

## PUERPERAL AND GESTATIONAL PARALYSES.<sup>1</sup>

BY

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VARIOUS forms of paralyzes may appear during gestation and may complicate any part of the puerperal period. Writers usually described these nervous affections as puerperal. It is obvious, however, that, both for convenience and accuracy, puerperal and gestational paralyzes should be considered apart. Hence in the following discussion all paralyzes occurring in the gestational period will be called gestational paralyzes, and all appearing during labor or the puerperal period puerperal paralyzes. Hysterical paralysis may occur in any form, and at any time during gestation or the puerperal period but will not be considered in this connection.

GESTATIONAL PARALYSIS may be due either to a central or peripheral lesion.

*Central Paralyzes (Gestational).*—Hemiplegia from cerebral lesion is rather common in pregnancy; it may be the result of cerebral hemorrhage, or less commonly from embolism; it is said to rarely occur from cerebral anemia. In grave renal disease complicating pregnancy, uremia has been known to produce cerebral paralysis without discernible lesion. Cerebral paraplegias have been reported, but are rare indeed.

Paraplegia of spinal origin may also complicate gestation. The literature shows, however, that its occurrence is extremely frequent.

*Peripheral Paralyzes (Gestational).*—Peripheral paralyzes occurring during pregnancy are neural in origin and are either toxic or traumatic.

The toxic varieties of gestational neuritis usually appear in women who have come to confinement weakened and cachectic.

Read before the Ohio State Medical Association, May, 1902

ome unusual drain upon nutrition, most frequently from red and intractable vomiting.

following report is typical of this form of neuritis; the case was exhibited to the Cuyahoga County Medical Society but some time since:

F. K., aged 27, a widow with three healthy children, the oldest of which is 6 years and the youngest about 10 months. She has not been very strong since her marriage. She has never had any children nor has she had any miscarriages. Her husband died on September 16, 1901, of acute tuberculosis. Careful examination reveals neither pulmonary nor cardiac disease in the patient.

During all of her pregnancies she has been very sick at the time and vomited continually from the second month until the commencement of labor. During the latter months of her last pregnancy she suffered from considerable swelling and much pain in the legs, and states that they felt numb, powerless, and heavy; the arms, however, were strong. This weakness, powerlessness, and numb feeling in her legs became more marked just previous to her delivery. Immediately after the birth of the child her legs became very painful and tender to touch. After the usual time in bed she attempted to get up, but found that, although the edema had disappeared, the legs were weak and numb. This weakness and numbness was particularly marked in the right leg. She now states that when she attempts to go up-stairs her legs feel heavy, and if she does not watch where she is going her feet slip on the carpet. Her legs are so weak and her ataxia so marked that she cannot go up-stairs unless there is a baluster for support, nor can she walk in the dark. She is not constipated and her sphincters are intact. She has no headache or backache, and no other affection except her inability to walk and the numbness of her feet and legs.

*Examination.*—She is intelligent, of medium height, and thin. Her general condition is good. The heart, lungs, abdominal and pelvic organs are normal. There are no ocular affections nor any weakness or paralysis of any muscle or group of muscles in the face or extremities. Tendon reflexes in the upper extremities are normal and distinct. The tongue is protruded straight; the labio-gonion fold is deepest on the right. The left pupil is larger than the right, both react promptly to light and accommodation; the

palpebral apertures are equal; the discs and fields are normal but a fine lateral nystagmus is present.

She walks with a distinctly ataxic gait, and marked foot clonus is present on the right side. She cannot stand with her feet closed; in fact, she stands poorly with them open. Both Babinski jerks are absent; front tap and Achilles jerk are also absent; plantar reflexes are absent; gluteal and abdominal reflexes are present. A well-defined anesthesia of the sole of the right foot and toes is demonstrable. There appears to be a distinct atrophy of the intrinsic muscles of the feet, which is most noticeable on the right foot, and there is evidently some atrophy of the anterior tibial and peroneal groups, most marked, however, on the right side.

Her sense of localization is not good. She thinks I am touching the great toe when, in fact, I am extending the foot straight out. On the anterior aspect of the left tibia is considerable periosteal thickening which looks very much like a specific periostitis though all other evidences and history of specific disease are entirely wanting.

Vinay<sup>1</sup> has reported a very similar case which is well worth a brief abstract: During the first months of pregnancy the patient lost a great deal of blood and during the latter months she suffered from hyperemesis, so that she came to confinement in a cachectic state. During the last two months of pregnancy she complained of paresthesiæ of the extremities and of pain and weakness in the legs. On the third day after delivery she had a chill with fever which lasted only forty-eight hours; on the fourth day she noticed that there was marked loss of power in the extremities. This rapidly increased until she was almost completely paralyzed. About six weeks after confinement she began to improve and four months from that time had fully recovered.

In some of these forms of neuritis it has been assumed with good reason, that circulating in the mother's blood is an unknown toxic agent which has a selective affinity for the nerves.

That something besides a cachexia or nutritional failure which conduces to the production of gestational neuritis in the cases reported by Elder appear conclusive. Both women were normal, neither being cachectic, anemic, nor exhausted. In both cases symptoms appeared at about the sixth month, and consisted of tingling and paresthesia, chiefly in the hands, but in one of the cases it was also in the feet. Sensation in both cases was

to some extent, but there was little or no muscular paralysis. No other known causes of neuritis could be excluded. After recovery immediately began and proceeded rapidly, one recovering in four or five weeks, and the other in about four weeks. Elder<sup>2</sup> takes the absence of hyperemesis, weakness, anorexia, which are usually characteristics in most cases of puerperal neuritis, as evidence that they are not necessarily concomitants, but expresses the opinion, which I believe to be well founded, that some toxin is elaborated within the body of the mother or child which has a selective toxic effect upon the nerve of the pregnant woman. Windscheit<sup>3</sup> states that the etiology of these rare cases is unknown, but they are supposed to be due to the action of some poison circulating in the blood. The clinical picture is usually that of a pure form of motor neuritis. The sciatic nerve seems to be selected. Usually there is a gradual development of the motor weakness of the extremities, pain is instantly complained of, but slight sensory changes may be present. The muscles usually atrophy and show the reaction of disuse. Other trophic changes have been observed to occur.

Wojewicki<sup>4</sup> and Polk<sup>5</sup> have reported fatal cases of peripheral neuritis occurring in pregnancy, and, since most of these cases do not recover when the uterus is emptied, one should be prepared to induce labor if there be indications of progressive and severe neural inflammation. Eulenberg induced labor in his severe case in order to save life.

The following personal observation possesses features varying somewhat from the preceding cases, but strong with suggestions of a case similar to that which Elder assumes in his case, that a clinical abstract will be of value:

Mrs. F., aged 36, family history good; has one daughter 7 years. She became pregnant in the early part of 1892. Worried very much about her condition and became quite morose over the fact that she should have another child when she had a daughter nearly grown up, and that it would also prevent her from attending the World's Fair at Chicago. She attended the Fair while she was in her seventh month, and while there noticed a beginning weakness and numbness of the legs. This grew worse, until she was able to be on her feet only a part of the day. She returned home and was confined nearly three weeks before her expected time. Her labor and puerperium were

normal. The weakness increased in her lower extremities was accompanied by some slight loss of sensation, considerable aching pains, and a very marked amount of ataxia. All the symptoms were distinctly those of a neuritis. Dr. J. H. Lovell concurred in the diagnosis of a gestational neuritis. She made a partial recovery, but still suffers some weakness and ataxia.

Since this form of neural inflammation usually terminates at birth, is it not possible that we have some toxemia as a result of the metabolic changes incident to the relations existing between mother and unborn child? Is it not possible that the neuritis is a direct result of poisoning from the blood laden with toxic matter from the failure of the established ratio between production and elimination of the poisonous emanations of the individual organisms?

Study of the literature of gestational and puerperal neuritis shows that many of the reported cases begin during gestation and become complete only after labor—truly gestational and entitled to be considered as transitional forms.

*Traumatic Neuritis (Gestational).*—Traumatic gestational neuritis has been assumed to exist by some authorities, and a re-amination of the literature seems to afford grounds for their claims. In placing it in this classification I wish to state my belief that some of these cases are ischemic paralyzes due to the pressure of the fetal parts upon the vessels of the mother, or to pressure neuritis caused by an edema of the nerve sheaths produced by disturbance of the pelvic circulation.

It is a well-recognized fact that a mixed nerve may temporarily lose its power to conduct motor impulses from its nutrition disturbed by any cause which either deprives it of blood or unduly congests its delicate elements. The same effect may supervene from the elastic and slowly developed pressure of exudations into contiguous tissues or even into the nerve sheath, or from simple elongation of the nerve by stretching. However, the pressure remains too long or becomes too great, the anemia or congestion continues too long, inflammation and degeneration of the nervous tissues will occur, with final involvement of the sensory power of the nerves, and paralysis of permanent character supervene.

Since the days of Albrecht von Haller<sup>6</sup> it has been known that the interruption of the blood supply of the lower extremities

cause them to become paralyzed and stiff. Later the great man<sup>7</sup> called the attention of the surgeons of the world to that rigidity and stiffness with contracture of the muscles frequently followed too tight bandaging, and conclusively stated that it was not pressure upon the nerve trunks, but the result of interference with the blood supply, that is now called ischemic paralysis.

It is the belief of the writer that some of the transient paralyses which have been noted to complicate gestation and to follow labor and have been formerly ascribed to reflex, dynamic, and spastic causes, were due to an ischemic paralysis which was produced by interference with the circulation from too long pressure of the head in the inferior strait, the pelvis being either narrow or misshapen, or containing a head whose proportions were too great, particularly in certain diameters; or perhaps the uterus exerted too much pressure upon the large arterial trunk above the brim of the pelvis or upon arteries pursuing an unusual and anomalous course.

The above view will probably explain some of the accounts of transient paraplegias which followed labor and recovered in two or three days. The disturbed circulation of the lower limbs during pregnancy is more likely the cause of the muscular cramps which precede and accompany labor than pressure upon the nerves, since it is a well-recognized fact that the portion of the brachial plexus which goes to the formation of the external popliteal nerve is more likely to be affected than the nerve supplying the calf muscles, which are well known to be most frequently the seat of cramps both before and during labor.

It is the opinion of the writer that some of the reported cases of transient paralysis of the nerves of the upper extremities immediately following labor may have been due to stretching the nerves by ill-directed muscular effort during the agonies of travail, or by the position of the member while under anesthesia.

There is also a case of pain, loss of power, and formication occurring during labor, due to the distribution of the ulnar nerve, which immediately followed severe but normal labor in a very excitable and muscular woman. This nerve lesion was thought to be due to pulling on the arms by bright rods in the bed above her shoulders, thus exercising excessive tension upon the branches of the brachial plexus.

**PUERPERAL PARALYSES.**—All those paralyses occurring during labor and in the lying-in will be classed under this general head.

As in the paralyzes of gestation, the paralyzes of the puerperium may be divided into central and peripheral.

*Central Paralyzes (Puerperal).*—Hemiplegia complicating the puerperium following labor is not particularly uncommon and the prognosis is usually good. I recall a patient who was confined by Dr. Lee, of Cleveland, who some hours after labor developed a paralysis of the right side, with considerable loss of speech, however, made a perfect recovery in less than eighteen months. The lesion was undoubtedly embolic, probably originating in the dislodgment of a small bit of fibrin or a fragment of a thrombus of blood from one of the uterine sinuses. While embolism is the most usual cause of cerebral palsies in the puerperal period, the occurrence of hemorrhage as the result of the hydremia of pregnancy has been recorded.

Cerebral monoplegias from like causes may occur, but a survey of the literature shows them to be so excessively infrequent as to suggest a coincidence rather than a complication.

It is also stated that a transient puerperal paraplegia due to exhaustion of the centres of the cord may occur. I have never met with such a case, from which all the organic and hysterical elements could be confidently excluded, that was not directly ascribable to a disturbance of the circulation of the lower extremities.

Mills<sup>8</sup> reports a case of paralysis of both legs which followed soon after labor and which was due to a transverse myelitis. In the presence of the septic conditions which complicate the puerperium the occurrence of a septic myelitis is not only possible but has been observed. Handford<sup>9</sup> has reported a case which is evidently diffuse myelitis resulting from puerperal sepsis.

*Peripheral Paralyzes (Puerperal).*—The peripheral paralyzes which occur during labor and the lying-in period are of two origin and for convenience of discussion may be divided into two classes which will to some extent indicate their cause, *traumatic* and *non-traumatic*. Traumatic is used in relation to the nerve tissue alone, and not in reference to the trauma of other tissues during labor, which open them to a septic inflammation and thus by a traumatism produce septic neuritis.

*Non-traumatic neuritis (puerperal).* The non-traumatic paralyzes of puerperal neuritis are either (1) *septic* or (2) *non-septic*.

(a) Any nerve of the body may be inflamed during a puerperium. Instances of neuritis of the median, ulnar,

nerves have been recorded as occurring as a result of puerperal sepsis.

Cases of extensive polyneuritis have been observed to precede or follow a septic lying-in period.

Many of these forms undoubtedly depend upon a true degeneration of the nervous tissue itself or septic inflammation of the perineurium. Quite a large percentage of the cases are localized in distribution. They are undoubtedly examples of ordinary septicemic neuritis, and literature contains not a few classical examples. The following case, quoted from Reynolds<sup>10</sup> affords an apt example:

*Chronic Pyemia Following Labor; Atrophic Paralysis of Legs; Gradual Recovery.*—Mrs. H. was quite well until her first child was born, in 1892, when she was 18 years of age. The puerperium took place in a Manchester hospital and she told me shortly afterward she was very ill and was operated on for an abscess. She was then removed to the Manchester Royal Infirmary, and I am informed by one of the late resident surgeons that she was found to be suffering from chronic pyemia and paralysis of the legs. She was removed to the workhouse and came under my care early in 1893 in the Manchester Workhouse Infirmary. She was then suffering from total atrophic paralysis of both lower extremities, with contractures of the feet; there was absence of knee jerks, but the arms and senses were unaffected. She was treated by massage and was gradually improved in eighteen months. At the present time (June, 1897) she is practically well and can do all her ordinary housework. She walks a little lamely because of the rigidity of all the joints of the toes into the soles of the feet, but all other muscles of the lower extremities are normal and well nourished. There was no previous history of alcoholism. There are occasionally occur examples of puerperal neuritis in women who have suffered no traumatism at labor nor have had septic symptoms. That they should be regarded as non-septic and non-septic is proved by the occurrence of similar cases previous to labor. These cases were first described by Reynolds<sup>11</sup> and later by other observers, especially Eulenberg,<sup>12</sup> who distinguished two forms:

*A localized form*, in which only one or two nerves are affected—in the *arm type*, the median and ulnar; in the *leg type*, the peroneal. Other isolated nerves have been found affected, even



the optic nerve. Pain, sensory or circulatory disturbances exceptional, but atrophy of the muscles and reaction of degeneration commonly takes place. While many of these cases cover, there are not a few of them which remain permanently crippled.

(2) *A generalized form*, in which there is widespread neuritis or inflammation which may take the ascending type and closely resemble Landry's paralysis. The cranial nerves have been observed to be affected in these cases, and death results from involvement of the respiratory nerves.

While some of these cases, as has been stated, present isolated neuritis, others widespread neural degeneration, yet they rarely develop the severe and rapid forms which are met with as a result of sepsis or other infections provocative of high fever and constitutional disturbance commonly noted in the so-called neuritis of infections.

Every grade of this form of neuritis may be met with, from a transient tingling, numbness, and weakness in the distribution of a single nerve to widespread paralysis with consequent atrophy, contractures, and permanent disability.

A recurrent type of neuritis has been noted to appear at each labor. The following case, reported by Rhein,<sup>12</sup> is of this type:

The patient was a healthy woman who, after a normal labor, began to suffer pain and numbness over the flexor surface of the left forearm. Weakness of the flexor muscles manifested itself. This condition remained with little change until after her second uncomplicated labor, when the symptoms and disability became more intense. Finally, after a third normal labor, the other arm became the seat of a like trouble and the left arm grew still worse. Examination revealed inability to raise the hand to the head; flexion of the fingers weak; slight loss of sensation over the flexor surface of the forearms; atrophy of the intrinsic hand muscles; nerve trunks not tender to pressure.

*Traumatic neuritis (puerperal).* The most frequent and important varieties of puerperal neuritis are those which are traumatic in origin and either the direct result from injury received by the nerve itself or from pressure exerted by exertions into contiguous tissues, thereby producing a temporary interference with its normal conductivity from a disturbance

molecular continuity rather than an actual disintegration of elements.

For a clearer conception of this subject these traumatic varieties of neuritis are best considered under two separate heads: (1) those which are strictly contusional in origin, and (2) those which result from pressure from exudations, which exudations may be septic or septic in origin.

The following case will serve to introduce us to a study of a variety of neuritis which I believe to be of contusional origin and very probably ascribable to the wounding of a part of the sacral plexus by the forceps or parts of the child during labor. The case was referred to me by Dr. Geib, of Cleveland:

Mrs. L. C. W., aged 28, after a normal pregnancy came to confinement November 9, 1900. After fifteen hours of labor, was delivered with forceps by Dr. W., without the aid of anesthetics, a laceration resulting. *Immediately after the application of the instrument she felt a sensation of weakness and paralysis in the right leg and thigh.* Following her delivery the leg remained motionless, became cold and numb, with considerable loss of sensation below the knee. She suffered but little pain, except in the lower back. The control of the urinary sphincters was lost at once and is still weakened. The anal sphincters were not affected. She had no chills nor did any fever develop during the convalescing-in period. Although the left leg felt cold, numb, and motionless, it has never been very sore nor tender to touch, and she has suffered but little pain except when she attempted to rise.

She was on crutches for over three months. Dr. Geib saw the case about eight weeks after the confinement, and, recognizing its peculiar features, very kindly referred it to me for examination.

*Examination.*—The patient is under-sized and the pelvic dimensions are all under normal. She walks with a slight limp. The right leg is found to be cold, but little discolored. The patient states that the toenails on the right foot do not grow as fast as on the left. The shin line is very plainly seen on the right leg, revealing the fact that the anterior tibial and peroneal muscles are considerably atrophied. The extensor hallucis, extensor pollicis, extensor brevis, and minimi digiti are intact.

The calf muscles are strong and not wasted. The knee jerks on both sides are equal and active; Achilles jerk is marked on the right but faint on left; no ankle clonus on either side; front tap-

present on the left, absent on the right. Three and one inches below the patella the right leg measures  $10\frac{1}{4}$  inches; left  $10\frac{3}{4}$  inches; five inches below the patella the right measures 10 inches, the left  $10\frac{1}{2}$  inches; seven inches below patella the right leg measures  $8\frac{1}{2}$  inches, the left 8 inches. Measurement of the forearms shows that the left is one inch smaller than the right. The patient being right-handed these measurements show the loss of nearly an inch in circumference of the right leg. The peroneus longus contracts slightly to a powerful faradic current, but no contraction of tibialis anticus can be secured. To the galvanic current peronei and tibialis anticus show the reaction of degeneration which is particularly marked in the latter. There is slight numbness of sensation on the dorsum of the foot, which continues up the outside and anterior aspect of the right leg and corresponds to the cutaneous distribution of the external popliteal nerve.

This case is so typical of a form of paralysis which occurs in the distribution of the peroneal nerve following labor, of traumatic origin, that I will give a brief recapitulation of similar cases which I have previously reported:<sup>14</sup>

*CASE II. Prolonged Severe Labor Terminated by Forceps; Right Peroneal Paralysis, Anesthesia, Paresthesia, and No Recovery from Neuritis and Sensory Disturbances, but Persistence of Paralysis and Atrophy.*—Mrs. L., aged 26; long and difficult labor; forceps and chloroform. The morning after delivery much pain in the right leg and hip, with inability to lift the member. All of her symptoms increased in severity, accompanied by all the distressing symptoms of a severe neuritis. The pain was manifested entirely in the distribution of the external popliteal nerve. Some anesthesia was present on the dorsum of the foot and toes. Eighteen months later the patient was re-examined and there was found considerable loss of volume in the right anterior tibial and peroneal groups.

*CASE III. Prolonged Labor; Instrumental Delivery; Slow Left Peroneal Neuritis with Paralysis and Atrophy; Limited of Tactile Sensation; Slow Recovery, with Weakness and Atrophy of the Anterior Tibial Persisting.*—Mrs. E., primipara, aged 30; small woman with contracted pelvis. Labor severe and fourteen hours in duration; terminated with forceps and chloroform. In the evening her accoucheur was sent for on account of severe pain in the left hip and leg, also numbness and tingling

outer and anterior aspect of the right leg. There was also power. The following week the sensory paralysis re- about the same, but the motor symptoms increased until was practically useless. She suffered no chills nor rise perature, nor could a pelvic exudate be demonstrated. -two days following her labor I examined the patient and to evidence of pelvic mischief, except the exquisite tender- the pelvic nerves. Quantitative and qualitative changes galvanic current were present in all the muscles supplied peroneal nerve. Examination disclosed inability to dor- x the foot or extend the toes. She could depress the outer of the foot, but could not draw it up. There was loss of sense on the dorsum of the foot and lower part of the and posterior aspect of the calf. Atrophy supervened, but ally made a good recovery. This patient has had two a since this case was reported, both of them being much children, and in each instance she has suffered a repeti- this attack, but much less severe than the first.

following case of extensive paralysis from injury to the plexus during an ill-conducted forceps delivery was seen r. R. E. Skeel, September 5, 1899:

P., aged 30, an under-sized woman with small conjugate r. Has been married nine years and has had two chil- One boy, now aged 6 years, was born after a severe labor id of forceps—a very small child, weighing but three . She states that she suffered a phlegmasia alba dolens her lying-in period. Her second pregnancy was normal e came to confinement February 25, 1899. She was in irty-six hours, during which her pains were strong. Her an then applied forceps, which she and her husband state ot on for a period of three hours without result; no anes- was administered. The forceps was then removed and a interval of three hours was reapplied, and after another ours of active traction, during which but little chloroform en, she was delivered of a dead child whose head was ed by the forceps and the under jaw broken. Evidence ot show that the child was large. Immediately after her y it was found that the sphincters of the bladder were ed, and she also experienced difficulty in securing a move- f the bowels. Her greatest suffering, however, was from out the hips and pelvis extending down to the posterior

portion of the legs; there was also a burning pain and sensation on the outside and anterior aspect of each leg and sum of the foot. The limbs were swollen, cold, and useless. Nothing would control the pain and she became a morphine habitué. About three months later she came under the care of Dr. [redacted] to whose courtesy I am indebted for the privilege of examining the case.

I found her lying in bed with marked contractures and considerable atrophy of the hamstring muscles, which was marked on the right side; complete paralysis and wasting of the anterior tibial and peroneal groups on either side; sphincter control had been regained. There was some numbness and tingling in the legs and feet. A complete foot drop was present on each side. Pelvic examination revealed great tenderness in the region of the sacral plexus, and a separation of the symphysis pubis sufficient to allow the finger to drop between the ununited ends. Involution had taken place; no evidence of pelvic exudate could be detected. This case was extremely interesting from a number of standpoints, but the greatest interest to me was the paralysis, atrophy, and loss of sensation, marked in the distribution of the external popliteal nerves.

That the paralysis in these cases is due to an actual contusion of some part of the sacral plexus during labor seems to require no further demonstration. When we take into consideration the very sudden onset of the paralysis with all the characteristics of a neuritis, the distribution and course of the paralysis, it is impossible to conceive of any other hypothesis which is adequate to explain its development. Indeed, the not infrequent occurrence of this particular type of paralysis following labor presupposes a common origin and one intimately related to the physiological process of parturition. Being a sequel to a difficult labor causes us to immediately suspect mechanical injury to some of the component or contributing parts of the sacral plexus within the pelvis, and a resulting descending necrosis of those nerve fibres which ultimately emerge from the sacral plexus to form the external popliteal or peroneal nerve. In order to appreciate the mechanism of the injury it is necessary to refer briefly to the anatomy of the parts.

You will recall that part of the fibres from the fourth and fifth lumbar segments unite to form the lumbosacral cord, which passes downward winding over the sharp

linea innominata into the pelvis, where it contributes to the formation of the sacral plexus. Since from the apex of the plexus arises the great sciatic, from which the peroneal or the external popliteal nerve is given off, and since the sacral plexus is formed by the junction of the first, second, third, and part of the fourth sacral nerves with the lumbo-sacral cord, it follows that any injury to the lumbo-sacral cord will result in damage to the branches of the great sciatic.

If the lumbo-sacral cord fails to join the plexus, but continues as a separate trunk down alongside the great sciatic to become the peroneal nerve without any plexal connection. Mills,<sup>16</sup> Bardeen,<sup>16</sup> and others have shown that the fibres which mainly form this lumbo-sacral nerve preserve their identity and are given off as the external popliteal or peroneal nerve. From these facts Hünemann<sup>17</sup> was led to believe that this form of leg palsy was caused from injury to the lumbo-sacral cord, and that the contributing causes were (a) an abnormal position of the nerve, (b) a lack of proportion between the head and pelvis, (c) a faulty position, (d) or careless instrumentation. Bardeen,<sup>18</sup> after a study of two hundred dissections, found that the common formation of the external popliteal nerve is from the fourth and fifth lumbar and the first and second sacral nerves (64.5 per cent). The common formation of the internal popliteal nerve is from the fourth and fifth lumbar and the first three sacral nerves (64.5 per cent)."

In view of the fact that these cases present a paralysis so well delimited to the distribution of the external popliteal nerve that injury to the internal popliteal renders their explanation not as simple as it appears, and, as Thompson suggests, Bardeen certainly fails to explain its occurrence as completely as science demands.

Thompson<sup>19</sup> quotes Bardeen, who has suggested a possible explanation which the former puts into the following words: "The upper roots of the sacral plexus do not lie upon the pyriform muscle, but against the bony wall of the pelvis, and are thus liable to injury from pressure during certain difficult labors. The dorsal offsets of these roots which lie against the bone which receive the chief injury. The external popliteal nerve arises up from these dorsal offsets, and therefore the paralysis is chiefly localized in the distribution of this nerve." I think

that this is on the whole the most scientific explanation of the action of traumatic force in producing this form of neuritis.

We have yet to consider those cases which occur from a developing pressure from exudation, which certainly may be looked upon as traumatic in origin. These cases possess considerable interest and may result from septic or non-septic exudates. As an instance of a pressure paralysis from a septic exudate, the following case was recorded by Hervieux<sup>2</sup> more than twenty-five years ago, and is a thoroughly scientific contribution, and, considering the time of its report, stamps its reporter as a man of great scientific attainments and powerful observation:

A healthy woman of 19 years experienced a natural delivery. On the sixth day pelvic pains, fetid lochia, and fever announced the onset of puerperal infection. The septic condition was marked two days later by a dry tongue, rapid pulse, increasing pains in the abdomen, and a typhoidal aspect of the face. On the thirteenth day there appeared loss of power in the legs; at this time she had great fever, pulse 128, rapid respiration, a distended abdomen, abundant lochia, and an erythematous face. Later pain developed in the left iliac fossa and meteorism increased; and temperature remained high, patient exhibited a dry tongue, loss of power and profuse perspirations. On the sixteenth day it was noted that she was unable to lift the lower limbs; there was no anesthesia nor involvement of the upper limbs. Tenderness and phlegmonous indurations could be demonstrated in the left iliac fossa; they seemed to be in relation with the adhesion to the left of the uterus. Under blistering and Neapolitan treatment she improved, and as her general condition mended she began to return in the legs; the pelvic indurations were gradually absorbed, but the patient continued to complain of vague pains in her thighs. At the end of two months the pelvic induration had disappeared, she had regained the use of her limbs, and was discharged from the hospital in a good way to recovery.

This is an apt illustration of septic infection producing endometritis and pelvic cellulitis marked by large exudates which evidently involved the nerve trunks, producing a paralysis due to pressure. That we may thus confidently assert its origin is further shown by the fact that the motor nerves were principally involved, the only sensory symptoms being the neuralgic

Moreover, the recovery occupied a space of time too short for a complete regeneration of the nerve tissues.

Paralysis following the peroneal distribution occasionally develops several days after labor, sometimes occupying a week before it is fully evolved, oftentimes presenting no evidence of inflammation and examination reveals little or no sign of pelvic exudation. Mills,<sup>21</sup> Dercum,<sup>22</sup> Sinkler,<sup>23</sup> and others have reported such cases. The following is an abridged report of the first of a series of cases reported by me in 1899.<sup>24</sup>

*Prolonged Labor; Right Peroneal Paralysis and Neuritis with Extension to the Other Side; Slow Recovery from Neuritis, but Persistence of Some Atrophy and Paralysis.*—Mrs. T., large, robust woman; second marriage; by first husband had one small child whose delivery was easy and normal. Second husband is a young man, and by him this was her first pregnancy, which had an uneventful labor. Her labor lasted ten hours, during which she experienced almost continuous pain, but terminated naturally, the child weighing nearly twelve pounds. Three days after confinement the pains began in the right groin and hip, extending down to the foot and toes, which were found to be slightly swollen and tender to touch; loss of power was apparent. No intrapelvic inflammation or inflammation could be detected; lochia were normal; temperature raised 1.5° F.; no chills. During this time she was under the care of Dr. Walker, of Carleton, Ohio, who states that she had much pain in the groin and leg. No anesthetics were used, but a distressing burning sensation was felt on the outer side of the right calf, the dorsum of the foot and toes. About ten days later she had a similar but much less severe pain in the region of the left leg and foot. There was no loss of sensation on either side; legs were sensitive to handling, considerable motor power in both, but most marked on the right. In eight days she walked without assistance, and in two and one-half months the sensory disturbance had disappeared. Three months later atrophy was present in the anterior tibial and peronei of the right leg.

Winkel<sup>25</sup> states that even such slight pelvic exudations as accompany phlebitis may compress the nerves, and, still more important, small extravasations into the nerve sheaths may occur. Hermann<sup>26</sup> refers to a case of severe sacral neuralgia where he disclosed numerous thrombotic veins in the pelvis, one



of which lay alongside of the sciatic, to which it was bound inflammatory deposits.

612 PROSPECT STREET.

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