Paralysis Of Peripheral Origin In Pregnancy And The Puerperium.

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Contents.

General Considerations.

The diseases associated with the pregnant state are many and various and may consist either of such as are due to the Condition itself, or of others which may be regarded as accidental complications. These latter may have existed before the inception of the pregnancy, or they may have been acquired during its course. As a rule all diseases which throw a considerable strain upon the organism are more serious when occurring in the pregnant state.

With regard to the diseases of the puerperium it may be said that they consist for the most part of affections of the genital organs, or the genital tract, and the breasts, and are of a septic nature. Apart from these, diseases are rare, and especially rare are the paralyses of peripheral origin with which this paper treats. This is known by the name of puerperal

Neuritis, although it occurs during pregnancy as well as during the puerperium. Sometimes it extends from the one into the other, and probably in the majority of cases the origin of the neuritis may be traced during pregnancy. The rarity of the condition is such that until recent years it was barely mentioned in English text-books.(1) Turney in his valuable paper upon Polyneuritis in relation to Gestation and the puerperium mentions that in the maternity Charity at St. Thomas's Hospital, with an annual rate of over 2,000 confinements, no case had been observed during several years. That the condition does exist is beyond question, and, in view of my own Experience as a General Practitioner, the rarity of the disease is probably more apparent than real. It is probably within the experience of every accoucheur that in certain cases of labour of more or less severity there is some pain and cramp

complained of during the second stage. For the most part these symptoms pass off within a short period; but there are occasions when the pain and numbness increases instead of abating and may even go on to paralysis. It would be difficult, if not impossible, at the present time with our present knowledge to state the percentage of cases of puerperal neuritis to all labours. In my own instance, I have seen two cases in my [redacted] practice within the past twelve years, and I was consulted for another occurring in a friend's practice. The number of labours attended within that time would probably be about 1400, which gives a percentage of .14 in my own cases. The importance of these cases is not to be overlooked, if only to avoid the risk, mentioned by Tarnier, of attributing the condition, in spite of the patients' denials, to the excessive use of Alcohol.

Within the past fifteen years many cases

of puerperal neuritis have been placed upon record, and the pathology has been thoroughly discussed. There is, however, much which remains to be discovered before the causative agents in the most form of the disease can be demonstrated. The connection between the toxic form of puerperal neuritis and the toxaemia of pregnancy is probably very close, and it is believed by many authorities that what causes the one is mainly responsible for the occurrence of the other. Even in cases which are directly due to pressure upon the lumbar or sacral plexus within the pelvis by the foetal head, or which follow upon an injury by forceps, it is thought by some observers that there is frequently, if not in all cases, a poison acting upon the nerve fibres. Some writers have made an endeavour to discriminate between local neuritis and a true polyneuritis of puerperal origin. But there is no necessity to make any such distinction;

the pathology of the several different Groups into which the disease is now divided may be identical. Owing to the Extreme rarity of the condition it is not easy to study the disease adaequately. In my opinion it occurs not infrequently in private practice, and few cases are reported. The difficulty of excluding alcoholic and lead poisoning in some of the cases, especially in those which occur in the poorer localities in large towns, must be very great, and fortunately the practice of midwifery in Country districts is not often encumbered with such serious complications. In the following pages no new discovery will be found disclosed; it has rather been my aim to present the leading features of the disease with brief references to the literature of the subject. No comprehensive review of puerperal neuritis has been attempted, and the allusions

to the views of standard authorities have been taken from articles and theses which have appeared chiefly in Continental and American publications.

Facts known about the Neuritis of Pregnancy and the Puerperium.

The paralyses which occur during pregnancy and the puerperium may have a central or a peripheral origin. When the lesion is central it may be cerebral or spinal.(2) The following classification has been proposed for paralysis of central origin: —

1. Paralysis — without anatomical lesion.
   (a) Hysterical.
   (b) Myasthenic.
2. Cerebral paralysis.
   (a) Cerebral Haemorrhage.
   (b) Albuminuric paralysis.
   (c) Cerebral thrombosis.
   (d) Cerebral Embolism.
   (e) Other Cerebral lesions.
3. Spinal paralysis.

(3) The following classification has been suggested with regard to paralysis of peripheral origin. It is the one which has been adopted in this paper.

Peripheral paralysis.

1. Myopathic.
   (a) Paralysis accompanying osteo-malacia.
   (b) Polymyositis.
2. Neuritic.
   (a) Traumatic.
   (b) Neuritis per contiguitatem.
   (c) Neuritis post infectiosa.
   (d) Toxic neuritis of pregnancy and the puerperium.

The peripheral paralysis sometimes seen during gestation have been ~~but lately~~, comparatively speaking, but lately studied and classified by observers. They arise under different circumstances, and are seen at different epochs. Thus paralysis may begin during pregnancy and extend into the puerperium. Or it may occur in the puerperium for the first time sooner or later after the

completion of labour. When the disease occurs during pregnancy it is always the outcome of a toxaemia and it may be a multiple neuritis or limited to a single nerve; when it shows itself only after parturition the symmetry of the condition may be more or less obvious, but it is generally believed that the poison circulating in the blood is identical with that which gives rise to neuritis during pregnancy.

In the neuritis of pregnancy there is frequently excessive vomiting and other signs indicative of toxins circulating in the blood. The phenomena accompanying puerperal neuritis led (4) Möbius in 1890 to attribute the disease to a poison in the circulation. (5) In 1887 he had described seven cases, which along with a case described by (6) Kast are the first recorded in literature. Discussing the pathology of puerperal neuritis (1) Turney endeavoured in 1897 to differentiate between a true toxic neuritis following upon childbirth and a

Traumatic Neuritis following upon pressure upon the sacral plexus by the foetal head or the forceps. He suggested that the term Obstetric Neuritis would be more appropriate for the traumatic inflammation of nerves seen after parturition. He quoted a case recorded by (7) Lamy in which paralysis followed upon an injury by forceps, and considered it ought not to have been included in the list of puerperal Neuritis Cases. He admits there was possibly a secondary Neuritis following upon the injury, but he considered the toxaemia as of quite subordinate importance in comparison with the local trauma. Cases of puerperal neuritis have been reported which were probably alcoholic, and so outside any of the groups into which the disease has been divided.

Symptomatology.

The first symptom usually complained of is pain. This is frequently severe and is

described as being of a lancinating character and extends along the limbs first affected. Usually the lower extremities are the first to be attacked. There is frequently a feeling of numbness and pricking (pins-and-needles) in the part affected. The pain and numbness may be followed in a few hours or days by loss of muscular power. This may go on to complete paralysis. There is rapid wasting of muscles and loss of reflexes with more or less complete reaction of degeneration within a week or two. There is tenderness of muscles and nerves which may be extremely acute. The disease most frequently attacks the anterior tibial and peroneal groups of muscles in the lower limbs, next in order the quadriceps extensor cruris and least often is seen in the gluteal muscles. In the upper extremities it is most pronounced in the posterior interosseous group. The sphincters are not often affected, and when this symptom

is present it soon passes off.
Sensation may be defective to a greater or lesser extent. Usually there is some interference with sensibility in the fingers and toes; but in severe cases anaesthesia may be complete. The vagus may be affected as shown by tachycardia and dysphagia, and there may be aphonia pointing to a lesion of the spinal accessory. The cranial nerves may be affected — optic neuritis and deafness may be symptoms. Mental symptoms are often shown and may range from some form of emotionalism to a true psychosis. Usually there is confusion of mind and loss of memory. The incubation stage of the disease occupies from one to three or four weeks. In a few improvement sets in soon after the symptoms have reached a maximum, recovery being complete in a few months. But the return to health may be delayed for months or years. The clinical picture of the

neuritis of alcohol and that seen during gestation are wonderfully alike. There is no one symptom seen in the one case which cannot be found in the other.

The neuritis may be confined to the upper or the lower Extremities. In the Generalised form there seems to be a special susceptibility in the nerves of the lower limbs. In the localised form of neuritis the upper limbs seem to be most frequently involved. It has been also noticed that the posterior nerve in the arm is often most severely affected in the Generalised form, whereas in the localised form the ulnar and median nerves bear the brunt of the attack. The difference between localised and generalised neuritis seems to be one of degree, otherwise, rather than character. Sometimes an upper limb or a lower limb type has been described, but they all run into one another, and it is not unlikely that nerves may be affected

without any clinical evidence of the fact, and the Extent of the neuritis may be found out only by a post-mortem Examination.

Description of three cases of Puerperal Neuritis.

Case I.

Mrs B., aged 36 years, was a multipara in her fifth confinement.

The family history was good. Both father and mother were healthy people, and there was no history of nervous disease in their families.

Personal History.

As a child the patient had been healthy and free from disease. During early girlhood she was treated for anaemia. Her first child was delivered with forceps, and she made an excellent recovery. Towards the end of her second pregnancy she had suffered from oedema of the lower extremities and vulva. Unfortunately no medical man was called in, and she had severe eclampsia at the eighth month. Delivery was expedited and the child, still-born, was extracted with some difficulty. Her other confinements were quite normal.

History of the illness.

The fifth pregnancy ran a normal course, and labour came on at term. After a lingering second-stage, extending over ten hours, forceps were applied and a healthy male child was delivered. The presentation was occipito-posterior (4th cranial position), and moderate force had been used. There was no rupture of the perinaeum. At the time of delivery, and for some time afterwards, the patient complained of pain and stiffness in the left hip and extending for some distance down the limb. This passed off in a day or two, and the case became an ordinary one until the end of the fifth day. On that evening the temperature rose to 101·5°F., and, as the lochia were somewhat offensive, the vagina was douched out with a solution of lysol. On the sixth day after delivery the patient for the first time complained of shooting pains in the left lower extremity. The pain was most severe in the gluteal region and extended

downwards from below Poupart's ligament, on the anterior and inner aspect of the limb, as far as the calf. The pain was evidently severe and was accompanied by tingling and numbness. The affected muscles were tender on pressure, and the patient screamed when any attempt was made to move the limb. The glutei and Quadriceps Extensor muscles seemed to be most affected.

The pain and numbness continued to spread along the length of the limb in spite of treatment, and at the end of a fortnight implicated all the front and inner parts of the leg from the groin to the heel. She complained of muscular weakness and could extend the knee only with pain and difficulty. There was some retention of urine about the End of the first week, and on Examination the bladder was found to be overdistended with urine. Catheterisation was practised regularly until the End

of the 13th day, when she passed water without assistance.

From the fifth day until the end of the twenty-eighth day the temperature varied from 99.5° F. to 101° F. On the twelfth day she complained of some pain and tenderness in the right thigh. This was comparatively slight, and there was no paresis. Examination at the End of the third week disclosed the following conditions:— The left leg is in a position of partial flexure and adduction at the knee. There is some muscular wasting in the gluteal region and also in front of the thigh and in the lower part of the calf. The musculature of the right leg is less affected. Motor power in the left leg is impaired throughout. The muscles are tender on pressure, as is also the great sciatic trunk in its upper half, as well as the anterior tibial nerve-trunk. Left knee-jerk is much diminished. Plantar reflexes are normal, as are also the other superficial reflexes on both sides. Sensation on the left side.

is somewhat blunted especially over the dorsum of the foot. (The urine was normal.) Electrical reaction shows diminished irritability to both currents in the left lower extremity; there is no appreciable change in the right.

At the end of six weeks the condition was apparently at its height. She suffered greatly from sleeplessness, and the nurse informed me there was some delirium during the night.

From that time under active treatment there was a gradual improvement, the pain became less severe and the limb could be moved without the patient experiencing the former agony. After three months she was able to get out of bed and go about her room with assistance. I examined her again at the end of six months from the date of her confinement, and beyond some muscular weakness and a sluggish knee-jerk all the symptoms in the limb had disappeared. Since then

She has had one child without any recurrence of the neuritis.

Case 2.

L. B., unmarried, a primipara, aged 22 years.

Father and mother both alive and healthy. There is a history of tuberculosis in the mother's family. Four brothers and two sisters are alive and well.

Personal history.

She has always been delicate in health and there is a history of convulsions during early infancy. She has had treatment for indigestion and constipation frequently, during the four years previous to her pregnancy.

History of the illness.

I was consulted for a severe attack of hiccup and vomiting which came on suddenly. Under treatment the symptoms improved, and during my attendance I noticed she was apparently pregnant.

On inquiry the patient confirmed my suspicions, and I was informed she was in the third month of pregnancy.
In the course of a month or so I was again called in to see her and found her suffering from severe vomiting. On Examination She was found to be Extremely weak with a temperature of 100.5°F. She complained of tinging shooting pains in her limbs and back. The tongue was furred and the pulse full and bounding. There was obstinate constipation. I supposed I had to deal with a case of influenza — there was an Epidemic in the District at the time — and treated her accordingly. The bowels were kept freely open, and she was fed upon milk; but the vomiting resisted all treatment. At the End of a fortnight there was paresis of both lower Extremities, followed by muscular weakness in the arms and trunk. She also developed some bronchitis and a troublesome cough. At the End

of another fortnight — a month from the beginning of the second attack — the temperature had become normal and the vomiting much improved. She was quite unable to move from her bed, and could only raise herself to the sitting posture with pain and difficulty. There was disturbance of sensation in the arms and legs, with a sensation of "burning" and of pins-and-needles in the parts affected. She also complained of a sensation of cold constantly in her lower limbs. Both knee-jerks were much impaired and the superficial reflexes on both sides were slightly diminished. Cutaneous sensibility varied, being exaggerated in some places and diminished in others. Pressure over the nerve-trunks gave rise to pain. Muscular sensation also varied; sometimes it was almost absent.

I inclined to the belief that there was an element of hysteria in the illness, and I asked for a consultation; but the

friends of the patient were unwilling to go to the Expense.

As time went on she became markedly Emotional and would frequently weep for hours at a stretch. She seemed to be keenly alive to the disgrace arising from her condition, and kept constantly referring to her unhappy state. There were delusions and loss of memory present. The vomiting continued intermittently throughout the Entire illness.

In the middle of the seventh month of pregnancy I Examined her carefully and found much muscular wasting in the arms and legs. There was a tendency to "dropped feet", both being held in the Equino-varus Position. There was a trace of albumen in the urine. The knee-jerks were almost abolished.

At the beginning of the Eighth month labour set in suddenly, and when I arrived ~~at~~ at the house she had given birth to a living male child. The placenta was

Easily delivered, and I left her apparently in a fair way to recovery.
That Evening she was seized with Eclamptic convulsions, and continued to have fits every ten minutes throughout the night. I saw her next morning and found her comatose. She died suddenly on the afternoon of the second day without recovering consciousness. I asked permission to make a post mortem Examination, but it was withheld by the family.

Case 3.

Mrs D., married, aged 34 years, in her third confinement.

Family History

Her father's family was a healthy one. He was a healthy man, and was killed in an accident. Her mother is alive and has a valvular lesion of the heart: she has been an invalid for many years. One sister and one brother are living and are quite

healthy people. one sister is said to have died from consumption.

Personal History.

She had convulsions in infancy. At the age of seven she had scarlatina, and was ill for nearly a year. In her fourteenth year she suffered from acute rheumatism, and made a tardy recovery. The heart is sound. At the age of puberty she suffered from anaemia and chlorosis, and ever since she has been subject to periodical attacks of gastritis. Before her marriage there was frequent dysmenorrhoea.

History of illness.

Her first two confinements were normal, and she made quick recoveries.

On January 1st of ~~this~~ last year she was delivered of a female living child, after a normal course of labour. All went well until the seventh day when she complained for the first time of a severe pain in the body. The temperature went up to 100°.5 F — pulse 120. On examination the

uterus was found to be soft and swollen and Exceedingly tender. The discharge was scanty and somewhat offensive. She complained also of shooting pains in the loins and about the hips. Next day the temperature rose to 102°7, with a pulse rate of 130. That evening the pain was very markedly increased, and the lochia almost ceased to discharge.

As the condition was evidently getting worse, along with my assistant, I curetted the uterus under an anaesthetic and, having thoroughly swabbed it out with tincture of iodine, it was packed with iodoform gauze.

Next day, the 9th after delivery, the temperature settled to 99°7 —— pulse 100. The tongue had improved, and the tenderness of the uterus was not so pronounced. She still complained of pain in the hips and down both legs.

The condition slowly improved until the twentieth, when she had a severe attack

of pain in the forearms. This was accompanied by numbness and tingling. It increased in area, and by Evening it had involved both upper limbs from the shoulder downwards to the finger tips. There was severe shooting pain all along the limbs, with hyperaesthesia of the skin. During the night she complained of pain in both calves which was of a like character to that in the upper limbs. The sphincters were examined and found normal. Urine was normal.

Twenty-second day — the pain was still severe in all the limbs. There was some difficulty in swallowing and the voice had sunk to a whisper — but no regurgitation of food. There was paralysis of the soft palate. Temperature was 99.5°F, with a very rapid pulse which could be counted only with difficulty — probably 170. The mind was somewhat confused, and there were delusions. She complained of pain in her neck and down both shoulders. The legs could not be moved without very

acute pain.

Twenty-fourth day — There was apparent wasting of the muscles of both forearms and hands — especially in the posterior groups — as well as in those of the lower limbs. The paralysis of the muscles affected was complete, and extended to the muscles of both sides. The paralysis was not so marked in the lower extremities. The temperature was 99°7 — pulse 156. Examination at the end of a month disclosed the muscles of both forearms obviously atrophied. The posterior interossei in both hands were affected. In the lower limbs the peroneal group of muscles seemed to be most severely attacked and showed most atrophy. There was already "dropped feet" to a certain extent. The muscles of the calves and forearms were most tender to pressure. Pressure on the nerve-trunks of all the limbs gave rise to pain. The knee-jerks were diminished, the left one more so than the right. Sensation in the arms and legs was blunted,

especially in the lower halves. The muscular sense showed some perceptible diminution, chiefly in the lower limbs. The mind had cleared to some extent, but the memory was still defective. Hearing and sight were unaffected and swallowing and speaking had improved. The aphonia and paralytic dysphagia were the first to show a return to the normal condition. The urine was quite normal — sp. gr. 1018. The temperature was 99. 8°7 — pulse 154. Electrical reaction. The affected muscles in the limbs all reacted indifferently to the coil. At the end of another month an improvement was beginning to be manifest. This continued, and the affected musculature gradually regained its functions. At the end of two months the patient could leave her bed and be moved about the room, and she could now stand with some assistance. The pulse continued to be rapid for some time

longer — over 100 — and three months from the beginning of the illness she was quite restored to health. She then complained of cardiac palpitations.

Four months afterwards I again examined her, and, with the exception of muscular weakness in the lower limbs, she seemed to be quite recovered. I saw her recently,* eight months from the date of her confinement, and she informed me she felt quite well, but she could walk but short distances without feeling a return of the muscular weakness in both legs. The deep and superficial reflexes in all limbs were found to react normally.

* This was written on September 1908.

Discussion of cases.

Case 1.

This seems to fall into the traumatic group of peripheral puerperal neuritis. In it there was a prolonged second stage of labour, the head being arrested at the brim of the pelvis. The patient was a woman considerably under the average in size, with a small pelvis, and she was of a somewhat neurotic type. During the progress of the case she had frequently complained of cramps and pain in the lower limbs, and probably the foetal head pressed for some considerable time upon the lumbo-sacral cord where it crossed the brim of the pelvis.(8) It has been noted that injurious pressure is more likely to occur in a pelvis that is generally contracted than in a rachitic one, possibly on account of the fact that the nerves are protected by the projecting sacral promontory in rickets preventing undue pressure by the foetal head or by instruments. It

is in favour of trauma that the symptoms began so soon after delivery, and that the forceps had been applied when the head was high in the pelvic cavity. The distribution of the nerves traversing the female pelvis is considered under the heading of Traumatic Puerperal Neuritis. In addition to the compression of the nerve-fibres there was also sepsis. Possibly under ordinary circumstances the injury to the nerves would have been transient, but the absorption of toxins from the generative tract was sufficient to convert a mild into a severe condition. In other words, the poison exerted a selective power in attacking the injured delicate nerve-fibres. This frequently happens in cases of neuritis caused by alcohol or lead &c. The pain and wasting on the front of the thigh pointed to an involvement of the anterior crural nerve, the largest nerve springing from the lumbar plexus.

The superior and inferior gluteal nerves, and probably the small sciatic, were

also involved, as shown by the muscular wasting and disorders of sensation in the gluteal region. And, from the muscles affected in the calf and foot, it was evident that the lumbo-sacral cord and great sciatic nerves were the seat of neuritis. Probably there was more or less severe pressure upon all the nerves on one side of the pelvis, with slighter pressure upon some of the nerves of the opposite side. It is interesting to note that although she had been delivered with forceps in her first confinement no untoward consequences had resulted. The first child had been a female, and possibly smaller than the other. The history of previous eclampsia is also a point of interest and indicated an instability of the nervous system, along with a toxaemic condition of the blood during pregnancy. There was no suspicion of alcoholism in the case, or any other of the more common factors in peripheral

neuritis.

Case 2.

This was evidently the result of an auto-intoxication, the so-called toxaemia of pregnancy, and belongs to that group of neuritis considered under Toxic neuritis of pregnancy and the puerperium. It began, as most of such cases do, with vomiting, going on to hyperemesis, and the symptoms of nervous implication steadily increased until there was a multiple neuritis. It was characterised by paralyses of the regions affected, associated with muscular atrophy. The sensation of cold in the lower limbs and the pain and numbness, followed by loss of muscular power increasing to complete paralysis, are all extremely characteristic of this form of neuritis, and have been described over and over again by observers. The mental change (? Korsakow's psychosis) was also a characteristic feature in the case.

Careful examination failed to reveal organic disease of the stomach or intestines, and no lesion could be discovered during life in the brain or spinal cord. Albumenuria was present to a slight extent, but there were no casts or blood in the urine. Alcohol could be positively excluded as a causative agent. Death was probably the result of toxins acting upon the higher nerve centers.

Case 3.

This case is the most recent in my experience, the woman having been delivered on the first day of ~~the~~ last year. The personal history points to nervous instability, along with a tendency to alimentary troubles. The case falls into the group described as post-infectious or Septic puerperal neuritis of a very severe form. The symptoms of neuritis were preceded by urticaria which probably arose from absorption, either of poison from the uterus, or of

by-products from the alimentary tract. The urine was never at any time other than normal —— at least to the ordinary tests. Notable is the fact — as has been commented on by others —— that the pulse of temperature was never at any time out of the common. The pain for about a week was extremely severe, and the mental condition was very marked. Cases of neuritis following upon septicaemia are by no means so rare as some others. The case reported by (10) Korsakow and Derbski and discussed by (11) Eulenburg has been frequently quoted. Turney would exclude all such from puerperal neuritis and considers they should be entitled septicaemic neurites. But septicaemia only produces neuritis when it is severe and prolonged. In puerperal neuritis, as in my own case, the septic conditions need be neither severe nor unduly prolonged. In this case also, as in the others, I could find no evidence of an indulgence in alcohol.

The implication of the spinal accessory and vagus nerves — shown by the paralysis of the larynx muscles and the tachycardia — is also an interesting feature and one that is not always seen in these cases. There had been no hyperemesis or digestive troubles at any time during the pregnancy.

Explanation of diagram.

1 The first four lumbar nerves, with a branch from the last dorsal, constituting the lumbar plexus.

2 The four upper sacral nerves with the last lumbar ~~a branch from the last dorsal~~, constituting the Sacral plexus.

3 The two musculo-cutaneous nerves.

4 Genito-crural nerve.

5 External cutaneous nerve.

6 Anterior crural nerve.

7 Its muscular branches.

8 Middle cutaneous branches.

9, 10. Internal cutaneous branches.

11 Internal saphenous nerve.

12 Obturator nerve.

13 Gluteal nerve.

14 Internal pudic nerve.

15 Small sciatic nerve.

16 Greater sciatic nerve.

17 Internal popliteal nerve.

18 External popliteal nerve.

19 muscular branches.

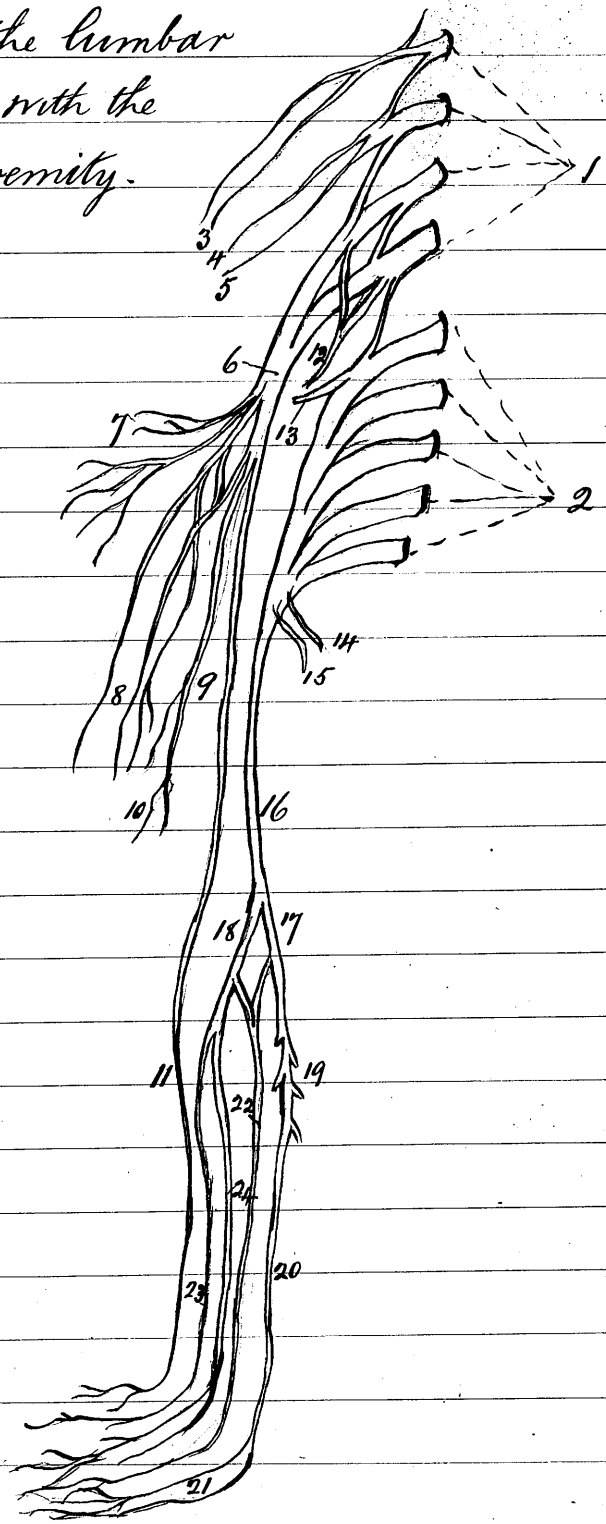
20 Posterior tibial nerve; dividing at 21 into two plantar nerves.

22 External saphenous nerve.

23 Anterior tibial nerve.

24 Musculo-cutaneous nerve.

Diagram showing the lumbar and sacral plexuses, with the nerves of the lower extremity.



Traumatic puerperal Neuritis.

This form of neuritis is usually the outcome of pressure upon the trunks of the sacral plexuses within the pelvis from forceps or the foetal head. Before considering the symptoms the anatomy of the Lumbar and Sacral plexuses of nerves may be recalled.

Anatomy of Lumbar and Sacral plexuses of Nerves. (Gray's Anatomy)

The Lumbar plexus.

There are five pairs of lumbar nerves. The roots of these nerves are much longer than those of any other spinal nerves, owing to the fact that the spinal cord terminates opposite the 2nd lumbar vertebra. They are also the largest of the spinal nerves.

The posterior branches of these nerves supply the Erector spinae, the interspinales and other muscles of the back, and also the skin of the Gluteal region.

Lumbar plexus, — continued.

The Anterior branches have communications with the sympathetic ganglia and pass through behind the psoas magnus muscle. The branches of the 4 upper nerves join together to form the lumbar plexus. The anterior branch of the 5th lumbar nerve unites with a branch from the 4th, and passing downwards across the sacrum unites with the anterior branch of the 1st sacral nerve and assists in the formation of the sacral plexus. This is the Lumbo-Sacral Cord.

The lumbar plexus, from the union of the 12th dorsal and the four lumbar nerves, is located in front of the transverse processes of the lumbar vertebrae. These various nerves are united by loops. The 12th dorsal sends a loop to the 1st lumbar, and this in turn sends a loop to the 2nd lumbar. From the 1st lumbar originate the ilio-hypogastric and ilio-inguinal nerves.

From the 2nd lumbar the External and internal inguinal nerves are given off.

Lumbar plexus — Continued.

The 3rd lumbar unites with the 2nd, and this trunk unites with the 4th to form the crural nerve.

The obturator nerve arises from three roots springing respectively from the 2nd, 3rd, and 4th lumbar nerves.

The 5th lumbar nerve has no connection with the other lumbar nerves, except by the loop which it receives from the 4th. It goes to form the lumbo-sacral cord, and forms a part of the sacral plexus.

The principal branches of the lumbar plexus are the following: — The ilio-hypogastric and the ilio-inguinal, the External and internal inguinal or cutaneous nerves, the crural and the obturator, and the lumbo-sacral nerves.

The Ilio-inguinal nerve emerges from the abdomen at the External abdominal ring in the male and the round ligament in the female. Company with the spermatic cord and is distributed to the skin of the scrotum in the male, and the labium in the female, and

Lumbar plexus. Continued.

to the upper part of the thigh.

The External Cutaneous nerve is largely a sensory nerve, and is distributed to the skin along the anterior and outer part of the thigh as far down as the knee, and also to the posterior third of the thigh as low as the middle third.

The obturator nerve supplies the obturator Externus and the adductors of the thigh.

The Anterior Crural nerve is the largest branch of the lumbar plexus. It supplies all the muscles of the front of the thigh except the tensor vaginae femoris, and sensory branches rise from it to the front and inner side of the thigh and to the leg and foot and also articular branches to the knee. Through the External Saphenous nerve it supplies sensory filaments to the front and inner side of the leg.

Thus the lumbar plexus supplies the adductors and Extensors of the thigh and leg, and gives sensory filaments to the anterior

Lumbar plexus - continued.

and interior surface of the thigh and leg

The lumbar plexus is not as a rule implicated in parturition, but its two main branches — the obturator and the anterior crural nerves — may be injured in the process.

According to (12) Winckel and others the obturator may be involved in pelvic Gradations from inflammations in the puerperium.

The symptoms of injury or disease of the obturator nerve are paralysis of the adductor muscles of the thigh and anaesthesia of the inner aspect of the thigh and leg.

The Sacral plexus

The sacral plexus is composed of the lumbo-sacral cord and the anterior branches of the three upper sacral nerves, and of part of the anterior branch of the 4th sacral nerve. It is triangular in shape, its base being situated against the sacrum with its apex towards the

Sacral plexus — continued.

Sacro-sciatic foramen. It rests upon the anterior surface of the pyriformis muscle and is protected by the pelvic fascia, which separates it from the viscera of the pelvis.

The Sacral plexus has 4 main branches — the superior gluteal, pudic, small sciatic, and great sciatic nerves.

The Superior gluteal nerve, the first main branch of the plexus, arises from the lumbo-sacral cord. It emerges from the pelvis just above the pyriformis muscle and has a superior and an inferior branch. The superior branch supplies the gluteus minimus and gluteus medius muscles. The inferior branch gives fibres to these two muscles, and then supplies the tensor vaginae femoris muscle.

The pudic nerve arises from the lower part of the sacral plexus and emerges from the pelvis below the pyriformis. It crosses the spine of the ischium and re-enters the pelvis and

Sacral plexus — Continued.

Gives branches to the dorsum of the penis, the perineum, and the External sphincter of the anus.

The Small Sciatic nerve arises by two roots from the lower part of the sacral plexus. It supplies one muscle, the Gluteus maximus, and gives sensory branches to the skin of the perineum and the back part of the thigh and the leg.

The Great Sciatic nerve supplies the skin of the leg, the muscles of the back part of the thigh, and those of the leg and foot. It is the direct continuation of the apex of the sacral plexus. Like the pudic and the small sciatic nerve it also passes out of the pelvis through the great sacro-sciatic foramen below the edge of the Pyriformis muscle. These three nerves as they emerge from the pelvis below the Pyriformis, occupy the following positions: The great sciatic lies farthest Exteriorly, then comes the small sciatic and then the pudic. The superior

Sacral plexus — continued.

Gluteal emerges from the pelvis just above the pyriformis muscle, so that the muscle not only is a basis of support to the whole sacral plexus, but also is a defence for these nerve-trunks as they issue from the pelvis.

The position of the lumbo-sacral cord is favourable for injury, as it lies directly on the bone. It contains the greater part of the fibres of the peroneal nerve (External popliteal) and the tibial nerve (internal popliteal). The fibres forming the peroneal nerve lie upon the bone, whilst the fibres forming the tibial nerve lie upon the peroneal fibres. Hence the peroneal fibres are much more liable to injury from pressure within the pelvis.

Sometimes there is a high origin of the peroneal nerve, the superior gluteal then arises directly from it. As we have seen the lower nerves of the sacral plexus are much less liable to injury, being protected by the pyriformis muscle.

Traumatic Puerperal Neuritis.

Among the most important causes of disease and injuries to the lumbar and sacral plexuses of nerves is childbirth. Long ago (13) Charpentier reviewed the subject and recorded the facts and theories by which authors attempted to explain them. (14) Romberg noted that paralysis could be seen accompanying certain diseases of the generative organs, and that it depended upon direct pressure upon the sacral plexus. He imagined there was a reflex origin of the paralysis. (12) Winckel recognised fully the neuralgias and paralyses of the lower limbs. He stated that injurious pressure may be caused by a large head in an unfavourable presentation in a small pelvis, and that the blades of the forceps may produce also pressure as well as severe contusions of the sacral plexus on forced closure, as well as during extraction. He also said that pelvic exudations and extravasations may give rise to nervous symptoms by extending

to the sheaths of the nerves.
(15) Stunermann and (16) Mills have written papers on the subject of puerperal paralyses.
Strist in a discussion at Philadelphia on Mills' paper made the statement that in a large experience of difficult labours and head injections he had seen but one instance of paralysis of the limbs as a result.
(17) Bianchi in a special treatise, the object of which was to establish the thesis that trauma acts as a cause of paralysis of the lower limbs in women during childbirth, regards these paraplegias as incontestable. He contends that the great sciatic nerve is incompletely protected from injury during labour, and that it is forcibly compressed in all labours, but to a variable extent.
In ordinary cases the pressure is exerted only towards the termination of labour, and is shown by cramps in the calves of the legs; but in certain long-continued cases pressure or contusion by the forceps may cause serious symptoms and even true paralysis can

supervene in the parts supplied by the sciatic nerve. Bianchi believes that the special conditions causing traumatic paralysis in labour are the use of forceps, a posterior position of the vertex, prolonged labour, and finally a contracted pelvis

(15) Hünerman considers that the chances of injurious pressure are greatest where the pelvis is generally contracted, as the promontory projects less and so allows the head to come in contact with the nerves. In the rachitic varieties he teaches that the opposite takes place, and the nerves are most protected. He Explains the common localization of the paralysis by showing that the External popliteal nerve receives fibres from the 4th and 5th lumbar roots and that these on their way downward to join the sacral plexus pass over the brim of the pelvis, where they are exposed to danger from compression, whereas the lower roots which lie upon the Pyriformis muscle are more protected.

(3) Hösslin says that neuritis may follow upon difficult or easy labours. Out of 80 cases collected by him, artificial aid to delivery was necessary 66 times, including 61 forceps cases. The remaining 14 cases included normal labour, precipitate labour, and face presentations. The large majority of the patients were primiparae, and many of them were elderly primiparae. Contracted pelvis was frequently present. The causation of injury in most was probably that the head remained too long above the brim, causing pressure upon the nerves of the lumbar or sacral plexus.

Symptoms.

The appearance of symptoms comes on during, or immediately after, labour. The localisation of the pain and paralysis will depend upon the anatomical facts, as already discussed. The early appearance of the neuritis is a proof that the condition arises from an injury, and not by an infection extending

from a metritis or pelvic cellulitis.

(18) Mills has divided affections of the sacral plexus into four groups: 1st Peroneal type; 2nd The sacro-distal and sacro-gluteal type; 3rd Neuritis, local or multiple, due to septic or other infection; 4th Neuritis with paralysis and pseudo-paralysis due to phlebitis (phlegmasia alba dolens), often septic but having special features.

The peroneal type is the only one that is interesting here, on account of the classification adopted in this paper.

(15) Hinerman largely devoted his paper to establishing this as a distinct type of puerperal paralysis. The symptoms of this type are as follows:— usually after protracted and instrumental labour there is severe pain in the course of the sciatic nerve, with a feeling of numbness and of tingling and creeping on the outer side of the calf, extending to the dorsum of the foot as far as the toes. The foot cannot be extended. Extension of the toes is impaired, and the inner border of the

foot is immobile. Flexion of the toes is usually not involved, and the movements of the knee and hip-joint are not impaired. In some of these cases there is no tactile anaesthesia. In long-standing cases the peroneal muscles waste and the reactions of degeneration occur. The explanation of the isolated paralysis of the peroneal muscles after instrumental delivery is given already.

The distribution of the peroneal nerve is as follows: It divides into the anterior tibial and musculo-cutaneous nerves.

The anterior tibial supplies the tibialis anticus, Extensor longus digitorum, Extensor proprius hallucis, and Extensor brevis digitorum muscles.

The musculo-cutaneous nerve supplies the muscles on the fibular side of the leg, and gives branches to the peroneus longus and the peroneus brevis muscles.

The sensory filaments of the peroneal nerve by its two main branches supply the great and second toes and the adjoining sides of

the second and third toes, the dorsum and inner side of the foot and the inner side of the ankle and all the toes excepting the outer side of the little toe. As a consequence of this distribution paralysis in the peroneal type involves the muscles which dorsiflex the foot and extend the toes. The sensory area involved includes most of the toes, the dorsum of the foot, the outer side of the ankle.

The tibialis anticus muscle is most likely to remain paralyzed. It elevates the inner border of the foot, dorsiflexing and adducting it at the ankle. The extensor longus digitorum extends the toes and dorsiflexes the foot.

The peroneus longus and brevis evert the foot and rotate it outwards.

Steinmann and Mills first clearly showed that in cases of paralysis following the high-forceps operation the lumbo-sacral cord is the portion of the sacral plexus most frequently involved, and that this is by

direct pressure or injury.
The neuritis arising from pressure or injury to the nerves may be limited to one side, or both may be involved. As a matter of experience it is found oftenest limited to one or other side. Where it passes from one side to another it is held that the cause is an inflammatory process set up in the tissues surrounding the lumbo. sacral cord. Mills considered it was due to the inflammation ascending to the roots of the Cauda Equina and passing from one to another by reason of their contiguity.
According to Hünermann's cases and others collected by Hösslin the symptoms are almost uniformly unilateral.
(10) Thomas reports a case in which both legs were affected. (11) Turney quotes a case recorded by Lamy in which both limbs became completely paralysed. The woman had been delivered by forceps and had experienced extreme pain at the moment the forceps.

were applied. Turney comments upon the subsequent long-continued paralysis of the tibial and peroneal groups in both legs, and suggests that the secondary neuritis in this case would not have occurred but for a morbid condition of the nerves. He considered, however, that the toxaemic Element was of quite subordinate importance in comparison with the trauma.

(26) Alldrich cites some instances of traumatic neuritis in the American Journal of Obstetrics.

My own case (Case I.) as already noted was probably traumatic, followed by sepsis.

In the sacro-distal and sacro-gluteal type of puerperal neuritis the symptoms are more of a neuralgic character, and there may be paralysis of short duration. Probably the paralysis is sometimes a pseudo-paralysis due to the inhibition of movement by pain. In these cases the anterior crural and obturator nerves may be involved, as

well as branches of the sacral plexus. If the nerves of the sacral plexus are involved the pain may radiate down the posterior aspect of the thigh and about the buttock.

The Toxic form of Puerperal Neuritis.

This form of neuritis which may be almost said to be the neuritis of pregnancy and the normal puerperium is seen usually associated with severe vomiting and other signs of a toxaemia. Before entering upon a description of toxic neuritis a short discussion upon the toxaemia of pregnancy may be introduced here.

The Toxaemia of Pregnancy.

The conception of a toxaemia in pregnancy has, within recent years, emerged from the state of speculation into something more definite, and is now spoken of as a distinct theory. Various opinions are held as to its pathology, but most authorities are agreed that it is a state of the blood and organism probably arising from the hepatic insufficiency so frequently associated with pregnancy; brought: pressed most commonly by trivial ailments, but exceptionally by severe and dangerous affections. The maternal blood contains

all that is necessary for the growth and support of the foetus in utero, and the liver, being the great Haematopoietic organ, is invested with far-reaching responsibilities. The influence of suppressed menses will go some way to the retention of toxic substances in the circulation. The woman may take more food than usual of a nitrogenous nature, and this, with the growth of the uterus and ovum, will also increase the katabolic work of the liver. Increased intra-abdominal pressure also favours the embarrassment of an overworked organ, while constipation, so commonly seen in connection with the pregnant state, favours the absorption of putrefactive products. To this may be added lessened Exercise, and so lessened respiratory activity, as well as the possibility of bacterial poisoning, addiction to alcohol &c. In the later months of pregnancy nitrogen and water are stored up to a very much greater extent than at any other time. The ureters may be pressed upon and the functions of

of the kidneys interfered with, so that the Elimination of the products of the foetal as well as the maternal organism is then liable to be diminished. In this way it is believed that pregnant women may suffer from the retention of metabolic products. (21) Bouchard considered that all pregnant women suffered more or less from auto-intoxication. Normally the destruction and Elimination of poisons are carried out so that no serious derangement of health results. In certain cases these processes may be imperfect. The French observers Bouchard and (22) Bouffe de Saint Blaise considered that all the abnormal manifestations of pregnancy rest upon some such basis, and that the slighter as well as the more severe conditions are all due to the same process. Thus headaches and pernicious vomiting may arise from like causes. Saint Blaise holds that the Conditions indicative of hepato-toxaemia are

1. Diminution in urea and increase in uric acid excreted. 2. The presence of Extractives ie. leucin, tyrosin, xanthin, and hypo-xanthin. 3. Urobiluria, peptonuria, indicanuria and albumen: uria 4. Glycosuria, the liver being unable to perform its complete glycogen: ic function if too much glucose be taken daily in the food.[23] Veit held the view that all the disturbances of pregnancy, from slight abnormal pigmentations to Eclampsia, result from Cytolytic processes following the entrance of Chorionic and foetal tissue into the maternal circulation.[24] Stone[25] Strauss and[26] Ewing group albumenuria, vomiting of pregnancy yellow atrophy of the liver, and Eclampsia under the heading of the toxaemia of pregnancy.[27] Williams demurs to these views, considering them erroneous, and he holds that characteristic differences Exist between the various Conditions thus grouped together. He believes they have

separate causes. The great difficulty in isolating certain specific poisons as giving rise to the conditions has hitherto been unsurmountable, and has led to much discussion and divergence of view. probably all the different theories contain more or less truth, but until the Exact Causative factors have been recognised it is only possible to attempt to demonstrate distinctive pathological lesions.

The Pathological Anatomy of the toxaemia of pregnancy includes alterations in the liver, Kidneys, and spleen. The peripheral nerves and the brain and spinal cord may also be affected, together with the thyroid gland.

In the liver great variations in extent and severity may be found. Necrosis and degeneration are frequently found. (Fatty metamorphosis to a certain degree is said to be invariably present in pregnancy). This may increase until

acute parenchymatous hepatitis is reached. Necrosis may develope in the foci of fatty degeneration until the condition goes on to acute yellow atrophy of the liver.

The blood-vessels may become involved and haemorhages and thrombosis result. Ewing regards necrosis of the liver cells as almost inseparable from the acute toxaemia of pregnancy. The necrosis may be limited to individual isolated cells in a lobule, or it may be so extensive as to implicate the whole lobule with the exception of a slight rim of cells at the periphery.

The Kidney appearances may also show great variation. During pregnancy they are under an increased pressure. Slight degrees of nephritis are sometimes assigned by the Germans to the "Kidney of Pregnancy". The existence of a specific pregnancy-kidney has been described by Von Leyden. According to this the kidney of pregnancy is an acute fatty infiltration

which does not affect the integrity of the organ, and which tends to disappear after delivery. This may be complicated with nephritis, which affection may also occur de novo. The acute toxaemia of pregnancy may on rare occasions be associated with an acute parenchymatous nephritis going on to atrophy.

Spleen. The spleen may show similar appearances to the liver and kidney when these are associated with toxaemia. The damage thus affected has been held by some to account for the obscure examples of anaemia and leukaemia which sometimes develop after the puerperium.

The blood ~~[illegible]~~ also shows characteristic changes resembling those of sepsis.(28) Thrombosis and Embolism sometimes occur, and have been held by some authorities to be especially seen in Eclampsia.

Nerves. Various disturbances of the nervous system occur during pregnancy,

from mild degrees to a true psychosis. The anatomical alterations are sometimes well-marked and are such as are found in neuritis arising from other causes.

Clinically the picture of the toxaemia of pregnancy probably depends in a great measure upon the intensity of the intoxication. The mild form as seen in the first half of pregnancy frequently resembles biliousness or hepatism. The gastro-intestinal symptoms include vomiting with nausea, various forms of indigestion and anorexia, constipation, and in some cases diarrhoea. The nervous and cerebral symptoms vary in intensity and may comprise nervous irritability, depression and alterations of character, headaches, hysteria, neurasthenia &c. The more severe forms include among other conditions pernicious vomiting, yellow

atrophy of the liver, Eclampsia, psychoses and polyneuritis. Of these, two are sometimes closely associated with each other, viz:— Pernicious vomiting and neuritis.

Pernicious Vomiting.

In the pregnant woman vomiting may be purely symptomatic in character and due to gastritis and other organic diseases of the stomach, the passage of gall-stones etc. Apart from disease, however, many women suffer from nausea and vomiting in the early months of gestation, the so-called morning-sickness. This has been compared to the morning vomiting of an alcoholic subject. It may continue throughout the entire pregnancy. Occasionally the vomiting becomes so severe as to be designated pernicious vomiting. According to [29] Pick it occurs once in 1,000 cases of pregnancy. In my experience and that of most General Practitioners who practise much

midwifery and with whom I have discussed the condition, it may occur much more frequently.

Aetiology. (30) Williams differentiates pernicious vomiting into three types, namely, reflex, neurotic, and toxic.

The reflex variety results largely from abnormalities in the generative tract. Retroflexion of the uterus or an ovarian tumour may cause it, and removal of the cause usually cures the symptoms.

The Neurotic variety has been described by (31) Kaltanbach who considered it due to a neurosis closely allied to hysteria, and readily amenable to suggestive treatment.

It is probably this kind of vomiting that is cured by applying various medicaments to the os uteri. At any rate, clinical observation goes to prove in my Experience, the soundness of Kaltanbach's view.

The toxic variety of pernicious vomiting is by far the most serious,

and has an important bearing upon the peripheral neuritis of pregnancy. In it there is a profound disturbance of metabolism as shown by changes in the urine and definite lesions in the liver and kidneys. (32) The urine as first demonstrated in America shows a much greater proportion of the total nitrogen excreted in the form of ammonia. Normally in the first half of pregnancy the ammonia coefficient varies between 4 and 5 per cent; in toxaemia it may rise as high as 20, 30, or even 40 per cent. In such cases, according to (24) Stone, lesions of the liver are found which are identical with those occurring in acute yellow atrophy. There is a necrosis of the central parts of the liver lobules, sometimes extending throughout the entire lobule with the exception of a band of healthy tissue at the periphery. In the

kidneys the secretory parts are chiefly affected and the epithelium of the convoluted tubules becomes necrosed. It is believed that the hepatic and renal lesions are due to the underlying toxaemia, and the destruction of liver-cells so interferes with proteid metabolism that much nitrogenous material fails to be converted into urea and is excreted as ammonia.

Pernicious vomiting usually begins insidiously and advances from the simple nausea and vomiting of pregnancy to severe hyperemesis. The condition may become still more severe, and the patient vomits large quantities of material like coffee-grounds (Black Vomit). Symptoms indicative of toxaemia supervene; torpor or violent excitement gives place to a condition of coma, sometimes accompanied by convulsions.

There are marked changes in the urine. This becomes scanty and may contain albumen and casts, and, in very severe cases blood. Sometimes the course of the disease is extremely rapid — a fulminant type — lasting only a few days. Blood stained material very soon comes to be ejected from the stomach, and the patient passes into a condition of coma and rapidly succumbs.

Diagnosis.

A correct diagnosis is most important in dealing with the vomiting of pregnancy, as the reflex and neurotic types can be readily cured, whereas in the toxaemic type probably it is better to induce abortion, so as to arrest the process before there has been irreparable destruction of tissue. Consequently a thorough examination of the Generative tract should be made, and any abnormality found should be corrected. Should no lesion be found the diagnosis lies between neurotic vomiting and

that due to a toxaemia. The ordinary tests applied to the urine are of no value as a guide to the Condition. Ammonia and urea give identical reactions by the Doremus method, so that an apparently normal urine may have a greatly reduced percentage of urea with a certain amount of ammonia and amido-acids. The amount of nitrogen can now be determined by Kjeldahl's method, and the ammonia by that of Schlössing, and then determining the ratio which the nitrogen contained in the ammonia bears to the total nitrogen a correct knowledge of the Condition can be ascertained. If the ammonia co-efficient be normal, the neurotic type is likely to be the one we have to deal with; if the ammonia be increased, and particularly if it much exceeds 10 per cent, a diagnosis of toxaemic vomiting should be made.

With regard to the period of onset of toxic neuritis (33) Reynolds has collected the records.

of 49 cases, including two cases reported at the time by himself, and he found that in 12 of them the neuritis was first noticed during pregnancy.[34] Turney found that out of 13 cases which he quotes and of which he made abstracts, 6 occurred during pregnancy or an average from the fourth to the fifth month. The remaining 7 at intervals varying from two days to a fortnight after delivery.

[35] Hösslin has made a collection of 92 cases from the literature of puerperal neuritis. Of these 36 began before, and 65 after delivery. The former only proceeded to full development after delivery.

Aetiology

Toxic neuritis is sometimes rarely seen after infections such as influenza and typhoid fever. It may arise when a macerated foetus is retained long in the uterus. A case recorded by[36] Korsakow and Serbski was that of a woman who was operated upon for a decomposing

foetus lying in an abscess in the iliac fossa, and in whom signs of neuritis came on afterwards. Sometimes neuritis during pregnancy or after delivery comes on where there has been a history of alcoholism. Reynolds suggests that, as during pregnancy and in the puerperium, not a few women take alcohol great care ought to be taken to exclude all such cases from a true toxic neuritis. For comparatively small quantities of alcohol will sometimes induce neuritis, and especially if the subject is not in robust health. No doubt cases of peripheral neuritis due to alcoholic poisoning have been included as puerperal neuritis. (37) Sandford's cases are supposed by (38) Lloyd to show unmistakably the action of alcohol. Both women were publicans' wives, and had been addicted to the excessive use of alcohol. But in a great proportion of the cases seen there is no history of alcoholism to be found and we are forced to seek for the cause in some

other direction. It has been pointed out also that alcoholic neuritis is never associated with paralysis of the cranial nerves, and in it there is never seen a severe affection of one limb without some symptoms in the other parts of the body.

In many cases of toxic neuritis there is a history of severe vomiting during pregnancy. (39) Of 92 cases reported in literature hyperemesis gravidarum was present 16 times. In 43 severe cases hyperemesis was present on 16 occasions, or 34 per cent. The conclusion to be drawn from these figures is — Firstly, hyperemesis and neuritis probably arise from a common cause; and, Secondly, hyperemesis leads to cachexia etc, and neuritis is very prone to develop. Either of these conclusions ~~is~~ is correct. The former is most likely to be the right one, for the conditions often arise spontaneously and sometimes the neuritis comes on before the vomiting. For the majority of cases of neuritis arising

in pregnancy and the normal puerperium nothing remains but to put them down to a toxin arising in the body. That is, they are due to auto-intoxication. The neuritis is very similar to other toxic neuritis cases, such as the alcoholic. Both have the occasional peculiar accompanying psychical changes. It was once considered that some exciting cause in addition to the auto-infection was necessary to produce the condition, this being found in the excessive vomiting or in delivery with its various Complications.(40) Nader stated that no case of neuritis during pregnancy has been recorded in which one or the other exciting cause could be excluded. Since then, however, cases have been reported in which neuritis developed during a perfectly uneventful pregnancy.(41) Lindemann held an autopsy on a case which was reported by(42) Solowieff and found extreme degenerative changes in the kidneys and liver which are characteristic of auto-intoxication.

In view of what we now know about the toxaemia of pregnancy it is almost beyond doubt that the neuritis of pregnancy is a result of the toxic condition of the blood. What the precise poison is we do not know at the present stage of knowledge; it is certainly a product of the anabolic or katabolic metabolism of proteids.

Symptoms.

The symptoms vary. Sometimes only one nerve-area or extremity is affected. The first, fifth, or seventh cranial nerves may be affected either alone or in combination with the nerves of the extremities. If the neuritis is limited to the upper extremity usually the median and ulnar nerves are attacked, but this is not so regular as in the post-infectious form. All kinds of combinations may exist, such as one arm and both legs; the right median and left leg may be affected together. Not rarely there develops,

in nearly half the cases, a general Poly: neuritis with severe symmetrical paralysis of all the Extremities. Sometimes the Sphincters, the phrenic nerve, and the larynx are involved. Disturbance of sensation, if it be extensive, is of grave prognostic import. An important symptom is (431) "Korsakow's psychosis". This was present in 16 out of 40 severe cases. (444) Stewart records a case of polyneuritis which came on during pregnancy, and in which also a poliomyelitis existed with changes in the posterior and lateral columns of the cord. The peripheral nerves showed the distinctive signs of a toxic parenchymatous degeneration. In the cord there was a scattered degeneration in the posterior columns, involving both Goll and Burdach's tracts. The author considers the condition was first of all a neuritis and later a localised myelitis (poliomyelitis). In this case there had been a history of severe vomiting. In many of the cases of toxic neuritis there

is a distinct history of previous nervous deterior-
ations. The powers of resistance of the peripheral
nerves are probably to a certain extent weakened and
the toxins are free to fasten upon them. The neur-
otic diathesis may be regarded as a predisposing
cause in the production of peripheral neuritis, as
of other affections of the nervous system.
Pathological Anatomy - The nerves have been
examined in several cases after death, and well-
marked evidence of neuritis has been found. In
the case already quoted which has been recorded by
Korsakow and Serbski degenerations were found
in both motor and sensory nerves, the ulnar,
median and phrenic nerves were much degenerated.
In (45) Solowieffs case, which was associated with vom-
iting, neuritis of the phrenic, vagus, peroneal
and median nerves was found. Stewart was
able to demonstrate a true parenchymatous de-
generation of peripheral nerves in his case, with
degenerations in the spinal cord pointing to a
poliomyelitis as well as a polyneuritis.

Neuritis per Contiguitatem.

This condition is seen secondary to parametritis and thrombosis of the pelvic veins. Here the neuritis is purely local, due to extension of the inflammatory Gaud: ate which involves the pelvic nerves. Von (46) Leyden has described such a contingency long ago. (47) Windscheit includes in this form those cases of neuritis following general pyaemia, in which there may be a multiple neuritis affecting all the nerves of the body, and not limited to the nerves of the leg. (48) Reynolds has placed upon record a case of atrophic paralysis of both legs in chronic pyaemia following labour, with a gradual recovery. (49) Stewart considers this group might well be designated septic neuritis (puerperal) with qualifying titles (a) local; (b) General, according to the distribution of the symptoms. One of the cases recorded by (50) Möbius was that of a pelvic inflammation coming on after delivery. The woman was

in bed for three weeks before she Complained of pain in her left calf, with pain and stiffness soon after in her right shoulder and Extending to the forearm and thumb. On Examination, there was paralysis with Complete reaction of degeneration of the flexor longus pollicis.

In these cases of pelvic phlebitis or phlegmasia alba dolens neuritis is the result of the inflammation spreading by Contiguity or by pressure. The neuritis may persist after the other symptoms have subsided. (51) Winckel quotes authorities to prove that in some cases of phlegmasia paralysis of the affected leg may remain. There is sometimes a pseudo-paralysis in these cases, the impairment of movement being largely due to the swelling of the legs, and not to a true paralysis. There may be localised pain in the leg, the great toe or the foot have been the seat of neuralgic pains. Or in other instances there

may be paralyses with atrophy of muscles and loss of sensation. Mills quotes cases in which such conditions existed. The paralysis in this form of neuritis is usually comparatively slight in extent and it develops slowly. It is not so complete as in the traumatic form of neuritis. The prognosis is very much better than in some other kinds of peripheral paralysis.

Neuritis post infectiosa.

This is seen in cases in which there is some form of sepsis. It does not usually occur after severe cases of pyaemia and septicaemia, but is more frequently associated with slight attacks. It involves mostly the upper extremities, especially the areas of the ulnar and median nerves. This selective influence in a poison is seen in the case of lead, as well as alcohol; the circulating poison in the puerperal type of neuritis evidently exerts an even more marked selection, the median and ulnar nerves being marked out for attack. Sometimes there is a severe multiple neuritis in this form, as in my own case (Case 3). Some observers have questioned the possibility of a wide-spread multiple neuritis due to sepsis, following childbirth. (52) Lloyd inclines to the view that such cases are always the result of alcoholism in childbed. In the case now reported by me in these pages alcohol could be definitely excluded as a factor in the disease.

Peripheral Neuritis — Myopathic.

1. Paralysis accompanying Osteo-malacia.

This kind of paralysis is supposed by some writers to be due to compression of the spinal cord or nerve-trunks from changes arising in the vertebral column or pelvis. Of this, however, there is no proof. Probably the true Explanation is that the same toxins which affect the bones also affect the muscles.

In the early stages of the disease there is muscular palsy, which is often accompanied by contractions of the adductor muscles of the thigh and increased patellar reflexes. Later in the disease rheumatoid pains are complained of in various parts of the body, and soon afterwards the bones become sensitive upon pressure, and softer. Various deformities appear which are particularly marked in the vertebral column and in the pelvis.

On examination, the affected muscles are found to be in a state of fatty

degeneration and atrophy, and there is a multiplication of their nuclei.

The disease may occur in any part of the World, but is rarely seen outside certain localities in the Continent of Europe. It may be said to be endemic in the Rhine Valley, the Engolz valley in Switzerland, the Olona valley and Calabria in Italy, and in the City of Vienna. It is rarely found in England or America.

Symptoms.

(53) The first symptoms are tiredness, muscular pains and tremors. Almost pathognomonic is the tendency for the occurence of Con. tractures in the adductor muscles of the thigh. These muscle changes appear very early, even before any signs of bone disease are evident. The paralysis also comes on very early and develops slowly.

The grouping of the affected muscles is characteristic:—— The first affected muscles are the great muscles of the

Pelvic girdle — the ileo-psoas, Quadriceps Extensorae femoris, the abductors of the thigh. The gait is 'waddling'. It is very exceptional for the smaller muscles to becme affected. The muscles of the shoulder-girdle are often affected.

Diagnosis.

There is a great resemblance to the juvenile type of Progressive muscular atrophy to which osteo-malacia is probably pathologically related. The electrical reactions are the same — quantitative, not Qualitative. In osteo-malacic paralysis there is no pseudo-hypertrophy. In progressive muscular atrophy the tendon-reflexes are always increased. Typical bony changes, in the case of osteo-malacia settles the diagnosis.

ii Polymyositis

This is a very rare disease. Apart from pregnancy it is sometimes seen in the infective fevers. It sometimes arises spontaneously, perhaps from an auto-

intoxication. Several cases have been reported and collected by Stöckel which occurred in combination with puerperal sepsis. It is said also to arise in normal pregnancy and during an apparently otherwise normal puerperium. It is sometimes fulminant and sometimes chronic.

Prognosis.

The danger to life in these forms of paralysis is comparatively slight. Complete recovery is the rule in the great majority of them, although this may be delayed for several years. The anterior tibial and peroneal groups of muscles show the effects longest.

The more extensive the paralysis and the quicker its development the greater is the danger of a fatal issue. Stöhlin computed that out of 50 severe cases 10, Equal to 20 per cent, were fatal. All localised forms get better.

Diagnosis

The diagnosis of a neuritis following upon disease or injury to the sacral plexus will depend upon a thorough Examination and Exploration of the parts. vaginal and rectal Examination usually give a clear indication as to the condition of the pelvic nerves. By rectum the nerve-trunks can be palpated and tumours

and inflammations in the region of the pelvis can frequently be demonstrated. The distribution of pain and paralysis depends upon the degree and extent to which the nerves are involved and are quite characteristic. The history of the case, the character of the labour — with instrumental delivery or septic complications — often clearly indicate the character of the lesion. Hysteria may simulate organic disease of the pelvic nerves. In such cases the characteristic stigmata of hysteria may be looked for and found, as segmental anaesthesia of the leg or hemianaesthesia involving both arm and leg. The mental state in hysteria is also an unfailing guide. In hysteria there is no degeneration in the muscles, and the paralysis usually affects all the muscles of the limb. The diagnosis of toxic vomiting and its sequelae is considered at page 67. It is necessary to exclude all other poisons. Excess in

alcohol is the commonest of all causes of peripheral neuritis, and strict inquiry should be made into the habits of the patient. Sometimes toxaemia and alcohol may co-operate, and it is very difficult to say which is mainly responsible for the condition of the peripheral nerves. There is no symptom, unfortunately, which is peculiar to the neuritis of pregnancy and the puerperium.

Treatment

The treatment will greatly depend upon the severity of the lesion and whether we have to deal with paralysis coming on before or after parturition. In the traumatic form the treatment will be similar to what has been found of benefit in other forms of neuritis. During the ingravescent stage the pain and tenderness do not permit of any active measures being employed. When the stage of irritation is over electricity and massage are the most effective means for restoring the functions of the parts affected. During the first stage of the treatment, while the nerves are functionless, the galvanic current may be used, afterwards the faradic current should be employed until the process of repair is complete. For contractures some kind of appropriate support will be necessary, otherwise there may be considerable shortening of tendons. Where the neuritis

follows upon pressure of the nerves from inflammatory adhesions in the pelvis an operation may be necessary in order to free the sacral plexus. In neuritis post infectiosa appropriate treatment must be directed to the septicaemia, afterwards applying the ordinary routine of electricity and massage to the affected nerves. The most difficult of all forms to treat successfully is the toxic variety. In a case of severe vomiting it is impossible, at the present stage of our knowledge, to foretell the onset of neuritis. Induction of labour does not always remove the toxaemia, for it very often comes on after delivery, or becomes worse after delivery. The toxin apparently develops sometimes in the puerperium. (54) Knaggs has placed on record a case of recurring optic neuritis in which abortion was brought on and the woman's eyesight was in all probability saved. The safe line of treatment therefore seems

to be to limit the interruption of pregnancy to those cases in which there is a danger to life, or where such a condition as optic neuritis is present.

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