1932

Peripheral neuritis complication pregnancy

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PERIPHERAL NEURITIS COMPLICATING PREGNANCY

SENIOR THESIS

April 15, 1932

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INTRODUCTION

Pregnancy is presumably quite a perturbing condition since it interferes greatly with the usual bio-chemical processes of the body, originates new reactions, and compells organs built to do certain things in a certain way to take on a new and temporary function. One complicating factor is that nature uses a makeshift apparatus. Comparison of the remains of extinct animals with the living forms proves and illustrates this fact. Man evolved, he was not created. Nature, when making man, instead of creating a wholly new machine took an old one, our prehuman ancestor, added new things to it, modifications and improvements, and made the old do new work. The human body is an old house to which new parts have been added to modernize and improve, but in which much of the out of date furniture has been allowed to remain. The old machinery coupled with the new cannot work without friction.

All the nervous abnormalities that occur during pregnancy are seen also in various other conditions; all of them, however, except pregnancy itself are pathologic. The occurrence of neuritis following exposure to various toxic substances, notably alcohol, lead, and arsenic, is rather frequent, and it

is not uncommonly observed as a sequel of the more severe infectious diseases. The incidence, however, of this disease in pregnancy resulting from the toxemias, pelvic pressure, or pelvic inflammatory conditions may be said to be quite rare. A search into the various texts of neurology, psychiatry, and obstetrics is rewarded only by scattered references, the authors evidently believing that the subject was included more or less completely in some other category. Nevertheless, quite a considerable literature has developed on the subject.

Peripheral neuritis, multiple neuritis, and polyneuritis are all terms applied to a complex of symptoms due to disease affecting the peripheral motor and sensory neurons. Feiling (7) states that the disease as a whole is characterized by the fact that one nerve or many nerves are affected at the same time or in rapid succession; that if more than one nerve is affected, the affection of the nerves is always bilateral and usually symmetrical; and that the more distal parts of the neurons are especially attacked, producing symptoms largely limited to the distal parts of the limbs. It is important, however, to realize that the conception of the disease as one limited always to the peripheral nerves is erroneous. As will be pointed out, the action of the infection or intoxication falls, in many cases

at all events, on the central as well as the peripheral nervous system. In some cases indeed other systems of the body, and especially the heart, are equally affected. The term "neuritis" is misleading, since it conveys the idea of an inflammatory lesion. The essential change, however, is a degeneration of the parenchyma of the nerves.

McCarty (17) states that the peripheral neuritis occuring during pregnancy may develop before or after labor, and may be local, involving a single nerve, or multiple, involving a number of nerves. It varies in severity from a simple disturbance of sensibility to complete anesthesia, paralysis and muscular atrophy. The less marked degree of involvement characterizes the cases occurring during the early months of pregnancy, whereas the more extensive processes may occur at any stage of gestation or of the puerperium. These symptoms may or may not disappear after the interruption of pregnancy.

CASE REPORTS

Within the past few years there have been several cases of peripheral neuritis complicating pregnancy under medical observation at the University of Nebraska Hospital in Omaha. Four cases will be described below:

CASE I: Mrs. G.H. #7535. The patient was a para I, aged 22, and white. Her last menstrual period was the last of September, 1921. During the month of October she was moderately nauseated and vomited occasionally.

The patient entered the University of Nebraska Hospital, 1/1/22, complaining of nausea and severe vomiting since November 1, 1921, headache, and a loss of weight from 210# to 160#. The past history was negative. The physical examination shows evident loss of weight, the sclera of the eyes jaundiced and injected, heart rate of 100/min., pulse weak and thready, and marked tenderness over the liver. B.P. 140/84. Urine examination and blood wasserman, negative. The blood examination was essentially negative.

The patient's condition became progressively worse until 1/14/22, two weeks after admission, when she complained of ringing in the ears. B.P. 160/80. Pulse 150/min. At this

time a vaginal hysterotomy was done and a three months old fetus delivered. Several days following the opertation, the patient was very irrational showing varying degrees of delirium. Three weeks after admission, 1/20/22, she developed a feeling of weight in the legs. Several days later, examination revealed a lateral nystagmus, fine tremor of the tongue, retraction of uvula in the midline, husky phonation, knee jerk and Achille's tendon reflex absent, slight tactile loss in feet and lower part of legs, and hyperalgia of lower extremities. The patient's memory became progressively poor, and on 1/29/22 she had a sudden attack of irregular respiration and tachycardia (160/min.). The following day she had a similar attack. The patient's condition became worse and on 2/1/22, one month after admission to the hospital, she expired with a repiratory paralysis.

The autopsy demonstrates a diaphragmatic paralysis, subinvolution of the uterus, congestion and edema of the lungs, and congestion of the central nervous system. Microscopic section and examination of the brain, cord, and peripheral nerves was unfortunately not accomplished.

Diagnosis: Toxic encephalitis and multiple neuritis in pregnancy.

CASE II: Mrs. D.M. #35883. The patient was a primipara aged 22, and white. Her menstrual periods had always been irregular;

and there was an interval of amenorrhea lasting nine weeks before the onset of her last period on March 26, 1931. Beginning May 10, 1931, she became quite nauseated and vomited considerably. This continued until June 7. At this time she had tachycardia, a moderate rise in blood pressure, and an albuminuria.

She was admitted to the University of Nebraska Hospital on August 8, 1931. Two weeks previous to entrance she had shown progressive mental dullness and loss of memory. One week previous to entrance she noted blurring of vision and spots before the eyes. Two days previous to entrance, choreiform movements began in the hands. The urine has been scanty, measuring only several ounces daily, with the patient being continually nauseated and vomiting occasionally. There was loss of weight from 145# to 106#. She also noticed occaisional numbness of the hands and feet. Her past history elicits only influenza in 1918.

Examination of the patient shows a tachycardia (120 to 150 per min.), normal temperature and rapid shallow respirations. B.P. 150/90. The pupils were dilated and nystagmus was present in all planes. Ataxia was present in both upper extremities with intention type of tremor, nerve tenderness, absent reflexes, motor weakness, and wrist drop. There was a distinct systolic murmur heard over the precordium. The fundus

of the uterus was found to be at the level of the umbilicus. Fetal movements and heart sounds were not discernable. Marked bilateral paresis and tenderness were elicited in the lower extremities with absent reflexes, ataxia, and foot-drop. Mental torpor and general choreiform movements were present. A lumbar puncture was done, the protein content being 7 mg. %. A spinal wasserman was negative, and a colloidal gold curve not remarkable. The blood and urine examination at this time were essentially negative.

The patient's condition became progressively worse with increasing difficulty in respiration and swallowing, loss of phonation, and frequent involuntary stools. Early on the fifth day after admission to the hospital, a caesarean section and sterilization were done under local anesthesia. Post-operatively there was no relief of the respiratory embarrassment, and respirations ceased on the fourth day.

Autopsy demonstrated a fibrinous pericarditis and posterior congestion of both lungs. I regret that a microscopic neurological examination was not accomplished.

Diagnosis: Polyneuritis gravidarum following toxemia of pregnancy.

CASE III: Mrs. E.S. #38450. The patient was a woman, aged 26,

and black. Her last menstrual period was October 10, 1931. She was a gravida 6 with four normal labors and one miscarriage. During the first half of December, she suffered severely from nausea and vomiting which continued until just before hospital admission. About the first of February, she had a severe pain in the abdomen while straining at the stool which caused her to fall to the floor. At this time she passed several blood clots. Bleeding continued until hospital admission. Irregular pains in the lower abdomen continued, especially when ever she moved about. She had noticed no additional pigmentation, no tingling, no dysuria, no frequency, no burning, no edema.

On March 18. 1932, the patient had been sick in bed for nearly six weeks and complained of pain in the abdomen, dizziness and spots before the eyes, nausea, vomiting, and some weakness.

The patient was admitted to the hospital on March 29, 1932. Examination revealed the fundus of the uterus to be three fingers above the umbilicus, head floating, and the fetal heart sounds in the lower right quadrant. The pelvic measurements were as follows: interspinous, 24 cm.; intercrestal, 29 cm.; bitrochanteric, 31 cm.; external conjugate, 21 cm.; right oblique, 22 cm.; left oblique, 22 cm. The temperature was 99.4, the pulse 135/min., and the B.P. 115/95. On April 1, 1932, the urine ex-

amination showed acid, sp. gr. 1.023, albumin 1 plus (75 gm. per liter), sugar negative, diacetic negative, acetone negative, and blood negative. The microscopic showed pus cells, epithelial cells, occassional granular casts, and a few crystals. The blood count showed the red blood cells to be 4,390,000, the white cells 10,420, polymorphonuclear leucocytes 66%, and the lymphocytes 34%. The blood chemistry showed sugar 101 mg. %, the N.P.N. 39.8 mg. %, the blood serum cholesterol 216 mg. %, the CO2 43.8 vol. %, and the cholrides as NaCl 444 mg. %. The spinal fluid pressure was 16 mg. Hg., the protein content 10 mgm., and the spinal fluid wasserman negative in all dilutions.

The patients condition became gradually worse, and on April 2 a neurological examination demonstrated general weakness, weakness of the extremities, weakness of the intercostal muscles, general flabbiness of the muscles of the extremities, absent reflexes, pain on deep pressure over muscles of the extremities, cloudy mentality, cloudy cornea, lateral nystagmus, pin-point pupils, blurring vision, past pointing, and speech defect.

The following day the patient could raise the knee but could not extend the leg after it was flexed. She showed increasing lethargy and mental deterioration, and sluggish sensory response to pin prick.

On April 4, the patient was very restless. The urine

was loaded with casts. Respirations were shallow and rapid and the heart rate 140/min. Respirations ceased at 1:30 P.M.

Autopsy, April 4, 1932, at 4 P.M. demonstrated a four month old fetus within the enlarged uterus. Gross examination revealed fatty degeneration of the liver and congestion of the kidney. Microscopic neurological sections have not as yet been finished.

Diagnosis: Polyneuritis Gravidarum.

CASE IV: Mrs. N. The patient was a colred woman, aged 33, a para 5 and a gravida 6. Her last menstrual period began on March 15, 1931. The fifth baby was delivered after a long labor one year previous. The history of the other four pregnancies and labor is essentially negative. Her menstrual periods showed no abnormalities, occurring every 28 days, and lasting four to five days. The prenatal history shows a negative wasserman, no vomiting, no headaches, no visual disturbances, no hemorrhage, and only a moderate constipation. The last two months of her pregnant period, the blood pressure was moderately high, ranging between 140/80 and 150/90. Urine examinations were negative and the body temperature within normal range. During the last month she

noticed a slight edema of the ankles which continued until after labor. On December 8, 1931, she first complained of a slight numbness and pain in the left arm.

The patient was admitted to the University Hospital on January 9, 1932. At that time she still had some edema of the ankles and complained of pain in her left arm. Physical examination, blood and urine examination were not remarkable. The day after admission she delivered a normal live baby girl. The day after delivery she complained mainly of pain in the left arm which was tender on pressure over the muscles. The post partum recovery was not remarkable, and she was dismissed January 21, 1932. One week after leaving the hospital, the pain in her left arm had entirely diminished and examination revealed nothing abnormal in the recently affected extremity.

THE HISTORY, ETIOLOGY, AND CLASSIFICATION.

For a long time it has been known that women during pregnancy and the puerperium are more or less often subjects of various paralytic phenomena. Nicholson (20) relates that many years ago Churchill published a paper in which there were collected a considerable number of cases of nerve lesions associated with some period of life - usually the child bearing process. As the majority of these appear to have been cases of uremia and as no definite facts as to presentation or pelvic measurements are included, their value is nil, except for the fact that the author states his disbelief in the influence of difficult labors or forceps extraction as causitive factors, and in advance of his time attributes them to autoinfection. On the other hand, Basedow, in 1838, asserted his belief in the efficacy of head pressure upon the pelvic nerves, as a cause, in certain cases which he had seen. His paper leaves but little to be desired so far as his description of his cases are concerned.

Coming to the period beginning about the year 1870, we find that observers are not agreed as to the cause of the condition. Bristowe, quoted by Sinkler (24), as recently as 1879, in his Practice of Medicine, remarks, in connection with the path-

ology of lead poisoning: "But as regards the paralysis of the voluntary muscles, there is no doubt that Ducheane is right in regarding it as a consequence of nervous disorders. For if it were muscular not only should we find the muscular fibers degenerated in proportion to their loss of function, but we should find faradic contractility surviving as long as any healthy muscle was left. On the other hand the rapid shrinking of the muscles without degeneration and their speedy loss of faradic contractility, obviously point to lesions either of the nerve trunk or of their nuclei of origin." As soon as it was pointed out that there was such a disease as an "inflammation" of the peripheral nerves, the knowledge of the subject quite rapidly advanced and the various forms of polyneuritis became well known.

We find, at this time, that one author will attempt to explain all cases on the ground of autoinfection when perchance he has only seen cases of general or diffuse neuritis, while another's experience will have been limited to local lesions in the arm or leg, and he will claim for pregnancy such influence as is shown by lead or the toxins of diphtheria in the production of the local paralysis. Still another with a large experience in the use of forceps in difficult cases of labor, will attribute the laming of the limbs to nerve-pressure. While all these reports are founded

on good and sufficient grounds, as a careful study of the reported cases will show, no one of them alone will explain , as has been attempted, all or nearly all of the cases encountered.

In considering the etiology, we find that the classification which various authors have made use of is of considerable interst; thus Ross and Bury, quoted by Nicholson (20), divide all cases into two general classes: 1). Those due to injury or disease of the pelvic nerves either as a result of trauma or inflammation. 2). Those due to multiple neuritis. In 1887, Moebius (19) called attention to the toxic origin of the disease and described a puerperal polyneuritis involving the median and ulnar nerves. He mentions that all the cases that he encountered in which the legs were involved were due to inflammatory pelvic changes. He also expressed the belief that the enlarged uterus or the forceps might cause direct injury to the ischiadic nerve. A little later Tuillant, quoted by Chlopicki and Stepowski (4) and Nicholson (20) divides all cases into two classes: 1). Those general in type due to autoinfection. 2). Those showing local lesions which he supposed to be due to a poison from without.

It is of interest to know that in the cases of general involvement reported by this author there was a very frequent association of marked vomiting in pregnancy together with muscle weakness, and he considered that both nerve and vomiting symptoms were the result of the same cause.

THE HISTORY, ETIOLOGY, AND CLASSIFICATION.

In 1891, Gowers, quoted by Handford (10), suggests that the "tissue health is lowered", and consequently slighter causes are sufficient to excite neuritis.

Hunermann, quoted by Nicholson (20), in 1892 assembles all cases under one or the other following heads: 1). The influence of pregnancy upon an unstable nervous organization. 2). Pelvic exudates. 3). Severe puerperal infections which may play the same part in the production of paralysis as do diphtheria and the scarlet fever. 4). Those cases due to the pressure of the fetal head or the traumatism of the forceps. Regarding this fourth division, he says that the head of the child is much more often operative than are the forceps, particularly if the presenting part be well flexed and attempting to enter a pelvis contracted in all of its diameters. He also emphasizes that the duration of labor, the degree of disproportion, and the strength of the pains are factors determining the gravity of nerve injury.

In 1893, Mills (18), approaching the subject of traumatism during labor from the standpoint of the neurologist, classifies unders the heads: 1). Traumatic paralysis of the peroneal type usually associated with severe neuritis. 2). Sacral neuritis aggravated by disease or displacement of the pelvic organs or tissues. 3). Septic or other infections which may cause either local or

multiple forms. 4). A special class, peculiar to this author, due to phlebitis of the pelvic veins and therefore to be classed as septic. 5). Myelitis due to some infection.

Windscheit, quoted by Thomas (27), and Nicholson (20), arranges these cases under the following heads: 1). Neuritis gravidarm (etiology not known, probablly toxic). 2). Infections (general pyemia may cause neuritis of all nerves of the body). 3). Mechanical injury. 4). Puerperal (localized or general).

Weber (28) in 1898 infers that it is now recognized that pregnancy, without any distinct septic change, may be a cause of peripheral neuritis.

In 1900, Thomas (27), attempts the explanation of neuritis due to traumatism of the pelvic nerves as follows: The upper roots of the sacral nerves do not lie upon the pyriform muscle, but against the bony wall of the pelvis, and are thus exposed to injury from pressure during certain difficult labors. It is the doral offsets of these roots which lie against the bone and which receive the chief injury. The external popliteal nerve is made up from these doral offsets, and therefore the paralysis is chiefly localized in the distribution of this nerve.

The superior gluteal nerve supplying the gluteus medius

and minimus muscles and the inferior gluteal nerve supplying the gluteus maximus also received their nerve fibers from the dorsal division of the roots of the plexus, and it is interesting to note that these muscles are not infrequently paralyzed in obstetrical paralysis.

Thomas continues in saying that it seems clear to him that these cases are due to trauma of the nerve roots during labor, although the view had not been universally accepted. Lloyd (15) considers the question fully and concludes as follows: "From the facts and authorities it is evident that there is some latitude for the differences of opinion as to the exact causation of lesions of the sacral plexus and its chief trunks during labor. The old writers evidently disposed to regard pressure by the head and injuries by the forceps in prolonged and difficult labor as important factors in causing these paralyses. The tendency of more modern writers is to dissent from this view, and to ascribe lesions of the sacral plexus and its branches to a septic inflammation, propagated directly to the nerve trunks from a metritis or a periuterine cellulitis. I do not see that it is necessary to ignore either one or other of these important factors, although I believe that the theory of septic infection is one that more satisfactorily explains the majority of these cases. There is little

doubt, however, that in case of a large head or a contracted pelvis, the instuments especially if applied in a faulty manner might make pressure upon the sacral plexus where it lies upon the body of the pyriform muscle, or especially upon the trunk of the sciatic muscle, and where by reason of its great size and its exposed condition, it is liable to injury. "

Eulenberg, quoted by Nicholson (20), considers two groups, basing the distinction between them simply on the extent and severity of the lesions presented, and so divides them into 1). The less severe and localized forms, while in 2). he groups the diffuse forms, which may simulate at times the Landry's type of paralysis, or may even be found involving the cerebral nerve areas. It may here by mentioned that Scottas, Leitz, and Eulenberg have all reported cases simulating the paralysis of Landry.

Nicholson (20), in 1904, writes that his preference in so far as the etiologic classification is concerned, would be as follows: 1). Those cases supposedly due to some toxemia. 2). Those cases arising from a septic process. 3). Those case resulting from some mechanical agent operative during labor or in the early puerperium period. He enlarges upon this in saying, "These three classes include all cases which can in any way depend upon pregnancy as the causitive factor. It should be remembered that myelitis or

other disease of the nervous system may show the first signs during pregnancy, and that in such cases the influence of pregnancy is not to be considered as causitive except as any other marked strain. With regard of the first division, namely, the toxic group, we know but little. Quite a number of cases have been reported which are difficult to explain under any other supposition. Of course, these are most usual in pregnancy, though rarely are they also encountered in the puerperium. As regards their true causation, we know practically nothing: they may either be general or limited to special nerves. ------- Until we become more familiar with the true nature of autointoxication we will, I think, be unable to explain these cases. The septic division (the second) is decidely easier to explain. It is perfectly justifiable in the light of our knowledge of septic processes to consider that either by continuity or contiguity there may be involvement of the trunks of the pelvic nerves, as in the case of exudates, or abcess, or that by transference through the blood or lymph channels, any group of nerves may be involved. Furthermore, we know that the gravity of these cases depends upon the individual susceptibility and the virulence of the poison, these factors usually determining a simple transity neuritis, and ascending myelitis, or any of the lesions intermediate between them.

"Of course, from the nature of things, sepsis is more

often operative during the puerperium than in pregnancy, and so we are less liable to meet these cases during pregnancy. We now come to the consideration of the last division, according to the above classification; this is probably by far the most frequent variety. ---- A considerable number of authors have considered that instrumental interference is the most prolific cause of this group, but investigation shows that such is not the case, but rather that the delay in the advancement of the presenting part, which occasions the employment of instruments is of itself the reason for the palsy. In order to understand the true etiology of this class, mechanical, we have only to turn to the anatomy of the sacral plexus. The nerve-supply to the lower leg is derived from the terminal branches of the great sciatic nerve, and the latter is derived from the lumbo-sacral plexus and the first, second, and third sacral nerves. The external popliteal, or peroneal, nerve takes its origin from a certain special group of filaments which are derived chiefly from the dorsal portion of the lumbo-sacral plexus, while the segments which form the internal popliteal arise from the ventral portion of the same nerve. From the arrangement of the nerve fibers with reference to the bones and muscles of the pelvis, it will be seen, on reference to fresh dissection, that the lumbo-sacral plexus is the only portion of the plexus which is exposed to direct violence between

any passing body, the remainder of the plexus being guarded by muscle fenders. While the lumbo-sacral plexus traverses the pelvic brim near the base of the promontory, i.e., in the sacral-iliac notch, it may nevertheless be easily exposed to pressure under certain conditions. Finally, the filaments to the peroneal nerve take their origin from the dorsal segment of the cord, and they are therfore in a position which will subject them to increased chances of pressure, lying, as they do, directly on the bony ridge known as the iliopectineal line.

"These anatomical facts will explain the most usual form of neuritis associated with pregnancy, namely, that involvement of the peroneal nerve on one side and resulting in a motor and to a less degree sensory paresis of the muscles of the anterior portion of the lower leg and dorsum of the foot. Now as the factors which occasion the pressure in the majority of instances: A glance at any pelvis will show that the forceps can not be held as the responsible agent, since the situation of the lumbo-sacral plexus can not be injured by their blades in any proper application; we question, indeed, whether the injury could occur to this nerve by any, even an improper, instrumental operation, as its position guards it so well from assult in any direction except one, namely, by a force operating directly on its anterior surface. Moreover, symptoms of nerve injury occur after labors that are easy and rapid, and in which no forceps have been used. This is

difficult to explain, but it certainly shuts out the necessity of instrumental causation; on the other hand, it is unquestioned that such injuries are more likely to occur in labors that are difficult, especially if such difficulty be due to a small pelvis, of the generally equally contracted variety. The simple flat and rachitic flat pelves do not show the same tendency to produce this condition, since in these types of deformity the projecting promontory serves as a protection to the lumbo-sacral plexus. In addition, it should be remembered that it is not the extreme grades of contraction which give rise to this lesion, since for the head to impinge upon the lumbo-sacral plexus it is necessary for it to be able at least partially to engage. Those generally contracted pelves with a true conjugate of from 8.5 cm. to 10 cm. are the forms of deformity which give rise to the greater number of cases of palsy of one or both limbs. It is possible that in severe cases there may be an ascending inflammation which will be transferred through the cauda equina or the cord itself to the nerves of the opposite leg, and give rise to a more or less paraplegia. It is even claimed by some that there may be a true degeneration of the cord secondary to peripheral neuritis. This then is the principle cause which explains a neuritis in the puerperium with consequent palsy of one leg, namely, a combination of the medium grade of contracted pelvis, usually of the generally

contracted variety, together with a presenting part which either with difficulty passes or is unable to be delivered with out instrumental aid. As an additional factor may be mentioned the strength of the explosive pains since the greater the "vis a tergo" the more pressure will be developed by the presenting part upon the nerves. It may be mentioned that the after-coming head has been known to produce the same injury as has been ascribed to the head-first presentations.

"Aside from the influence of direct fetal pressure upon the nerves there are other causes which have been considered in certain cases; These are a decidedly enlarged uterus (this we doubt), a perimetritis, a previous severe laceration of the supravaginal tissues, with resulting scar formation, any pelvic inflammatory collection, and, finally, a phlebitis of the pelvic veins."

The present day conception of the etiology of peripheral neuritis in pregnancy has not changed considerably from the views presented by Nicholson in 1904 as cited above. The modern writers, however, seemed more concerned with the toxic and ideopathic etiological phases of the subject. McCarty (17) in 1916 writes that neuritis in general may be of either central or peripheral origin and it is the latter type which is commonly associated with the pregnant state. The causes of such a peripheral neuritis are many and they vary with the time of onset of the condition. Cases

which develop after delivery are most often due either to trauma from pressure of the fetal head, the branches of the sacral plexus, being involved, to pressure in the popliteal space from prolonged acute flexion of the knees or of the use of leg holders, or marked puerperal cachexia.

During the course of gestation and before delivery the frequent association of neuritis with hyperemesis gravidarum or some of the various other manifestations of toxemia of pregnancy points to the probable causal effect of toxins circulating in the body. Instances have been reported where such conditions have cleared up immediately after delivery or after the termination of pregnancy.

Syphilis, profound anemia, general debility, exposure to cold, chronic alcoholism and local pressure or irritation may also be factors.

Frequently none of these conditons are present, and such cases constitute the group of so-called ideopathic origin. These are supposed to be due to a disturbance of metabolism resulting in the absorption and circulation of toxic substances through out the body. Intestinal stasis may well be considered in many cases, although direct evidence of the presence of such a condition is lacking in cases which have been reported. The occurrence of such a process is suggested by the frequent finding

of indican in the urine. In a case observed by McCarty the presence of viceral ptosis with a moderate degree of stasis have been previously determined by laparotomy and the degree of stasis had been markedly increased prior to the onset of neuritis.

McCarty also suggests that the cases of ideopathic origin resemble greatly the neuritis and paralysis caused by reflex irritation, to which attention was called by Brown-Sequard who quoted instances of genito-urinary disease especially, which were accompanied by paralysis in various regions of the body. Similarly, uterine disease, such as cervical erosions or uterine displacements have been complicated by paralysis which have disappeared after correction of the pelvic disease. That such cases are found in most instances to be associated with genito-urinary disease in either sex would seem to imply that the explanation may be found in the rich supply of those parts of nerve fibers from the sympathetic system. In addition to true neuritis there may occur various sorts of neuralgia and paresthesia associated particularly with the onset of pregnancy. Most of these disturbances are without real foundation and are attributed to the mental state of the individual. Sometimes, however, true neuritis does occur at that time.

Acosta-Sisson (1), in 1928, writes an excellent paper reviewing neuritis in Filipino parturients. He collected the

histories of 327 cases of numbness occuring in pregnancy or the puerperium and presents the following report as to the possible etiology of the lesions.

Seven showed heart affection as follows:

Hypertrophy and dilitation ----------2

Hypertrophy and dilitation and kidney insufficiency --1

Mitral insufficiency ----------4

The following is the list of diseases or conditions associated with, if not causitive of the neuritic symptoms:

Pernicious vomiting of pregnancy of the toxemic type-3

Dysentery ----------5

Typhoid fever ----------2

Post-partum hemorrhage ----------3

Puerperal infection ----------15

Toxemia of pregnancy -- with herpes labialis -------13

Eclampsia ----------3

Vomiting due to toxemia ----------1

Pulmonary Tb. ----------15

Gonorrhoea ----------3

Syphilis ----------4

Malaria ----------1

Albuminuria ----------50

Liver abcess ----------1

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Impetigo ----------1
Twins ----------5
Bronchial asthma ----------4
Nephritis ----------3
Hydatidiform mole and hemorrhage ----------1
Beriberi ----------2
Grief ----------1

It is interesting to note that eight of the cases that had muscular atrophy and inability to walk for three or four months had one of the following complications:

Dysentery
Typhoid fever
Post-partum hemorrhage
Puerperal infection
Pernicious vomiting of pregnancy of the toxemic type

A severe case of pseudo-paresis who had a premature delivery gave the history of profound depression caused by the abandonment of her husband who had left her as soon as he knew that she was pregnant.

Blood count was not made in all the cases; a large portion of them had palid complexion showing anemia, and many of them had also caries of the teeth.

No difference was found in the diet of those who had neuritic symptoms and those who did not present neuritc symptoms. It consisted mainly of semipolished rice, fish, and vegtables. Meat was consumed in limited quantity by some while others partook of it freely. Milk and eggs, however, were taken sparingly.

"The above data would seem to indicate that not all of the parturient cases presenting neuritic symptoms are beriberic in origin, but that any condition or disease that weakens the general resistence of the mother, or any invasion of the maternal organisms by toxic agents may give rise to neuritc lesions.

"Is it not likely that these lesions are, in the great majority of cases, manifestations of the toxemias of pregnancy, just as herpes gestationis or asthmatic attacks, that occur only during pregnancy, are toxic in character? One characteristic of these neuritic symptoms is their tendency to recur in succeeding pregnancies or puerperia. On the other hand, recent observations on beriberi in the medical wards of Philippine General Hospital suggest s that complete cure from one attack confers immunity to subsequent manifestations of the disease.

"Pressure on the nerves has no negligible influence on the development of neuritis, as shown by the frequent occurrence of the symptoms in the lower extermities during the last two months of pregnancy and, in cases of twins and hydramnios

where the pelvic nerves are subjected to much pressure, difficult and prolonged labors, whether instrumental or spontaneous, often cause neuritis in one or both extremities.

"While the existence of beriberi among mothers and infants is not to be denied, yet I believe that to say that all the neuritic symptoms among the parturients are of beriberic origin is far from true."

Tashjian (26) in 1929 suggests that all though it is well known that polyneuritis occurs in cases of vitamin B deficiency in countries where the main diet is polished rice, general practioners seldom take more than an academic interest in it, and for get that it may develop in our best hospitals.

Fink (8) points out that etiolgically we must differentiate between a neuritis resulting from an exogenous course, i.e., a neuritis due to the trauma associated with labor, and a manifestation of a neuritis resulting from some process due to pregnancy, labor or puerperium, the etiology of the process being in most of these cases unknown. "If we consider the fact that the known pregnancy toxicosis such as eclampsia may occur in the rudimentary form which ## have but little in common with the cerebral symptoms characteristic of the severe forms, such as symptoms in pre-eclamptic stages, or the symptoms resulting from a slight increase in pressure and hemianopsia or chromatopsia,

then we could concieve that the gestation process may also lead to neuritides which would have nothing in common with the neuritides resulting from trauma due to labor, puerperal infections, or an extension of the exudate through the nerve sheaths. We can also say that toxic neuritides may occur in various forms, i.e., may be very pronounced or only rudimentary and may manifest either in form paralyses of the muscular regions or individual muscles which are quite distant from the genital sphere. The theory of toxic neuritides is supported by the fact that these toxic neuritides may occur and disappear at any phase of the pregnancy, i.e., may be fully independent from infection, trauma or local inflammatory changes."

Chlopicki and Stepowski (4) point out that the unknown toxin probably derives from the fetus or the placenta but that the possibility exists that it originates in the liver as urobilin is usually present in the urine and because neuritis observed sometimes in patients with infectious jaundice is similar to the polyneuritis of pregnancy. Various vascularization of the nerves may be the cause of one nerve being affected and another not. Biochemical factors also may be one of the reasons why the vulnerability of the nerves varies considerably.

PATHOLOGY

When a peripheral nerve is injured or diseased the fibers distal to the point of injury soon undergo definite pathological changes. The noxious agent may be of the nature of trauma, inflammation, or toxic degeneration; but if the effect is sufficiently severe the result is an interruption in the structural and functional continuity of the nerve. The clinical symptoms are the direct expression of this structural and functional impairment. It matters a great deal, of course, whether it is partly or severely traumatized or entirely severed, or whether the inflammatory process, for instance, is mild or severe. But while the nature and extent of the pathological process influence, in a great measure, the course of the disease, it is essentially the structure and location of the particular nerves involved which are responsible for the clinical picture of peripheral neuritis.

In reviewing the pathological anatomy presented in the various cases reported in the liturature we find that Korsakoff and Serbski, quoted by McCarty (17), reported a case of the multiple variety in which there was found parenchymatous neuritis of the nerves to the extremities, with changes in the lumbar and sacral plexuses and in some of the cranial nerves. There was also an increase in neuroglia in the columns of Gall

and in the lateral columns of the cord.

The fact that the action of the toxins is not limited to the peripheral nerves is supported by the report of Stewart (25) who presented a case whose symptoms began in the seventh month of pregnancy following hyperemesis gravidarum and continued until the fourth month after delivery when death insued from paralysis of the diaphragm. He found, on complete systemic microscopic examination of the peripheral and sensory nervous systems, that in addition to the classical changes in the vagus and phrenic nerves and the nerves of the extremities, there were marked degenerative changes in the posterior and lateral tracts of the spinal cord, and also in the cells of the anterior horns.

Lindermann, quoted by Acosta-Sisson (1), in a fatal case showed that the lesion in peripheral neuritis were associated with degenerative changes in the liver and kidneys.

The histological examination of the nervous structures of a fatal case reported by Hornumg and Creutzfeld (12) did not reveal infiltrative changes in the spinal cord and peripheral nerves. The medullary fibers of the peripheral nerves, however, showed marked degeneration and swelling. Fatty granular cells involve the markedly granular degenerated cells with very little impregnation of the nerve fibers with silver. There were found in

all the preparations fine fibers, either in bundles or singly, running along with the degenerative fibers. Frequently they terminated in knob-like endings, some times it appeared as though they branched from the larger fibers. The sheath of Schwann was well preserved. Thus Hornung thought that regeneration was in progress. In many of the anterior horn cells of the spinal cord, the only changes found were a swelling, a paler central area of cell plasm, with displacement of the nucleus, the Nissl's bodies, the pigment and the fibrills to the periphery of the cell. It was a characteristic picture of primary irritation, as described by Nissl following separation of the nerve fibers from their motor cells. The changes in this case were evidently due to a degenerative process of the peripheral nerve fibers. The entire picture presents nerve changes which can be said were due to severe intoxication.

The term neuritis for the condition we are describing is some what misleading, though sanctioned by long usage. For it is in reality in nearly every case a degenerative rather than an inflammatory lesion that is responsible for the symptoms. The inflammatory changes are present, it is true, in some forms of multiple neuritis but they are an inconspicious feature of the histological picture, Feiling (7) points out that a toxic degeneration is the most essential cause of all forms of so-called multiple neuritis. That this degeneration affects principally the parenchyma of the nerve trunks and the actual microscopic changes found

are very similar to those occurring in the distal part of a divided nerve, the Wallerian degeneration of physiology. Feiling continues stating that to the naked eye there is little or no apparent change; in a few cases, however, the nerve may appear thickened; this thickening being due to serous effusion which takes place into the sheath of the nerves.

Wechsler (29) offers a very concret description of the anatomy and patholgy involved in the peripheral neuritides as follows: "Every nerve-fiber contains an axis cylinder or axon which is the largest protoplasmic and functional continuation of the nerve cell. The axis cylinder, except in the case of the sympathetic and olfactory nerves, is insulated, as it were, by a myelin sheath. The sheath of Schwann with its many nuclei forms the external limiting membrane of the nerve-fiber. The individual fibers are held together in small bundles or fasciculi by a connective tissue endoneurium. Several of these bundles are united into larger ones by a perineurium and the whole peripheral nerve is surrounded by epineurium. The lymph spaces, which are continuous with the subarachnoid space of the brain and spinal cord, run in the endo- and perineurium. When a nerve-fiber undergoes pathologic changes of sufficient severity -- this almost irrespective of the cause -- the myelin sheath breaks up into lecithin bodies and fat droplets, the axis cylinder splits and becomes fragmented, the

cells of the sheath of Schwann proliferate, the "debris" is absorbed, and a connective tissue tract, as it were, takes the place of the nerve fiber. Often this process is retrograde in the sense that the part of the nerve proximal to the point of injury undergoes similar degeneration. In some instances the degeneration up the motor fiber to the anterior horn or up the sensory fiber to the posterior ganglion."

MacCallum (16) writes as follows: "It has been pointed out that lipoid bodies form the coatings of nerve fibers, as though to insulate the axis-cylinders within these myeline sheaths. When the nerve dies through being cut through or from degeneration of its cell-body, the lipoids of the myeline about the dead axis-cylinder disintegrate, leaving globules of the decomposition products which now blacken with osmic acid in a way foreign to the myeline itself. Saponine attacks and combines with the lipoid sheath of the nerve and causes paralysis. Many other substances, most of which have certain affinities for lipoid materials, cause injuries to nerves, followed by inflammation or by paralysis. Lead palsy, arsenical and alcoholic neuritis, the neuritis occurring in the intoxication associated with pregnancy and diabetes, are examples of this vague connection."

SYMPTOMS

Because the peripheral nerves are both sensory and motor, subjective and objective sensory disturbances and motor weakness or paralysis occur practically in every case of neuritis. Wechsler (29) in his Textbook of Clinical Neurology describes in general the peripheral neuritides as follows: "Pain, is the most frequent and often the most severe manifestation of peripheral neuritis of what ever nature. The pain may vary in intensity and be sharp, boring or burning in character. It is always in the course and distribution of the affected nerve or nerves. It is apt to be very severe in mild, irritative lesions; absent after a nerve is completely separated. Only rarely is it negligible. Disagreeable sensations -- paresthesia and dysesthesia -- may procede, accompany, follow, or replace the pain. Movement often intensifies it. Frequently there is tenderness along the course of the nerves.

Practically every type of peripheral neuritis (except in the case of pure motor nerves) is accompanied by objective sensory disturbances. The characteristic of peripheral sensory impairment is the involvement of all forms of sensation, vis., touch, pain, temperature, position, and vibration. (It is worth noting

that the fibers carrying deep sensation run in the motor nerves -- Head.) The intensity of impairment may range from slight hypesthesia to complete loss of sensation. One form of sensation (delicate touch, point discrimination) may possibly be more affected than the others; or there may be hyperirritability (hyperesthesia and hyperalgesia) in certain regions; but all the objective sensory disturbances are more or less within the cutaneous distribution of the affected nerves.

"The third very important sign of almost every type of peripheral neuritis is motor impairment often in excess of sensory disturbances. The motor impairment ranges from mild weakness to complete paralysis. The muscles feel flabby because of loss of tone peripheral or lower motor neuron paralysis is always flacid in nature. It is accompanied or followed by atrophy in the muscular distribution of the affected nerves, by loss of deep reflexes (interruption of the lower reflex arch) and by qualitative electrical changes up to R. D. Fibrillations, on the other hand, except in very few instances (not infrequently in the tongue), are generally absent in peripheral nerve lesions. Cramps and muscle spasms occur only occaisionally. An atrophic muscle may show heightened local irritability.

"Among the other fairly constant changes may be mentioned trophic and vaso motor disturbances. These are due to impairment

of sympathetic and trophic fibers which run in the peripheral nerves. There is sufficient evidence to show that the cells in the posterior sensory ganglion have trophic functions. The skin is often thin and glossy, sometimes wrinkled, bluish, and cold. Keratitis, desquamation, furrowed, curved or brittle nails, hypertrichosis, hyperidrosis, or loss of sweat may occur. These trophic changes are often observed in mild, irritative, or recovering cases as well as in those with complete paralysis. Occaisionally, when there is marked interstitial inflammation, the nerves may be swollen sufficiently to be palpable. Slight fever is only rarely present in the early stages of peripheral neuritis. In many cases of chronic neuritis muscular contractures frequently occur. These contractures may be due to over action of the normal muscles which are unopposed by their paralyzed antagonists, or to actual fibrosis of the muscles. Secondary joint changes, namely, effusion and ankylosis, may occur."

Nicholson (20) writes that in regard to the actual symptoms, it will be found that they vary from a transient pain often ascribed to rheumatism, etc., and with out localizing nerve signs to a symptom-complex resembling Landry's type of paralysis or myelitis. The usual type of case is one intermediate between these extremities, presenting some pain, loss of power, perhaps

some sensory disturbances as paresthesia and atrophic changes. The nerves involved are in less severe cases the median or ulnar, or if the leg be affected, the sciatic, the gluteal, and especially the external popliteal, the particular symptom peculiar to the latter being the presence of the so-called foot-drop.

Local or multiple neuritis may occur before or after labor, and according to the time of labor, are classified by McCarty (17) as conceptional, gestational, or puerperal and local or multiple.

Conceptional Neuritis

The patients frequently complain of vague sensory disturbances such as numbness, tingling, or even acute pain at or about the time of conception, and it is a popular notion that such conditions are to be expected at that time. How many of these changes are due to the real disease and how many to the mental attitude of the patient is impossible to determine, but the latter is unquestionably the essential factor in most cases. A state of expectancy or anticipation places the mind in a conceptive mood and the individual becomes susceptible to misinterpretations of ordinary sensations. Paresthesia or neuralgia is certainly much more frequent at this time than is true neuritis or paralysis, but the latter is occasionally observed.

SYMPTOMS

Gestational Neuritis

Multiple neuritis is more common than a local process during pregnancy, and it may occur either in the early or later months, due usually to pressure ofthe descending head on the fibers of the sacral plexus. When not due to pressure there is frequently evidence of toxemia or renal disease. It has long been stated that the cranial nerves are rarely affected before delivery and that such cases as are ocassionally seen may be of central rather than of peripheral origin.

Mild cases show paresthesia or hyperesthesia with no paralysis, and recovery occurs before delivery. More severe types occur, with paralysis and anesthesia first in the legs, later perhaps in the arms also, and recovery may be delayed for some time after labor.

Mental changes together with muscular paralysis, as described by Korsakoff, from a considerable proportion of these cases.

Cline (5) points out that "the coincidence of Korsakoff's psychosis in the neuritides is of considerable clinical interest. This syndrome, of which the salient features are, impairment of the immediate memory, mild mental clouding, confusion, and fabrication was first described in 1887 by Korsakoff, who found it to be a very frequent complication or symptom of alcoholic multiple

neuritis. Since the advent of the automobile and the more extensive use of coal-gas, several cases of Korsakoff's psychoses have been reported as occuring in carbon-monoxide neuritides. Of interest is the statement of Van Hosslin, who finds that the Korsakoff's syndrome is reported as having been observed in multiple neuritides following toxemias of pregnancy long before Korsakoff placed his syndrome before the scientific world, in addition, observes that, "pro rata", the incidence of this psychosis following gestational toxemia is greater than in alcoholic neuritis.

Ely (6) describes four cases of multiple neuritis following hyperemesis gravidarum, and in each patient a memory defect of the Korsakoff type was observed.

Puerperal Neuritis

Local paralysis is much more freguent after labor, even in those cases which can not in any way be attributed to direct pressure on nerve trunks. Pressure paralysis affect most often the sciatic and peroneal nerves and occur especially in elderly primipara or after a complicated labor. Pelvic inflammation with massive exudate may also exert pressure on the nerves of the pelvis and result in neuritis. Aside from such mechanical causes a definite local neuritis occurs, involving most frequently the median and ulnar nerves. The anterior crural, obturator and occaisionally one of the cranial nerves may be affected. Koester reported a case in

which the axillary and musculocutaneous nerves were involved, and Nothnagel reported a paralysis of the deltoid, serratus and pectoralis muscles. Many of the cases reported have occurred in patients who have shown evidence of puerperal sepsis.

Local Neuritis

The onset is sudden, with or without fever and with few constitutional symptoms. The process may vary from a simple paresthesia to complete paralysis and anesthesia. Severe pain of a stabbing or boring character may be an early symptom. The area of distribution of the affected nerve becomes sensitive to touch and perhaps to changes in temperature. This is followed be decreased sensibility and impaired muscular action. The skin may become reddened and hot and the nerve itself sensitive to pressure and even some what thickened. After a variable time, usually brief, sensation may be entirely lost and paralysis become complete. This paralysis may be transient or may result in real atrophy and permanent impairment of sensibility.

Electrical reactions vary, as in ordinary neuritis, with the extent of involvement of the nerve and the nature of the lesion. The prospect of repair and recovery is indicated by a careful electical examination. In mild cases there is no change in muscle or nerve reaction, whereas in severe cases a typical reaction of

degeneration appears.

The nerves of special sense are rarely involved in peripheral neuritis not due to local irritation or pressure, but cases have been reported where ocular paralysis, hemiopia, amblyopia, and deafness have occured. Edgar states that neuritis localized in the facial nerve is extremely rare and usually accompanies profound anemia. Saenger reported one case of bilateral paralysis. These cases are to be carefully distinguished from neuritis of central origin, due to hemiplegia, anemia, thrombosis, cerbral hemorrhage or paraplegia.

Case IV presented by the author is a typical instance of a mild type of mononeuritis of ideopathic origin. The pain in her arm began in the last month of pregnancy and was quite indefinite in character. Both arms were of the same thickness and had the same power. She demonstrated no tenderness on deep pressure over the muscles and the reflexes were normal. The symptoms cleared quite promptly after delivery. We are inclined to believe that we had here to deal with a mild neuritis resulting from pregnancy.

Multiple Neuritis

Attacks of multiple neuritis appear spontaneously during pregnancy, usually in the middle or latter months, and may be accompanied by marked general reaction. The onset, with headache,

anorexia and fever, may suggest autoinfection. Tingling of the hands and legs with tenderness along the course of the nerves involved follows, and this may shortly be succeeded by sensations of numbness or anesthesia with loss of muscular power. Pain may be severe or slight even with extensive paralysis. Mild cases may develop only stiffness of the limbs and increased sensitiveness of skin surfaces, where as severe cases may go on to muscular degeneration with complete paralysis or even to death. If the condition assumes an acutely rapid ascending form, it may not be differentiated from the Landry's type of paralysis. Recovery is slow in proportion to the extent and severity of the process, and the condition often persists after delivery. Pokozdy (23) points out the possibility of a recurrence of the symptoms, and cites a case of polyneuritis during pregnancy with three relapses after termination of the pregnancy.

It is interesting to note that Case I, II, and III presented by the author were all incidents of an acute ascending polyneuritis developing about the middle of pregnancy and following hyperemesis gravidarum. These three cases had the following symptoms in common: nausea and vomiting, gradual development of weakness especially in the lower extremities, loss of knee jerk and Achille's tendon reflex, pain on deep pressure over the muscles

affected, slow response to cutaneous sensory stimulation, dullness and progressive loss of memory, nystagmus, tachycardia and irregular respiration. These three cases present a striking resemblence to each other.

DIAGNOSIS

McCarty (17) points out that in a well-marked case the diagnosis is easily made from the history of tingling or sharp pain, followed by numbness or anesthesia and paralysis of the muscles supplied by one or several nerves. Similar nerve changes due to mechanical factors, to intercurrent infection or toxemia, or to central nerve lesions should be distinguished, and in any case a focus of local irritation should be sought.

The mechanical conditions producing neuritis includes the pressure of exudate in pelvic inflammatory disease. This probably includes many of the so-called ideopathic cases associated with fever or other evidence of infection. Other mechanical conditions may include pressure of the fetal head late in pregnancy or during labor; and direct pressure of the apparatus used to maintain flexure of the legs during delivery. Two other conditions simulate neuritis: rupture of the symphysis pubis and separation of the sacral iliac synchondrosis. Both of these conditions produce great disability due to acute pain. There is no real nerve injury in either instance.

Nicholson (20) writes that the symptomatology varies according to the severity of the lesion, and that careful inquiry

will sometimes be rewarded by the history of the inception of the trouble having occurred in pregnancy. This may be true of the forms of pressure neuritis, as well as toxic and septic. In the former the mere friction of the presenting part in the latter part of pregnancy upon the nerves at the pelvic brim is at times sufficient to produce at least there transient irritation. The time of onset may then by in the latter part of pregnancy, during labor, or at any time subsequent for a period of several weeks; the cases due to intrapelvic fetal pressure occur, as a rule, promptly after labor, and generally show a considerable amount of pain during labor. Those cases due to sepsis occur later according to the special development of the case.

Syphilis, alcoholism, and local infection all produce neuritis and recovery may be delayed until the pregnancy is terminated.

It was pointed out in the discussion of a paper read by Plass (22) that the Korsakoff's syndrome as it occurs in the chronic alcoholic disturbances differs from the syndrome as it occurs in polyneuritis complicating pregnancy, in that the former is of more chronic development and that hallucinations may be produced on rubbing the eyeballs.

McCarty (17) continues in saying that the neuritis of

central origin is often associated with toxemia of pregnancy. The lesion may be of cerebral origin as in septic thrombosis, edema of the brain, cerebral hemorrhage or cerbral anemia. In occular paralysis, especially, it is well to bear in mind the probability of central origin. Spinal paraplegia is only coincident, except in conjunction with the toxemic conditions. Bilateral sacral neuritis resulting from pelvic inflammation may simulate myelitis, from which it is distinguished by the abscence of sphincter paralysis.

Wechsler (29) states that the diagnosis of multiple neuritis is generally not difficult if one bears in mind a gradual onset, bilateral, often symmetric, involvement of the extremities, the pains and tenderness of the nerves, the distal impairment of sensation, the loss of deep reflexes, the muscular atrophies, the trophic and vaso motor disturbances, and the absence of bladder and rectal involvement. Tabes is much more slowly progressive and there are usually no atrophies, the nerves are not tender, bladder disturbances are not uncommon, pupillary changes are the rule, crises are frequent, and the serology is generally positive. In poliomyelitis sensory disturbances are absent, the paralysis of the muscles is segmental and not peripheral in distribution, there is no nerve tenderness, there are signs of meningeal involvement, the paralyses are usually not symmetrical,

and the disease picture reaches its maximum in a few days. Trichiniasis shows tenderness of muscles, edema of the eyelids, preservation of deep reflexes, absence of sensory disturbances, and eosinophilia, and the possible demonstration of trichina in the muscles. Acute polymyositis is characterized by swelling and edema of the muscles, which are frequently rigid, by the absence of sensory changes, and the presence of deep reflexes, and by an erythematous rash. In osteomalacia, the weakness is more in the flexors of the hip, the muscles of the back and upper extremities. Tremor may be present, but the deep reflexes are apt to be lively and there are no sensory disturbances.

PROGNOSIS

It is a good sign that in the past few years neuritic symptoms among parturients as well as mortality among infants less than one year old have been steadily decreasing due to the better education of the masses in matters pertaining to health and to the greater facilities they have for securing skilled aid during the period of expectant motherhood and medical advice for their young infants.

Nicholson (20) infers that in a considerable number of mild cases of peripheral neuritis the patients recover without any diagnosis of the condition having been made, as the period usually spent in bed after the delivery would be amply sufficient for this in most cases. In the more severe forms the prognosis is doubtful or good, according to whether the reactions of degeneration are present or not. In any advent, considerable time frequently elapses, even under careful treatment, before a cure results.

McCarty (17) states that "death occurs rarely, especially so when there is no central involvement. In general the prognosis is better in local than in general neuritis, the latter more often resulting in permanent paralysis and muscular atrophy. The

process is especially severe in cases suffering from obstinate vomiting, and septic cases show a tendency to early atrophy. Mental changes in connection with neuritis give a poor outlook. Lesions appearing during pregnancy and which do not disappear in three or four weeks may persist until shorty before or after delivery.

"Recurrences are unusual, although Gowers reported a case of a woman who had puerperal ulnar neuritis after two confinements. Relapses may occur more often but in such cases an undiscovered focus of irritation should be sought.

"When caused by a mechanical factor the prognosis is good if the condition is due to pressure applied for a short time, but recovery is slow in cases of extensive pelvic inflammation when pressure of the exudate is continued for some time. In the latter case the process may extend until it involves the whole sacral plexus."

Pokozdy (23) writes that polyneuritis has a tendency toward recurrence. Therefore it is not surprising that it reappears during a second pregnancy; but only one case had been described where a recurrence occurred without a second pregnancy. He reports a case with three relapses after the termination of pregnancy, and states that the case proves that a certain hypersensitiveness remains after polyneuritis of pregnancy not only towards the original toxins but also towards their endogenous

PROGNOSIS

or exogenous products.

Ledoux (14) writes that the symptoms do not change during the entire course of pregnancy and do not become worse as the pregnancy approaches term. Usually most of the symptoms disappear after delivery; the pains cease immediately and then the mental disorders diminsh and disappear. In some cases a certain degree of amnesia persists for several weeks. On the other hand, the motor disorders and the amyatrophy require several weeks or even months to disappear.

Fink (8) points out that the prognosis in these cases is guarded. In general, it may be stated that the gestation neuritides improve following the termination of labor. Occasionally, however, a complete cure can not be obtained, and some cases are refactory to treatment in general. He concludes that the gestation neuritides do not, as a rule, justify a termination of labor.

Plass (22) reports eight cases of toxic polyneuritis following hyperemesis gravidarum with four deaths and another case pending fatality. In one case the interruption of pregnancy apparently did some good. Plass states that emptying the uterus may not stop the advance of the disease; and if the vital (bulbar) centers are attacked, the prognosis is much more grave.

Electrical reactions are a valuable guide in the prog-

PROGNOSIS

nosis.

Acosta-Sisson (1) writes that in mild cases the symptoms entirely disappear immediately after parturition or with in a few days thereafter. In the more severe cases *the* numbness in the extremities affected last longer; that is, they do not disappear until two or three months after parturition. In extreme cases, there are accompaning muscular atrophies, absence of patellar reflexes, presence of foot-drop -- in short, all signs and symptoms of pseudo-paresis. Recovery in these cases takes five months or more.

TREATMENT

Nicholson (20) in 1904 writes "I feel that a study of the propholaxis of the condition points out some very important facts which practical obstetricians may well take cognizance of. I refer to the treatment in cases of delayed labor, whether due to contraction of the pelvis or not, for it will be remembered that cases of neuritis may occur from toxic causes which we can not govern and after easy labors, by far the greatest number are the result of delayed labor from some of its many causes. The necessity is therefore enforced that pelvic examination be made before labor in order to determine whether or not there may be any factor such as contraction, which will tend to produce delay and that when labor has once begun that an accurate diagnosis of position be made and forceps applied when indicated. The use of chloroform to diminish spasm of the pelvic muscles and thus decrease their bulk would seem also an important agent. As regards the application of forceps, it is well to be remembered that while their use is advised in proper cases it is presupposed that they are intelligently applied and used, for while we do not feel that the lumbo-sacral plexus is at all likely to be injured by even a faulty use this does not hold good as regards the lower sacral

nerves. It is hardly needful to emphasize the importance of asepsis as the production of neuritis from pelvic foci of infection is but one of the many evils resulting from its neglect."

Karr, quoting Tashjian (26), "concludes that there is a definite relationship between the desire to partake of food and the amount of the so-called vitamin B in the diet.

"It seems that where there is a nutritional disturbance or a nutritional deficiency, there will also be a vitamin deficiency. Soon a vicious cycle will set in, as the vitamin B deficiency will cause further loss of appetite.

Hornung and Creutzfeld (12) write "in what way may one now consider the question of interruption of pregnancy in a case of Landry symptom complex? In the acutely progressive cases, whether they are considered to be toxemia of pregnancy or a separate intercurrent disease, one can hardly minimize the violent course of the catastrophy, to say nothing of being able to prevent further development. Nevertheless we would again interrupt in a second similar case, and indeed more certainly the farther the pregnancy has progressed; for in this regard we are not confronted less with the cure than the purpose or object in mind.

"In the interruption of pregnancy we are then first concerned to improve the possibility of better aireation of the lungs of the mother by emptying the uterus. And ofcourse this is

the only way in which help may be given even though there is so little hope.

"Now in the subacute and chronic cases of Landry's paralysis: should we interrupt pregnancy? Not every neuritis and also polyneuritis in pregnancy justifies interruption as a medical indication --- this is definitely understood. However, as soon as it is definitely determined that the condition is one of Landry's paralysis type, especially the progressive ascending paralysis type, we are, in accord with Sachs, definitely in favor of interrupting pregnancy ------. We want to emphatically advise against waiting until vagus or (phrenic) diaphragmatic symptoms appear because then the interruption may be too late; the changes of the toxic stages in the nerves or spinal cord are then probably too well fixed to successfully combat the major danger, the threatened respiratory paralysis; even though that were possible it may be that following disturbances of swallowing areas of lung may be involved by aspiration material, which as in our one case led to death in spite of apparent improvement of the neurological symptoms following interruption of pregnancy."

McCarty (17) points out that the treatment varies with the nature and extent of the process. An attempt should be made to eliminate all sources of irritation or toxemia. Mild cases

clear up in two or three weeks with out treatment. In more obstinate cases massage and electrical stimulation may be advisable, but many cases will recover only after delivery. In these cases treatment is directed toward prevention of muscle degeneration until such time as delivery occurs.

In discussing the general treatment of peripheral neuritis it is convenient to follow Pershing (21) and divide the disease into three stages: 1) the stage of advancing muscular paralysis; 2) the stage of arrest; 3) the stage of convalescence and regeneration.

Diet. -- In all forms of neuritis and in all stages the food should be as abundant and as rich in proteins and fats as the patients condition will permit. It should be given in moderate quantity four to six times daily rather than in a large amount three times a day. The patient's emotional state is of the greatest importance and the physician must use his utmost skill and tact to conduct the feeding process so as to occasion the least possible distress or alarm.

Elimination. -- If an abundance of food is to be taken in a disease caused by a poison, free elimination is obviously necessary. Small doses of calomel with tonic and saline laxatives should be used to secure sufficient action of the bowels, but with out such a degree of purgation as will weaken or interfere with

rest. Plenty of water should be taken for its diuretic action.

Tonics. -- Strychnia is especially indicated on account of its influence in increasing appetite and digestion, its tonic effect on the heart and respiration, its general tendency to stimulate motor neurons. Ifthe heart is weak and rapid, digitalis should also be given. If respiration is threatened, atropia hypodermically will tend to sustain it.

Rest. -- In the first stage rest is by far the most important desideratum. Every muscular contraction presses upon and irritates the nerves, thus hastening the destructive process as well as increasing the pain. In all but the lightest cases, rest should be in bed, and as nearly absolute as possible. If the heart and respiratory muscles are seriously weakened the patient ought not even to sit up in bed.

Warmth. -- In the first stage thermal rest is as important as mechanical rest. Cold is depressing, uncomfortable, and dangerous to the tissues. Moderate warmth as nearly equable as possible is most soothing and most favorable for subsidence of the inflammation.

Posture. -- In the most painful stage the position of greatest ease is naturally choosen. The tendency to deformity must be borne in mind. The feet should be supported in an anatom-

ical position and protected from the bed clothes. To prevent fixation of any of the joints passive motion should be begun as soon as it can be carried out with out causing severe pain at the time, or more than slight pain persisting for a short time after manipulation.

Massage. -- In the early stage of acute pain and advancing paralysis massage ought to be entirely omitted. After the advance of the disease is arrested and pain has subsided massage should be begun. At first it should be merely a gentle superficial upward stroking of the limbs, but as tolerance is ascertained the rubbing may reach the deeper tissues so as to favor the flow of lymph and venous blood to the trunk. The passive motions already begun can now be combined with massage and made more vigorous. If any muscles, especially the calf muscles, show a tendency to shorten they may be stroked to favor their relaxation while being stretched by the appropriate passive motion.

Electricity. -- In the early stage of acute pain and advancing paralysis electricity should be also omitted entirely. Galvanic electricity may be used with advantage during the second stage. It is essential that each group of muscles as it is treated should be relaxed by posture and thus be free to contract. The pull on the muscle should be the one which causes the greater

contraction. The current should be slowly made and broken and strong enough to cause fairly vigorous action.

Convalescence. -- In the third stage electricity, massage, and passive movements are to be kept up until returning motor power and acitve exercises render them unnecessary. In sitting foot-drop must still be guarded against by seeing that the ball of the foot is supported while the heel is free to drop. If the calf muscles tend to s horten, they can be stretched by attempts to stand or walk. As soon as the patient is able to walk at all they generally yield and improvement goes on rapidly. Warm baths favor relaxation. In only a few cases willsection of tendons be necessary. The open air, tonics, food, recreation, and remedies to favor digestion and elimination will naturally be suggested. Finally, as in other cases of long-continued illness, convalescence can often be hastened and made more complete by change to some agreeable place in a climate which favors out door rest and recreation.

SUMMARY

1). Local or multiple neuritis may occur during early or late gestation or during the puerperium.

2). Etiologically neuritis complicating pregnancy may be due either to a toxic, inflammatory, or mechanical process.

3). The pathological lesion is essentially a degenerative process within the myelin sheath of the peripheral nerves.

4). It varies in severity from a simple disturbance of sensibility to complete anesthesia, paralysis, and muscular atrophy, and may be accompanied by psychic disturbances comparable with the Korsakoff's syndrome.

5). The prognosis is favorable in local neuritis and grave in the multiple type, particularly with evident signs of vagus or phrenic nerve involvement.

6). Therapeutic abortion is perhaps too long deferred in many cases, and is perhaps the best remedial measure and the most sure means of preventing multiple neuritis in pregnancy.

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