one of the abdominal intercostal nerves or, possibly, the irritation may have been transmitted through the
Urachus to the bladder, and by the vesical nervous nerves to the hypogastric plexus, and cord.

Paraplegic symptoms following on nervous irritation have for a century and a half engaged the attention of physiologists, and under the various titles of 'Reflex and Urinary Paraplegia' and 'myelitic lesion on Rehn's' they have given rise to a copious and bewildering literature.

Excluding these cases, for the most part antecedent or serial in their etiology, in which glossosteci, or other microbes may be purely suspected of playing a leading role in the maddening process, three remain on record a large mass of cases, to sustain which numerous theories have been invented.

From Siemantel's suggestion of temporary paralysis of the functions of the cord, produced by deprivation of blood supply, through von Winiwarter's spasm, has been so severely handled in this country by Hall and others, as to be, perhaps, generally discredited.

There remain to be considered:

1. The theory attributed originally to Froga, but restated by Lehoux, of an ascending meningitis, leading, by propagation along the nerve fibers, to inflammation of the spinal cord. This, however, according
to Charcot himself ("German clinical lecture," 5p. 
Soc. trans. Vol. 28, p. 163.), and to Charcot, 
an undemonstrated lesion.

2. The suggestion, favoured apparently by Charcot, 
(cited), of a teraphic myelitis set up in the cord 
by peripheral irritation, but without direct 
transmission by neuritis. This theory is supported 
by Ricci's experiments (Lay ecs. ib. p. 103.)

3. The theory of paralysis of function by inhibition, 
supported by the experiments of Lewyson and 
Weiglein (Lay ecs. ib. 157).

It must be allowed that experimental or 
pathological facts can be adduced in favour of both 
views. And if myelitis, in the place of being an 
inflammation of the nerve tissue of the cord, can 
run its course without being associated with the 
variable concomitants of inflammation elsewhere, 
i.e. without rise of temperature, without pain, 
and without the circulatory disturbance which, in 
other parts of the nervous system, rise to motor 
and sensory irritation symptoms; if this can be, 
then there is no need to go further afield in seeking 
to explain cases such as the one first described. 
It is difficult however to frame a conception
of nerve-tissue inflammation which shall not include these fundamental symptoms, and their absence in cases of so-called myelitic onset to raise a natural doubt as to whether the process has been an inflammatory one at all. Such a doubt must pertain to the case first described, where the cord ran through extreme in enough to altogether the function of a considerable area of the cord, yet ran its whole course without pain, without pyrexia, and without an irritation symptom sensory or motor. It seems reasonable therefore in such cases to assume some non-inflammatory process at work of a kind similar to that, which in other parts of the central nervous system produces paralyzing effects. Such a process is the softening of nerve tissues which follows deprivation, for even a very short interval, of their blood supply.

The analogy of the brain would suggest hemorrhage as the most likely cause of the Ischemia.

The prevalent anatomical conditions of the circulation in the lower segments of the cord and the imminent tendency to starvation and stagnation of the blood current which this condition bring about have been clearly pointed out by Dr. Moson ("Coronian Lectures," 1871).
Given such a blood current, slowed to the point of imminent, if not actual, stasis by varicose arterial spasm acting locally, and a starting point for such thrombosis might easily be furnished. The fact that the region of the end where softening most frequently occurs, e.g. in the anterior horns, are those where blood supply is by a series of terminal twigs, favours the suggestion that such a pathological process actually does take place. And if so, the explanation it furnishes, in accounting for such cases as the one first described, appears to be a more rational one than does the already overburdened phrase 'myelin.'
A Case of Paralysis into the Pons with Recovery.

J. M. a farmer, aged 47, whose antecedents, however, were older than his years, was first seen on Sept. 23rd, 1841. He had then been drinking raw whisky for a week, keeping it for convenience in a pint mug by his bedside. He was lying helpless and stupid in bed. Pulse 120. Respiration 79. On the evening of the following day, being ailed, and having had, as far as is known, no access to stimulants during that day, he suddenly at 8 p.m. complained of being sickly, and vomited. His attendant noticed nothing peculiar about him till midnight, when the patient's breathing attracted his attention. This had become irregular, heavy snoring respiration alternating with intervals of shallow breathing.

On the 25th, at 9 a.m. he was in the following condition: semi-comatose, but could be roused by speaking loudly to him. Unable to speak except in incoherent splutters, a word being responsible here and there. The face was deeply flushed. Respiration 16. Chryse Stools in character. Pulse 64, feeble, irregular. Temp. in left axilla, which was sweating.
freely) 100°. In the right, (not sweating), 99° 40'.

The pupils contracted to size of No. 6. catheter,
gauge - active to light, but slowly.

There was complete motor paralysis of the left
arm and leg, and of the trunk (left side) and of
the lower half of the left side of the face. There was
complete loss of sensation to touch and pain over the
same area of the body and limbs, and over the
right side of the face as well. The plantar
reflex was abolished on the left side but present
on the right. The tongue was protruded, with
difficulty, to the left.

On the following day, the comatose state being
left unaltered it was noted that the facial paralysis
was not complete on the left side of the face,
he being able to move the left angle of the mouth.
He was not able to contract the orbicularis oculi, so
as to whistle. The nasal labial fold was obliterated.

The orbicularis oculi, and occipito-frontalis
were unaffected. There was slight ptosis of both
brows, most marked in the right, which eye was
congested and irritable. There was only paralysis
of the right external and left internal rectus.

No conjugate deviation of the head.

Sensation to touch and pain was diminished.
over the right half of the face, including the conjunctiva, but this anaesthesia was left marked in the lips and was absent in the tongue. The muscles and teeth and jaws were unaffected. Hearing, taste, smell were unaffected. He had difficulty in swallowing solids, and fluid frequently passed into his windpipe on several occasions also, when no cough had occurred fluid returned through the nose. He was still unintelligible in his speech. The optic discs were healthy. Vision was good in the left eye, but impaired slightly in the right, and there was no extinction of the visual fields.

Urine free from albumen and sugar. No carci
crin present.

On the 29th, the breathing was regular. a linear corneal ulcer, with mucous purulent ophthalmia had developed in the right eye, and on looking to the left there was lateral nystagmus.
The globe paralysis to the right still continued. The left pupil was somewhat larger than the right.

On Oct. 8th the oculo motor paralysis was still present. The nystagmus disappeared from the left eye, but still present in the right.
The anesthesia was disappearing from the left side of the body, and there was some improvement in the motor power. He could swallow well.

The pupils were now normal, and the ulcer of the cornea healing.

On March 16, 1892, he was able to walk, but with a slow step and stagger, due to incoordination of the left leg. There was a constant feeling in the toes of the left side, as if they were in scalding water, but objective sensation over the limb. This was still present, even fairly good. On attempting to walk, spasm and stiffness occurred in the leg (left), and the toes were dropped, and occasionally caught in the ground. Both knee jerk and plantar reflex were now increased on the left side. There was no clonus.

Movement in the left arm was much improved but the hand actions were clumsy and inco-ordinate. He could not pick up a piece of bread or button his coat. The pulp of the fingers were numb, but sensation was otherwise good in the limb. A tremor occurred in it on voluntary movement.

Speech was still indistinct. Swallowing and ocular movements were normal. The discs were healthy.
The patient remained in much the same condition until his sudden death two years later. A P.M. coma was not be obtained, but the circumstances pointed to internal cranial hemorrhage.

Remarks

The grouping of symptoms, cerebral and general, the partial paralysis of the right VII\textsuperscript{th}, ophthalmoplegia of the right VI\textsuperscript{th} and left palpebral retraction, interfered with the left VII\textsuperscript{th} and XII\textsuperscript{th} with sensory and motor paralysis of the left half of the body - pointed clearly to a lesion, probably hemorrhagic in the right upper half of the tons, affecting at once: (1) the supra-nuclear tract of the left facial and the palpebral, (2) the tract passing between the left oculo-motor and right abducens nuclei, and concerned with conjugate deviation to the right, (3) some portion of the sensory nucleus of the fifth, and (4) interfering with the conduction through the tegmental and pyramidal fibers of the tons on the same side.

The palatal and pharyngeal muscles noticed on the morning following the stroke, but not subsequently of interest. The recovery of the
patients, and the escape of the lower motor and medullary nuclei on the same side, practically preclude the possibility of the hemorrhage having extended into the medulla. It seems more likely that the connecting fibers between the bulbar motor nuclei and the basal ganglia— the "second link" of Meynert's "projection system," pass through the pons in proximity to each other, and that some supra-nuclear fibers connected with the reticular nuclei, shared with the hypoglossal tract in the compression effects of the hemorrhage.

The cases: ataxia of arm and leg, with hearing disturbance, on the one hand; and the spasm, increased reflexes, and tremor on the other, which were noted at the last examination, and continued till the patient's death, pointed no doubt, to ascending and descending degeneration secondary to the hemorrhage.
A Case of Cerebral Tumour, with Hemianesthesia.

James M., aged 45, a labourer, was admitted into Whitechapel Hospital on Nov. 20th, 1893, complaining of severe headache, inability to walk, and numbness, with some weakness of the left side. His family history was unimportant. His personal record includes a chancery in his twentieth year, and malarial fever when on foreign service. Otherwise his health has been good, and he has not drunk too much.

The present illness began, nine weeks before, with severe pain in the head. He had, however, noticed aching, and occasional headaches for some months before. Two weeks after the severe headache began, he had cramps in his leg, chiefly in the calves, but they did not persist, merely becoming stiff and painful. During an attack of this sort in the week before admission he fell downstairs, striking his right ear, and the head behind the ear, in the fall. A month later he had a shivering attack, with aching in the back, neck, and head (influenza?) for which he went to
bed, and remained there till his admission to hospital. There were no convulsions, or motor spasms at any time during his illness, nor did he ever lose consciousness. While in bed he noticed a gradual onset of numbness in the left side of his body, which also became somewhat weaker than the right. Coincident with this his wife had noticed increasing torpor and stupidity. He had had some attacks of vomiting, independently of taking food. He complained also of feeling, and of failure of sight.

Examination, which showed the patient to be a well built muscular man, who lay on his back with closed eyes, seemed stupid and heavy, and appeared to have difficulty in understanding and answering the questions put to him. When pressed for an answer, he would, after a pause, say impatiently, "I did not know", or make a quite irrelevant reply. The information he volunteered, however, apart from questioning, was intelligible, coherent, and to the point. He never mixed up incoherent words in his sentences, nor made meaningless sounds. He could read accurately the letters in Snellen's type, and write small words correctly.
His condition therefore suggested some aphasia of the variety described as word deafness.

He complained greatly of pain in the frontal region, and cranial perfusion showed tenderness over the right temporal-parietal area. His motor power was good on the right side, but slightly impaired on the left. He had still however, considerable power in the left hand-grasp and in flexing and extending the left leg. Muscular nutrition was everywhere good. There was no rigidity on passive movement. Electrical irritability was diminished in both currents on the left side of the body, but there was no qualitative change.

The knee jerks were normal on both sides, and there was no clonus. The plantar and cremasteric reflexes were impaired on the left side.

Bladder and bowel control were normal. The right pupil was dilated to about No. 12 Catherider gauge, and did not react to light. The left was of medium size and reacted, but sluggishly. Coordination was unaffected on the right side, but appeared defective on the left. Muscular tone was much impaired on the left side. He was quite unaware in what
position his left arm and leg were placed, imagining them to be straight when they were flexed. This was not the case with the right side.

There was great impairment of this arm to touch, pain, and temperature over the left side of the body. He did not feel a pin prick which on the right side made him wrinkle and withdraw the limb. He did not recognize as warm a brolke of water, a little below boiling point. He was quite unconscious of light touch over the impaired area. This area includes the left half of the face and trunk, and the left arm and leg. The pain, thenia appeared to be deepest in the leg. Normal sensation is recorded at about an inch to the right of the medias line.

The condition of the cranial nerves is as follows:

I. He cannot distinguish in either nostril individually, the smell of hyacinth, clove, peppermint or tobacco. He complains that there is no flavour in his meat, or his tea, and that every thing tastes alike.

II. Vision. R. 60. T.o. at 22 em.

R. P.D. only.
There is optic neuritis on the right side. On
the left the disc cannot be examined on account
of clouding of the cornea. There is conjugate
contraction of the visual field on the right side.
The right pupil is dilated and motionless.
The left is of medium size, and its reaction to
light cannot be ascertained. Reaction to
accommodation cannot be accurately determined
in either eye.

\[ \text{III} \] Tonic, and paralysis of the internal
Rectus on the right side.

\[ \text{IV} \] Nearly complete facial anaesthesia on
left side, which includes the momentary surface
and extends slightly to the right of the meral
line. Commissural nerve paralytic ophthalmoplegia
in left eye. In May the Contraction Equal on
both sides.

\[ \text{VII} \] Unaffected.

\[ \text{VIII} \] He hears a watch tick, which is normally
audible at 30 inches, at a distance, on the
right side, of 2 inches, and on the left at 12.
The relatives cannot say whether he was deaf
before his illness, but think he was not. The
tympanum are healthy in both ears.
IX. He cannot distinguish on either side of the tongue, solutions of salt, tartaric acid, & sugar, or quinine. The tongue is thinly furrowed. The solutions are applied to the back as well as the front of the tongue.

IX
IXI
IXII

There is some swelling of the left side of the face, and a slight flush on the left cheek.
Urine, acid, sp. gr. 1018. Free from albumen and sugar. h/o = 200 cc. per diem.

On the 23rd it was noted that the h/o was lip marked, and on the 25th the right eye was opened equally both the left, and the right internal rectus has regained its power. The right pupil was still dilated, but now tracked slightly to right.

During his stay in hospital the observations made were frequently verified. He was given several times to move all his limbs, to raise himself and to sit on the side of the bed. He was unable to stand, or walk on account of vertigo.

Subsequently the ophthalmia of the depreys went
on to perforation of the cornea, and his tears began to drop. He had still however occasional
intervals of clear speech, when he would ask questions quite intelligently, but could never be
got to return a relevant answer. On Dec. 12th he became comatose, his temperature rose to
106°, and he died shortly afterwards.

Autopsy, fourteen hours after death, at which I was assisted by Dr. Asle. On opening the skull
there was thickening and opacity of the Dura
over the right Temporo-Sphenoidal Lobe.
The arachnoid space contained several ounces
of blood-stained serum, which was seen to flow
from a deep sulcus surrounding a tumour in
the right lower Temporo-Sphenoidal region,
and the peritoneum of the floor suggested communication
with the right lateral ventricle.

The tumour (Fig. 5) was bony in shape,
measuring 4½ cm. in each diameter, by
2 cm. in thickness. It occupied a bed of
softened brain tissue which had absorbed
half the breadth of the middle, and had
completely obliterated the lower. J. S. Convolution
The Sag. J. S. Convolution was greatly enlarged
in breadth and length, and was, like the remainder of the brain, displaced upward and forwards. During its displacement the frontal and parietal lobes were compressed somewhat, and the figure of Sylvius was anterior to its usual position. Moreover, the J. S. lobe occupied more than its normal proportion of the surface-area of the hemisphere.

The left hemisphere was normal in shape and proportion. The nerves at the base of the brain showed no change beyond compression and flattening of the right cerebellum motor, so marked no change could be detected in the fifth of the left side, nor in the G. Herrellian ganglion, nor in the auditory of the right side. Unfortunately these structures were not preserved for microscopical examination, as was intended.

On the median aspect of the right hemisphere there is evident compression, and softening of the hippocampal gyrus, in the neighbourhood of the striae, and between this and the thalamus. The uncinate and occipito-temporal gyrus (beneath the collateral fissure) are also superficially softened. The lingual gyrus
appears outwardly unaffected. The rest of
the median surface of the hemisphere appears
normal. A vertical (frontal) section was
made through the hemisphere in the plane of
the center of the tumour, (Fig. 5. line A.B.).
This plane passed through the sphenoid of the
Corpus Callosum in its greatest diameter, and
met, at the superior margin of the hemisphere,
the posterior superior extremity of the Sinus of
Rolando.

It was seen by this section (Fig. 6.) that the
decipito-temporal gyrus was compressed to a
mere sherd from two hallowed men, in its
greatest thickness. The medullary matter of
the lingual Gyrus, and that at the bottom of
the Calcarine Fissure (which here include the
medullary matter of the hippocampal lobule)
was softened to pulp. The bed of the tumour
occupies most of the thickness of the Temporo-
Sphenoidal lobe in its posterior half, and latter
two thirds - and from the softened medullary
matter composing it, a strand of softened
tissue passes upwards, and forwards toward,
the floor of the Lateral Ventricile. This softened
track extends up both walls of the Ventricile,
- on the outer side as far as the plexus matter forming the bottom of the parallel figure, and on the inner side—formed here by the Cornu Ammonis, below, and, higher up, by the alveus of the hippocampus (posterior pillar of fornix)—the softening extends as far as the Sphenum of the Corpus Callosum, which is not softened. The cavity of the ventricle is obliterated, and the softened walls are in contact with each other.

A horizontal transverse section through the anterior portion of the hemisphere (Fig. 5, plane of line E.F) displayed the internal capsule in relation to the softened medullary matter of the 3rd lobe. It was found (Fig. 8) that the posterior fibres of the capsule did not share in the softening, but were separated from the disorganised tissue by a space, a few mm. in breadth, of apparently healthy medullary tissue. Beyond this distance, at about 5 mm. from the posterior extremity of the ventricular nucleus, the medullary fibres were softened, and were in anatomical continuity with the softened bed of the tumour. A tract of softened arcuate fibres could also be traced passing into the white matter of the adjacent convolution.
A section through the posterior portion of the hemisphere, in the same plane, shows that the area of softening in the medullary matter extended backward only for a space commensurate with the superficial area of the tumour. The medullary matter of the hemispheres was unaffected.

The cerebral peduncles and pons showed, on section, no visible change.

The brain was hardened in Müller's fluid for two months, and for a month in alcohol. Sections were then cut from:

(a) the tumour.
(b) the posterior parietal of the left capsule.
(c) the tract of medullary substance posterior to this, including the boundary line between healthy and softened tissue.
(d) the softened right hippocampus major.
(e) the inferior J. S. convolution in its middle portion.
(f) the crus cerebri (right) and pons.

(a) The tumour was found to be included in its outer surface in a vascular capsule which appeared to be the thickened pia.
Internal to this was a zone containing numerous arterioles, in connective tissue which was densely infiltrated by small, round, crenate, looking bodies, which took on a deep, blue, wood stain, and resembled the granulation tissue of epithelial growth. The arterioles in this zone and in the capsule showed engrafted connective tissue obliteration, most of them being either obliterated or in process of being obliterated by the invasion of the outer and inner tunic by the small twilled growth mentioned. Many of these so obliterated showed abundant fibres of white fibrous tissue replacing the normal tissue of the walls, and some of them on section had the appearance of being round dense cords of fibrous tissue. More internal to this granulotic zone were areas of connective tissue and strands of fibrous tissue, the latter enclosing tracts of cataract, and elsewhere of haemorrhagic debris.

(b) The posterior fibres of the internal capsule showed no marked change. The nerve fibres were of equal size, their axis-cyinders torn in a deep stain with aniline, blue black. The myelin lamellae, while sheathed, exhibited no enlargement, nor degeneration.
(c) The tract of softened tissue posterior to the internal capsule could not be included in sections, even when cut in celloidin. The broken edges from which tissue had escaped exhibited at the boundary line swollen and irregular fibers, with numerous dark stained connective tissue nuclei. Tissue treated in formic acid showed numerous myelin globules in the irregular fibers, compound granular bodies, and fatty debris.

(d) The hippocampal region proved difficult to recognize, owing to the dis appearance in mounting of the long central fibers of the albas, and the apparent absence of large pyramidal cells from the subiculum layer. There appeared to be a great proliferation of connective tissue nuclei, but the normal structure of the part could not be recognized.

(e) The superior temporal-splenial convolution was nowhere softened except at its anterior portion already referred to. Elsewhere there was an increase both in size and in relative map of the hemispherical fibers and cells. The nuclei were frequent double, and were more numerous than in normal tissue.
(f) The crura cerebri and pons exhibited a normal appearance. The longitudinal fibres had uniform sheaths in cross section, and remained axo cylinders. No degeneration could be anywhere detected.

The uterine were stained by a silver carbonate, and by an Alkali method, and by van Gieson. I regret that my want of skill as a microscopist prevents me from representing their appearance.

Remarks. The scarcity of clinical pathological records, throwing light upon the localization of the cerebral sensory centres, has been frequently remarked. The remarks cannot yet be said to be in a form of being confirmed. A search of the only available literature at my disposal having yielded but scanty notes of such cases. Ferrier reports a case in which a tumour pressing on the left temporal and parietal lobe caused right hemi-anesthesia, in which mantaining two cases of left hemi-anopsia, in one of which right hemi-anopsia was present. In both there was atrophy of the

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1 [Footnote]:1/87.
2 Medical Week. Vol.I. No 54.
Convolusions boring the calcaneus figured, atrophy of the "central portion of the dorsal oculomotor figure", and atrophy of the part of the Camer, extending to the lower extremity of the hippocampus. Unfortunately, however, no detailed criticism is given of the association of symptoms with lesions in each individual case.

Data? in a collection of 192 cases, quotes 30, as being cases in which no anaesthesia was associated with lesions of the body of the fornix or gyrus hippocampi. In his own showing however, some disturbance of sensation was present in nearly one third of the number.

San in reports left anaesthesia and trophic changes occurring in a case in which a cavity was present beneath the cortex of the right gyrus hippocampi.

Hammond reports a case, in which (among other lesions) a pleuritic lesion of the left gyrus hippocampi was associated with anaesthesia of the left side.

If clinical observation however lags in rear of experimental research in this matter, it is satisfactory to note that the direction of the former

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Footnotes:
1. Journal of Mental Science, 1888.
3. ib. 1891.
is in the forerunners of the latter. Thus Horsley, and Schefler, expanding Ferrier's previous conclusions, are of opinion that the gyrus forniciatus represents in the hemisphere the area of cutaneous sensibility, and on the clinical side, Willis, after a digest of recorded cases, concludes that no part of the brain is more likely to contain the areas (for cutaneous sensibility) than the gyrus forniciatus, the hippocampal gyrus, the precuneus, and the posterior parietal convolution.

The difficulties which beset a solution of the problem on the clinical side, appear to arise partly from cases, apparently anomalous, in which the specific symptoms are associated with lesions of areas other than those indicated by experiment, and, in the reverse place, from instances, apparently negative in import, in which most of the conditions of the region indicated by experiment are accompanied by no specific symptom. Concerning the latter, the phenomena of functional compensation are probably equally valid in explaining the apparent anomalies of sensory "negative instances" as they have proved to be in the case of the motor functions. And as regards the first mentioned difficulty, the

\(^1\) New. Med. Soc. 1885.  
\(^2\) Brain, 1889.
accumulation and careful criticism of faithful observations will certainly, with a widening knowledge, reveal the explanation of these apparent discrepancies.

In the case I have recorded the abscence of morbid changes from the sensory tracts of the Pros. Crus and internal capsule points to the cortical areas involved, and the pan, underlying them as the seat of the sensory disturbance.

The location of the softness thus present in the internal capsule is such that it would involve the temporal portion of the Insom. occipital thalamo-thalamic fibers, which, lying to the outisde of the pyramidal fibers in the crus, are known to radiate outward and backward, to the temporal and occipital cortex. As these fibers are presumed to be motor, or at least not sensory in function, however, the sensory impairment cannot be associated with their damage. It is reasonable therefore to connect the anesthesia, general and special, with the cortical regions on the outer and medial surfaces of the temporal lobe and with the destruction of the subjacent medullary fibers.

Such a conclusion would be in complete accord with the observations of Ferris and Gro. who showed that lesions of the hippocampal region, hippocampus, and the neighboring head of the calcarine figure were associated, in aphes, with loss of vision on the opposite side of the body, and in as much as the hippocampal and uncinate gyrus are subordinate divisions of the gyrus, fornix, and cingulate, the conclusion is equally in harmony with the latter observations of Horsley and Schäffer.

With reference to the special nerves, it is probable that the loss of hearing on the right side was due to intracranial pressure on the right fornicus motoris. The right ins. convolution in its posterior part was not affected otherwise than by upward displacement, and hypertrophy of connective tissue.

The impairment of taste and smell on the left side were probably contributed to, if not accounted for, by the facial anesthesia on that side. On the right side however the loss may be associated with the damage to the gyrus, and medullary matter of the hippocampal gyrus, consequent on the pressure of the tumour.

[Footnotes: Functions of Brain 1886. Chap. IX. Supra, etc.]
In considering the almost total loss of sight in the left eye, the difficulty is to assign to each of several morbid conditions present, its legitimate share in the result. There was extreme cloudiness, with, later, perforation of the cornea. Possibly too, optic neuritis was present. This, however, had not, in the opposite eye, gone so far as to produce amblyopia. I therefore allude to possible destruction of some fibers of the optic Radiation, in the white matter of the temporal lobe. I am not aware whether this lesion has ever been definitely associated with crossed amblyopia. But the association of lesion of one angular gyrus with crossed amblyopia has been established by Ferris, and clinical cases of similar import have been recorded by him, by Graft, and by Sherrington. A case of hemi-anopsia, hence with homonymous hemi-opia, in which the softening involved the fibers of the optic Radiation is reported by Kelner, but in his case the area of softening was much more extensive than in mine, extending back to the surface of the occipital lobe. If, however, I have not been able to refer to the original.
the fibres of the optic radiation proceed, in part to the Angular Gyms, it seems possible that a lesion of small area might affect those fibres only which are in connection with the Angular Gyms of the same side, and so produce crossed hemiplegia, rather than hemianopia.

Although in the present case the patient's condition remained futile any careful parametric investigation, yet I am of opinion from the observations made, that his visual condition,

(of any further explanation of it were required than the corneal lesion supplies) indicated hemianopia, and not hemianopia.

The condition of the right eye was sufficiently accounted for by the ascertained optic neuritis.

The patient's mental condition, which, on admission was recognised as suggesting a form of purely aphasia, is extremely interesting in connection with Starr's investigations into the pathology of that condition.  

In an analysis of fifty cases of purely aphasia, Starr found that the convolutions most frequently affected were the 1st and 2nd Temporo-Sphenoidal.

In seven cases pure word deafness was present

\[1\] Kraus, 1889.
and in all of these, the lesion was limited to the 1st and 2nd D.S. convolutions, in their posterior two thirds.

As regards Diagnosis, I may say that the only approach to one made was the suspicion of a summatory tumour or tumours, somewhere at the base of the brain. The difficulty, of course, was to harmonise signs which pointed at the same time to Compression of the right oculo-motor and auditory, with injury to the left trigeminal, and to explain at the same time interference with the trunca, track passing to the left side of the body, without serious implication of the motor fibres.

Our weight was probably not given to the subacting indications which might have led to a localising diagnosis of injury in the region of the uncinate gyms and hippocamatal lobe but none were sufficiently present to my mind the localising indications afforded by the trunca aphasia - according to Stare.

Here let us say no single lesion could be imagined associating all the signs present, and the impression of multiple lesions did not afford much assistance. It is to be hoped
That sufficient clinical material will eventually be accumulated to allow of greater confidence in the localization of cerebral sensory symptoms, and the present case is recorded with a view to that end.

Drugs.
May 1894.
Appendix.
Fig. 1. Ellen W., age 21. (From a photograph)

Showing shortening and broadening of the feet, with tendency to inversion of the left foot. This is more marked when the limb is unsupported. It then appears in both feet.

Both hands show extension at the metacarpophalangeal and flexion at the metacarpophalangeal joints. See also fig. 2.
Fig 2 - Case of Ellen W. Side view of feet, showing shortening of metatarsal-phalangeal extrusion and inter-phalangeal flexion in left foot and mutation mutandis in right hand.
Fig. 3.  Henry W. aged 26. [From a photograph].

The head had to be raised by an assistant and studied in order to obtain a photograph. The left hand shows the condition of satiation at the metacarpal-phalangeal and flexion at the interphalangeal joints. This position when unassisted is one in which the flexion of the trunk and head is much more marked.
Fig. 4. Case of Mabel B. (From a Photograph).

Showing the position of the feet in dorsal decubitus. There was occasionally much greater flexion of the toes than appears in the print.
Fig. 5. Right hemisphere, case of T.N.

with tumour in situ. (from a Photograph)

To show the tumour more clearly, the point of view is from a little below the transverse axis of the brain. Consequently the upper margin is not visible.

1. P. Interparietal Sulcus
2. R. and S. Fissures of Rolando, and of Sylvius

Reticular refers to planes of section mentioned in the text.
**Fig. 6.** Section through brain in plane of line AB from photograph.

D. vermous.

The following letters apply to both figures:

A. Angular convolution.

B. Parallel fissure.

C. Middle T. S. convolution.

D. gyrus lingualis.

E. hippocampal gyrus.

F. Splenium of Corpus Callosum. The gray and soffted appearance is due to the inertia of exam work into the lateral ventricle to allow hardening, fluid to subside.

G. Hippocampal Fissure.

H. Hippocampal gyrus.

I. hippocampus major.

J. Hippocampus minor.

K. Calcarine Fissure.

L. occipito-temporal gyrus.

M. Juxtaposed gyri.

N. Optic thalamus, ant. polar lenticyle.

O. Callosal Convolution, of Corpus fornication.
Fig. 8. Section through anterior portion of brain-case (case of Mr.)
from a tracing, in plane of line.

A. optic Nudus. B. Caudate Nucleus. C. Commisural fibers of Optic Corpus Callosum. D. Area of Softening, in
region of optic Radiation, posterior to internal capsule, but
separated from it by an intervall of healthy tissue (two densely
stained areas in surr.ing). E. Softened bed of tumour, a strand of
softened fibers passing into F. the sup. T. convolution. G. Hippoc.
pyriform. H. Hippocampal nucleus. The convol. external outline is not represented sufficiently firm.