THE PARALYSES OF PREGNANCY AND THE Puerperium.

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by

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THE
PARALYSES OF PREGNANCY
AND THE
PUERPERIUM.
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Introduction.
History.
Classification.

Paralyses of Central Origin.
Cases.
Etiology and Pathology.
Prognosis.
Treatment.
Paralyses of Peripheral Origin
Cases.
Etiology and Pathology.
Prognosis
Treatment.

Summary and Conclusions.

References.
THE
PARALyses OF PREGnANCY
AND THE
PUERPERRYM.

Introduction:

Maternal paralysis arising out of the pregnant state and frequently following parturition is a subject which has not received a great amount of attention in later years. The evidence of medical literature and experience in the general practice of medicine alike shew that certain morbid products of gestation produce deleterious effects - direct and indirect - on the nervous tissue of the mother.

Eclampsia and puerperal insanity are striking effects of these morbid products on the central nervous system, and these have received their full meed of attention from obstetricians and psychiatrists without yet resulting in definite conclusions as to the true nature and cause of these phenomena.

While from the standpoint of the neurologist the phenomena of eclampsia and puerperal insanity are more complex and involved - because of the 1.
affection of the higher brain centres - than those of paralysis, yet the apparently simpler problem is no less difficult when the question of causation is considered. Eclampsia and puerperal insanity are truly related to gestation and parturition, but the same cannot be stated with regard to paralysis appearing during gestation or after parturition. Paralysis may result from many causes independent of the pregnant state, and its occurrence during pregnancy or the puerperium may simply be a coincidence without any causal relationship to these states.

It is the aim of this thesis to support the opinion that diverse forms of paralyses in pregnant and puerperal women arise directly from the pregnant or puerperal states without existing disease.

There are distinct sex characteristics in paralyses. Functional paralyses of hysterical origin are limited almost wholly to the female sex, while general paralyses of the insane is almost limited wholly to the male. Paralysis from cerebral haemorrhage caused by arterial degeneration and cardiac hypertrophy is largely a feature of the male sex. Some statistics give
a proportion of 3 of males to 1 of females.  

On the other hand paralyses from embolism of the cerebral arteries arising from rheumatic endocarditis is more common in women. Osler 2.

Paralyses of peripheral origin are also more common in women. In lead workers the proportion of women attached to men is four to one, and alcoholic women are much more frequently attacked by neuritis than alcoholic men. The peripheral nerve tissue of women is thus more susceptible to the poisons of alcohol and lead than that of men. Other forms of peripheral paralyses such as paralysis of the oculo-motor nerve in a recurring form occurs chiefly in women. There is no evidence to shew that the toxin of diphtheria has any selective powers over the peripheral nerve tissue of one sex more than another, and one can hardly believe that it can, still it is possible that if diphtheria were a toxaemia of adult life when sex characteristics are fully established that some selective affinity might be disclosed.

Clinical experience has shewn in addition to the ordinary morbid and toxic causes of paralyses in women that gestation, parturition and especially the puerperium may give rise to certain forms
of paralyses serious in character and calling for attention. These puerperal paralyses as they have been named because they arise most commonly during the puerperium are by no means infrequent. In my experience of six years in hospital and general practice they have been the most frequent form of paralyses I have met with in women, but this experience is, as far as I can gather, rather exceptional.

The following is a list of the cases of paralyses in women met with during the last six years:

<table>
<thead>
<tr>
<th>Year</th>
<th>Place</th>
<th>Age</th>
<th>Diagnosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>1906</td>
<td>North Shields.</td>
<td>50</td>
<td>Paraplegia Myelitis.</td>
</tr>
<tr>
<td>1906</td>
<td>&quot;</td>
<td>34</td>
<td>Peripheral Neuritis Puerperium.</td>
</tr>
<tr>
<td>1906</td>
<td>Oldham Infirmary</td>
<td>40</td>
<td>R. Hemiplegia, Septic Endocarditis.</td>
</tr>
<tr>
<td>1907</td>
<td>Oldham.</td>
<td>25</td>
<td>Paralysis going on to Catalepsy.</td>
</tr>
<tr>
<td>1911</td>
<td>Warrington.</td>
<td>38</td>
<td>R. Monoplegia and Aphasia Puerperium.</td>
</tr>
<tr>
<td>1911</td>
<td>&quot;</td>
<td>43</td>
<td>R. Hemiplegia and Aphasia Puerperium.</td>
</tr>
<tr>
<td>1912</td>
<td>&quot;</td>
<td>33</td>
<td>&quot;</td>
</tr>
<tr>
<td>1912</td>
<td>&quot;</td>
<td>19</td>
<td>R. Hemiplegia Cardiac</td>
</tr>
<tr>
<td>1912</td>
<td>&quot;</td>
<td>23</td>
<td>Paraplegia Hysteria.</td>
</tr>
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Continued:

<table>
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<th>Year</th>
<th>Place</th>
<th>Age</th>
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</tr>
</thead>
<tbody>
<tr>
<td>1912</td>
<td>Warrington</td>
<td>45</td>
<td>L.Hemiplegia C Haemorrhage.</td>
</tr>
<tr>
<td>1912</td>
<td>&quot;</td>
<td>50</td>
<td>Facial Paralysis.</td>
</tr>
<tr>
<td>1912</td>
<td>&quot;</td>
<td>27</td>
<td>Facial Paralysis Puerperium.</td>
</tr>
</tbody>
</table>

In all 12 cases of diverse forms of paralysis and of these 5 were associated with puerperal state. These five cases of puerperal paralysis can be divided into 2 classes - central and peripheral.

Three cases being of central origin

Two cases being of peripheral origin.

Paralysis associated with the puerperium is not confined to the human subject. Some domestic animals particularly the cow are afflicted by what is known by veterinarians as parturient apoplexy. In the cow this comes on from one to three days after parturition and the cow drops paralyzed and semi-comatose. Veterinarians believe this condition to be due to a poison from the udder and treat it by injections of oxygen into the udder. The sow and bitch are also liable to be attacked by paralysis two or three weeks after
parturition.

The subject of puerperal paralysis receives scant notice in most books on obstetrics. In the larger works a paragraph or two at the end is considered sufficient, while in the smaller books the condition is hardly mentioned, or only a passing reference is made to blocking of the systemic arteries in old endocarditic cases of rheumatic origin or where acute endocarditis has arisen from puerperal sepsis. Physicians have considered the matter more fully. Taylor\textsuperscript{3} and Osler\textsuperscript{2} have discussed the occlusion of the cerebral vessels in the puerperal state apart from existing or evident disease, and several explanations have been offered.

Recent literature on the subject is not very extensive and consists chiefly in the publication of isolated cases in various periodicals. The majority of these cases have been reported by American and Continental physicians - British records are few. Only two cases have been reported to the Obstetrical Society of London during the past fifty years and five other cases have been recorded in the journals.

About the middle of last century the subject engaged the attention of many obstetricians of note, among them being Sir James Simpson.
Charpentier of Paris in 1872 wrote a discursive study of the subject and gave its history. He was able to quote 149 cases more or less complete from the literature. These he classified from the clinical standpoint and discussed their etiology, diagnosis and prognosis. In this thesis I am able to quote 5 cases which I have personally met with, and a colleague of this town - Dr. Anderson - has given me a short account of two cases which have fallen to his experience. Dr. Llewellyn Morgan of Liverpool has given me some notes on a case which happened quite recently in his practice there. From the literature available in the library of the Manchester Medical Society I have only been able to glean nine cases, but a recent number of the British Medical Journal brought two more cases by Dr. Gillies of Dumfries.

In all 19 cases.
HISTORY.

From the study of Charpentier I have translated a short summary of the history of the subject as given by him. Puerperal paralysis as a clinical entity was known to the ancients and of these Hippocrates gave it the most marked attention, and attributed the paralysis to the suppression or retention of the lochia. In later times Mercatus (1608) Tulpius (1640) Obs. Medic. Co.II) Primerose 1655 (De morbis mulierum) and others, and among the latter are the distinguished names of Sydenham and Boerhaave - discussed the subject and attributed the condition like Hippocrates to the suppression or retention of the lochia. Primerose, Sydenham and Boerhaave however admitted that apoplexy might be coincident with the puerperium. Mariceau later pointed out that convulsions were the chief symptoms associated with paralysis and that convulsions often terminated in apoplexy. After Mariceau the doctrine of the suppression of the lochia gave place to that of the metastases of the milk, and this theory was upheld by many during the 18th century. For example by Pugos (Traité d'accouch. 1759) Levret (Traité d'accouch. 1775)
Towards the end of the 18th century and during the first half of the nineteenth, closer observations were made with the view of localising the cause of the paralysis and amongst the more important works were those of Bestchke (Ueber Schlagflussen und Lahmungen Leipsik 1793) Portal (Mémoire et Traité de l'apoplexie 1803-1812) Bandelocque neveu (Thèse 1822) Burns (Traité d'accouchements)

Ménière (Memoire sur l'hémorragie cérébrale pendant la grossesse, pendant et après l'accouchement. Arch de Med 1832)

Ménière concluded that while the hypertrophied left ventricle of pregnancy - a fact noted by Schedel - played a part in the production of cerebral haemorrhage yet there were other causes, such as the increased quantity of blood in the mother, the violent expulsive efforts during parturition, dyspnoea, oedema, and moral causes: he rejected the theory of the suppression of the lochia.

Ménière's explanations were not fully accepted, especially as he did not lay sufficient stress on the hypertrophied left ventricle, and further investigations shewed the heart to be affected in many of the cases of puerperal paralysis.
Thus Kirkes (Edin. Med. & Sur. Journal, 1853.) pointed out a variety of endocarditis which he named ulcerative typhoid and pyaemic and which was studied later by

Virchow (Gesamm Abhaulbengen 1856)

Simpson (1854 - 1856)

Bamberger (Lehrbuch der Krankheiten der Herzens Wien 1857)

and others.

Charpentier says:--

Mais c'est à Simpson (Obs. work tII 1856 Edin. Monthly 1854) que revient l'honneur d'avoir clairement demontre' l'influence de l'etat puerperal sur la production de cette forme d'endocardite.

From the study of puerperal endocarditis others were led to a study of the blood itself. Pigeaux 1839 had already pointed out that there was an alteration of the blood in pregnancy and Regnault (1847) These Paris) by his haematological researches had shewn that in pregnant women there was a diminution of corpuscles, an increased amount of serum, and an excess of fibrin.

Grisolle, Niemeyer, Lancereaux, Virchow and Simpson agreed to this and added that the retention of urea and lactic acid together with the fact that
the blood was charged with new material, vitiated the blood and favoured the onset of endocarditis.

Decorniere from 22 observations of puerperal endocarditis showed that the most frequent cause of hemiplegia was red and white softening of the brain, caused by emboli from the heart valves.


Simpson divided the causes into 5 classes:

(1) By the detachment of old and organised concretions from the heart to the arteries.
(2) By the passage in the circulation of recently formed masses of fibrin from the heart cavities or great arteries.
(3) By local arteritis.
(4) By a local lesion in the internal coat of the arteries.
(5) By a morbid material forming in the venous system and lodging in the pulmonary artery or its branches.
While endocarditis rheumatic or otherwise and alterations in the state of the blood explained satisfactorily a great number of cases, there were some other cases which required another explanation, and the next most important addition to our knowledge was due to


Simpson (Edin. Med. & Surg. Journal) also pointed out that albuminuria was a special complication of paralysis.

Imbert Gourbeyre 1860 laid great stress on albuminuria, and for him puerperal paralysis was only an apoplectic form of eclampsia. The presence of albuminuria led others to suspect that defective action of the kidneys might lead to the accumulation of waste products such as urea, ammonium carbonate, oxalic acid and other chemical bodies.

Charpentier also considered the question of anaemia following excessive postpartum haemorrhage as a cause of paralysis, but could give no satisfactory evidence to support this view. He also
considered reflex action as a cause of paralysis and mentioned several cases of short duration which were reported without recognisable cause. No cardiac disease, no albuminuria, no eclampsia, no anaemia or puerperal infection.

In the foregoing history Charpentier is chiefly referring to hemiplegia for he reserves a separate section of his study for paraplegia, partial paralysis and paralysis of the senses.

Frequency: Charpentier believed puerperal hemiplegia to be very infrequent, could only collect 57 cases in the literature, and among 1600 observations in his own practice he did not have one.

He found that the condition was most common between the ages of 25 and 30 and at the first pregnancy.

Prognosis: Among the 57 cases, there were 20 deaths, and out of the 20 deaths there were 13 autopsies. Some of these shewed cerebral haemorrhage with and without kidney lesions. Some shewed heart lesions and others cerebral thrombosis.

Paraplegia.

Charpentier mentions that Mereatus 1608 first pointed out the occurrence of puerperal
14.

paraplegia and others like him attributed it to suppression of the lochia or metastases of the milk.

Churchill 1854 pointed out the relation between paraplegia and albuminuria, but Imbert Gourbeyre who favoured the association of albuminuria and hemiplegia did not mention a case, and divided puerperal paraplegia into two classes (1) those following traumatism of labour (2) those following myelitis.

Charpentier quotes two cases of haemorrhage into the cord and a few others associated with puerperal infection giving rise to meningitis.

He divides puerperal paraplegia into three classes:

(1) Organic paraplegia with well defined lesions.
(2) Reflex paraplegia.
(3) Paraplegia from morbid conditions of the blood, anaemia.

Frequency: He concluded that paraplegia is not nearly so frequent as hemiplegia and was only able to quote 25 of the former against 57 of the latter. Age has no influence and the condition is most frequent in the first pregnancy.
Partial Paralysis.

Under this heading Charpentier quotes several cases of traumatic paraplegia following labour, and mentions the opinions of various writers notably Burns 1839 and later Bianchi Farnier and Pollen, all of whom regarded the condition as due to pressure of the foetal head on the sciatic nerve the sacral plexus, and in particular on the lumbo-sacral cord. In some cases only one leg was affected, in others both legs. He also mentions various cases of partial hemiplegia and several cases of facial paralysis - 9 cases out of 21 of other forms. He attributes these partial paralyses to albuminuria, rheumatism and reflex action.

Frequency: He is able to give 30 cases of partial paralyses and of these 19 were incomplete hemiplegias and 11 incomplete paraplegias. Thus comparing hemiplegias with paraplegias

<table>
<thead>
<tr>
<th>Complete hemiplegia</th>
<th>57</th>
<th>Complete paraplegia</th>
<th>25</th>
</tr>
</thead>
<tbody>
<tr>
<td>Incomplete</td>
<td>19</td>
<td>Incomplete</td>
<td>11</td>
</tr>
<tr>
<td></td>
<td>76</td>
<td></td>
<td>36</td>
</tr>
</tbody>
</table>

Age has no influence and as before these paralyses are most common in the first pregnancy.
Paralyses of Special Senses.

Charpentier states that paralysis of the special senses in the pregnant or puerperal state are in the great majority due to albuminuria, although some may be due to hysteria or anaemia. These paralyses are rarely isolated, and are usually associated with other forms such as the limbs or face.

Vision is the most frequently affected of all and amaurosis is usually the result of albuminuria.

Hearing is the next most frequently affected and the deafness of pregnant women was alluded to by Hippocrates and others. This is also associated with albuminuria. Paralysis of taste and smell has not been well established by authentic evidence.

Summary of Charpentier's Main Conclusions.

That puerperal paralyses resolve themselves into two main groups:

I Paralysis from organic lesion

II " from reflex action.

The first group is divided into

1. Primary organic lesions - congestions haemorrhages meningitis, lesions of the membranes of brain and cord.
2. Secondary organic lesions - affections of the heart thrombosis of cerebral vessels, albuminuria, uterine affections and compression of nerves. He lays special stress on the co-incidence of albuminuria and paralysis, and also considers that the condition of the blood in the puerperal state conduces to the production of paralysis.

The second group - Paralysis from reflex action - arises from peripheral irritation.

The total number of cases quoted is 149 divided as follows:

- Hemiplegia 57
- Paraplegia 25
- Paralysis (Traumatic) 12
- " partial 21
- " of the senses 34

Such is a brief summary of Charpentier's treatise.

**Classification.**

Charpentier's classification is purely clinical and arbitrary and leads to confusion both as to the cause and site of the lesions producing the paralysis. It is manifestly absurd to separate the complete from the incomplete hemiplegias.
In this thesis I propose to divide the forms of paralyses into two main anatomical classes:

I Paralyses of Central origin.

II Paralyses of Peripheral origin.

These classes will later be subdivided into divisions in accordance with their pathological factors so far as these are brought out in the discussion to follow.

The classification ultimately arrived at will thus be anatomico-pathologic.

Before proceeding to record the cases, it is appropriate under this heading to recapitulate the succeeding etiological theories of puerperal paralysis.

1st. The Hippocratic theory of the retention or suppression of the lochia.

2nd. The eighteenth century theory of the metastases of the milk.

3rd. Cerebral haemorrhage. Hypertrophied left ventricle.

4th. Puerperal Endocarditis (Middle 19th Cent.)

5th. Changes in blood during pregnancy.

6th. Albuminuria.
ADDITIONAL CONTRIBUTIONS
to the
Knowledge of the Subject.

Cases
and
Discussion.
Paralysis of Central Origin.

Case I. Craik 6

Woman 36. First pregnancy.

When six months pregnant the patient experienced a feeling of numbness and burning in the hand and fingers of the right side, with gradual weakness of arm and leg of that side and increasing difficulty of speech. At the 9th month labour came on and was tedious requiring forceps. After parturition there was a gradual recovery until at the end of 3 months speech and movement were completely restored.

No history of syphilis, lead or alcohol.

Heart and kidneys healthy.
Paralysis of Central Origin.

Case II \hspace{1cm} Rusch 7

A woman, age 33, ninth confinement, normal puerperium. Three weeks after parturition she was seized with paralysis of the right side and aphasia. Three and half weeks after the onset of the paralysis she was admitted to hospital.

No abnormal physical signs were discovered in the circulatory respiratory or alimentary systems. A considerable amount of the paralysis had passed off at the time of admission but the aphasia was marked. The condition of the urine was not noted. The patient went on well for a week or so but died suddenly nine days after admission to hospital.

Autopsy: This revealed a large embolus at the origin of the left middle cerebral artery and a large clot was present in the left lateral ventricle. An area about the size of a walnut near the posterior part of the left 3rd frontal convolution was transformed into a red diffluent mass. All the other organs were healthy excepting the liver and there only a small blood cyst was discovered on its surface.
Paralysis of Central Origin.

Case III. Scougal

Woman. 37. 7th confinement, easy labour, good recovery, suckled child. At 9.30 p.m. on the 7th day after confinement she complained of numbness of 1st, 2nd and 3rd fingers of left hand, and 4 hours after she was noticed passing her hand down her left shoulder. At 3.30 a.m. Scougal arrived and found left arm completely paralyzed with paresis of left leg and divergent strabismus of right eye. Speech was unaffected and patient was conscious. Temperature normal, pulse 96. Two days later there was complete paralysis of the left side and on the 4th day after the onset of paralysis the patient died.

Autopsy: Brain only. Clots were found in the vein of the right middle meningeal artery and in the vein of right middle cerebral artery. No sign of thrombi in the sinuses, or extravasations of blood in other parts of the brain. The clots were distinctly ante-mortem and adherent to the vessel. Brain substance free from morbid appearance. Condition of urine not mentioned.
Paralysis of Central Origin.

Case IV. Horrocks 9

Woman 28. 2nd pregnancy.

This patient had had influenza during May 1891 followed by pains in head and vomiting. In a few days she became drowsy and passed her motions under her. Pupils equal, no noticeable paralysis, urine clear, no sugar or albumen. Labour set in during the last few days in May and progressed slowly. On June 1st she was delivered of a living child. She remained unconscious. The right leg and arm were constantly moving. The left leg flaccid and motionless and only partial movement of left arm. She died next day, 31 hours after confinement.

Autopsy: Thrombosis of veins of Galen and straight sinus, also of the right temporo-sphenoidal vein and other cerebral veins. Extravasation of blood into the optic thalamus right and left, and on the right side extending as far as the internal capsule. Suppurative nephritis in right kidney. Cystitis present. Heart and uterus healthy.
Paralysis of Central Origin.

Cases V & VI Gillies 5

Mrs. M.H. aged 22. was confined of her second child on June 6th. She had "flitted" on the preceding day from a farm distant thirty miles from her new home; the journey had been done perched upon a quantity of household furniture on an open cart. About 8 p.m. on June 5th she felt her first pain and the doctor was sent for at 4 a.m. The doctor found the head on the perineum, and the child was born in a few minutes; the placenta was expelled ten minutes afterwards. The whole confinement was perfectly natural; the child was a poor thing, at about the seventh month. The course of after events was normal. The doctor paid his last visit to the mother on June 14th, and allowed her up the next day.

On July 2nd, nearly a month after the birth, while sitting at the fireside nursing the child, the mother "was seized with a fit, her face was working, and she was all twisted to one side, her eyes were rolling in her head." The doctor found her in bed; the face was pale and covered with a cold perspiration, the breathing was stertorous;
the head, face, and eyes were drawn to the right side (conjugate deviation); the left arm and leg were flaccid, and when lifted dropped on to the sheet heavily. The pupils were dilated, and the conjunctival reflex was not quite abolished, though lazy. The sounds in the heart areas were normal, and there was no sign of any valvular trouble. The pulse was 78, the vessel wall soft, and the wave full and regular.

Next day she was completely conscious, and four days after the power had to a great extent returned in the affected parts, the hand being the last part to recover. A month after, the recovery was perfect in every part, with the exception of the thumb, which is to-day weak and shrunken.

On inquiring into the history after her recovery, the only thing of any importance was a story of chorea when she was 9, otherwise there was no neurotic history. There was no albumen in the urine either then or at the confinement.

**Case VI.**

J.W. aged 25. her second child was born on August 1st; the confinement perfectly normal and the puerperium uneventful.

On September 27th, while sitting by the fire
with her baby, she fell off the stool, dropping the baby beside her on the floor; in this position she was found by a neighbour, who procured assistance and had her lifted into bed. She never lost consciousness, but remembered all that happened quite well. Paralysis affected the left arm and leg, the face and eyes being quite normal. She complained of most intense headache, which seemed to be of maximum intensity over the right side and above the right ear. The heart impulse was in the fifth interspace and powerful; a murmur was heard, of greatest intensity in the aortic area and systolic in rhythm; the pulse was 90.

The paralysis of the limbs lasted for three days, and then quickly recovered. She is now quite well. In this case also there was a history of chorea during girlhood, but nothing else of any importance.
Paralysis of Central Origin.

The remainder are unpublished cases:

Case VII. Manson.

Mrs. M. aged 33. This patient had always been a healthy woman following the arduous occupation of a hospital nurse. She was trained at the London Hospital where the medical examination is strict before entry and was afterwards 4 years a sister at the Oldham Infirmary. She was liable to attacks of urticaria.

During the spring of 1908 she was feeling run down and was examined by Dr. Graham Steele of Manchester who could find no trace of organic disease.

Marriage took place in April 1909 and in July she miscarried when about six weeks pregnant. In December she had a severe attack of urticaria and angio-neurotic oedema which lasted nearly 6 weeks. The urine was examined and no albumen found. In 1910 January/she again miscarried at the 6th week. In July, 1910 another very early miscarriage occurred. On all occasions she was very sick while pregnant. For a year she was in good health, but
when on holiday in June 1911 the sickness of pregnancy commenced and she had to return home. She then stayed in bed for 4 months and afterwards felt exceptionally well and attended to all her duties. The urine was examined in December and no albumen was found.

Towards the end of January 1912, the patient began to swell about the eyes and feet, and when the urine was examined a large amount of albumen was found - .45 per cent by Esbach's albuminometer. The patient was then sent to bed and dieted on milk and farinaceous foods. The swellings diminished and the albumen decreased to .3 per cent on Feb. 10th. After this the albumen gradually rose to 1.0 per cent until parturition took place.

The S.G. of the urine varied from 1012 to 1025 and the amount from 203 to 353 per diem.

Labour commenced on night of March 4th and progressed slowly. The dilatation of the os was extremely slow. On night of March 7th the os was nearly dilated and forceps were applied under chloroform.

The child - a girl - was small and was with difficulty resuscitated. She had facial paralysis on the right side for over 6 weeks besides three
cephalhaematoma over the parietal and occipital bones.
The child lived and is now thriving.
There was considerable but not excessive post
partum haemorrhage.

After delivery and just as she was recovering
from chloroform she had an alarming syncopal attack
which passed off after a rectal saline. Eight hours
after another syncopal attack was passed through
and the patient's condition improved slowly during
the first week. The albumen declining to .2 per
cent.

On the 10th day she got out of bed but soon
returned.

On the 13th day a slight shivering fit occurred
and temperature rose to 99.4 but soon declined to
normal again, at no time during the puerperium was
the temperature above this and the pulse kept to
about 70. On this day she also complained of pain
in the right groin and an examination revealed a
laceration in the right side of the vagina. On the
14th and 15th days severe pain was complained of
on the left side of the head and the general condi-
tion was one of lassitude and apathy, and no sleep
was obtained. On the night of the 16th day after
parturition a sleeping draught of 12½ grains of
chloral hydrate and 20 grains of Potass. bromid. was
given.
The patient slept well during the night but at half past five in the morning the nurse called the medical attendant because she thought the patient's condition strange.

The medical man found the right arm and right leg and right side of face completely paralyzed and speech lost.

The right knee jerk was greatly exaggerated and there was an extensor response in the great toe. Patient was only partially conscious and the pulse was weak and frequent.

Next day Dr. Reynolds of Manchester saw her in consultation and diagnosed cerebral thrombosis and gave a grave prognosis as to recovery from the brain lesion but was hopeful as regards life.

He could find no trace of organic disease of the heart and regarded the condition as due to the increased coagulability of the blood, and would not accept the idea of an embolus from some possible pelvic inflammation. The patient's condition was extremely low and critical for three weeks and there was a distressing restlessness of the non-paralyzed left leg and arm.

On the 3rd day after the attack the temperature began to rise, and on the 4th day the right leg began to swell and become very painful. The
temperature on the affected side was from $1\frac{1}{2}$° to 2° higher than on the non-affected side.

There was complete incontinence of urine and faeces. Liquid nourishment was frequently administered in small quantities.

On the 8th day after a vaginal douche the patient had a violent rigor and became unconscious. An injection of pituitary extract was given and following this there was an almost explosive action of the whole alimentary canal - the contents of the stomach and bowel being simultaneously evacuated. Warmth was applied and the patient allowed perfect rest. Towards evening there was some improvement but on moving the patient a large bed sore was discovered on the right buttock. The patient was put on a water bed and her condition during the night was extremely grave. Next morning there was a slight improvement and some nourishment was taken.

On April 5th Dr. Wallace of Liverpool saw her in consultation but he could discover no phlebitis or evidence of sepsis in the pelvis.

The improvement was maintained with slight intermissions during the week and the temperature gradually fell to normal on April 11th.

On April 12th the first movement since the onset of the paralysis was noticed in the movement of the right thumb.
On April 13th the left leg became painful and the temperature rose again and fluctuated until April 19th when normal was reached.

After the first movement of the thumb the movements gradually returned to the arm and leg and speech began to return.

Slow and gradual improvement was maintained and patient was helped out of bed on June 9th. Continued improvement was maintained and soon the patient was able to go out in a bath chair. It was noticed at this time that she had some degree of homonymous hemiopia. The present condition eight months after the onset of the paralysis shews continued improvement. The patient can now walk out and take some part in household duties.

The right arm is still weak, but all the movements are present. The right leg is still swollen a little, but the movements are perfect. Speech is still very imperfect. The patient can copy writing and can write a few familiar words. She can read a little to herself but not aloud. There is evidently some word deafness and word blindness as well as anarthria.

The urine is now practically free from albumen.
Mrs. C. aged 39.

This patient says she has always been a healthy woman, and her appearance confirms the statement.

She was married at the age of 33.

1st pregnancy: A year after marriage her first child was born. Instrumental labour and normal puerperium.

2nd: Five months after this confinement she had a miscarriage.

3rd: One year and 9 months after this her second child - a girl - was born, confinement instrumental, puerperium normal.

4th: A year and nine months after this second child's birth a boy was born on the 5th April, 1911. Labour pains came on during the night and the child was born before the midwife could arrive. On the 3rd and 4th day she had painful piles.

During the puerperium she felt weaker than usual and had pains at the back of the head. In spite of this weakness she determined to get up on the 10th day. After dressing and going down stairs she became very ill, lost her speech and use of the right arm. Although she could not
speak, she was partially conscious and remembers people attending to her and putting her to bed.

The right arm was only partially paralyzed and chiefly affected the grasping power of the hand. The movements at the shoulder joint could always be performed.

Her speech during the first week was limited to Yes and no, and these words were often misplaced. The right leg was unaffected and after a stay in bed for six weeks she was able to get up.

Three months after she was at her ordinary duties, but speech was slow and defective, the right arm weak, and she still complained of headaches.

For some months after she could neither read nor write, but at the present time - 18 months after - she can read and write as well as before, but she cannot enjoy a book as formerly.

The heart is sound and there is no sign of kidney disease.
Mrs. B. aged 44.

Previous illnesses. When 13 years old was supposed to have inflammation of the hip-joint otherwise a healthy woman.

Was married at age of 31.

**Pregnancies:**

1st. 7 months after marriage a 7 months' child - a girl - was born, but only lived an hour.

2nd. About 18 months later a 6 months' child - a boy - was born dead.

3rd. 18 months later an 8 months' child - boy - born dead. There were no complications at these labours. All were born naturally without help.

4th. In May 1903 when aged 35 and living at Stacksteads, Lancashire she had pains at the 7th month, and her doctor ordered her to bed to prolong gestation. She carried child to the 8th month when labour set in. She was delivered with forceps, although patient thinks she would have had a natural labour if left alone. The child - a boy - was dead born.
Patient had a normal puerperium and got up on the 10th day. She went out on the 14th day and was feeling well. On the 22nd day after confinement she felt dizzy and staggered about, but slept well at night. Next day she felt queer about the head and staggered and went early to bed. About 11 o'clock she had an attack of sickness and vomiting and at 3 a.m. sent for the doctor because of persistent sickness and vomiting. On the forenoon of this day - 24th day after confinement - she felt her right side becoming numb and losing its power and by the afternoon she had lost power of her right arm and leg. Next day she lost her speech completely. She is certain that she did not lose her speech until the day after the paralysis of right arm and leg. She never lost consciousness nor did she have incontinence of urine and faeces. There was no swelling or arms or leg.

Ice was applied to her head and massage was commenced at end of 2nd week of paralysis when she had regained slight movements of the thigh.

Two or three days after this movements returned to forefinger middle finger and thumb and gradually movements returned to the leg and arm.

Speech began to return at end of 3 weeks, but it was fully a year before she could speak
5th Pregnancy: In the beginning of 1905 she gave birth to an 8 months' child - boy - still born. This was a tedious confinement, but required no aid. Puerperium normal, got up on 10th day.

6th Pregnancy: In early part of 1910 she had a 7 months' child, born alive, but only lived 7 hours.

7th Pregnancy: On November 5th, 1912, she gave birth to a 7 months' child - a girl - which lived 35 hours. The child was small but fully developed. No signs of disease. The urine was examined a fortnight before this birth and found to contain albumin.

At none of the confinements did she lost much blood. She had no white leg or swellings, but while pregnant she was always sick with vomiting.

Examination: Heart sounds normal, no evidence of cardiac disease. Arteries soft and compressible. Her speech is somewhat slow and jerky. Right arm is poorly developed when compared with left. She cannot supinate hand fully. The fingers are flexed on the palm and the thumb is in contact with the forefinger. There is great weakness of the supinators and extensors of wrist and fingers. The right arm is generally kept close to side of body.
Her gait presents a marked limp and her right foot drags slightly. The muscles of right leg are poorly developed as compared with the left leg. Knee jerk is greatly increased and there is an extensor response of the great toe.

Case X. Morgan.

Wife of a clergyman at Liverpool under the care of Dr. Llewellyn Morgan. Age 35. Paralysis came on after the 5th confinement. Labour was easy and everything went on well. On the night of the 5th day she was seized with complete left hemiplegia. Dr. Wallace of Liverpool saw her in consultation and found everything normal with regard to the puerperium. Dr. Hill Abram also saw her and came to the conclusion that the lesion was due to cerebral thrombosis.

There was no albuminuria, no heart or other organic trouble.

After a former confinement she had 'white leg' without signs of sepsis. Dr. Morgan concluded that the condition was due to the great liability of the blood to clotting in pregnancy.
Recovery was slow and power gradually returned to the face, flexors of arms, and then flexors of legs.

Ten months after the seizure, she was now going about but her gait was quite hemiplegic and there is no return of power to the extensors of the arms.

Case XI  
Anderson.

Mrs. L. now aged 50, lives at Fearnhead, Lancashire. 2nd confinement when she was aged 29. She became completely paralyzed on the left side. Before the confinement her face was swollen. After the confinement she went on well for 3 days but on the 4th day she became completely paralyzed on the left side, but did not lose consciousness. In 3 months she was able to get about a little, but she has a distinct limp and drags her left leg a little. There is still some paralysis of the extensors of the left arm.

She has had two confinements since and both children are alive and healthy.
Case XII  Anderson.

Mrs B. age 28. 2nd confinement, tedious labour, chloroform and forceps.

This patient had albuminuria during the latter months of pregnancy. No heart trouble.

Puerperium normal until 9th day when she developed left hemiplegia during the night. She was in bed 7 weeks and was then able to get up. At the end of 3 months she was considerably improved, and was able to remove to Burnley from Warrington.
Paralysis of Central Origin.

Pathology and Etiology.

In a study of the foregoing twelve cases it will be seen that the amount of detail relative to the cases, varies considerably, but in all the association of paralysis with pregnancy and the puerperium stands out prominently. That this association must be something more than a mere coincidence is evident when it is considered that none of the well recognised causes of paralysis of central origin exists in any of them. In only one (Case VI) was there any mention of a heart murmur, and in none was there any evident heart disease - chronic or acute. Only one case shewed evidence of sepsis and this was in the kidney (Case IV) and not in the uterus: in none of the others is there any distinct evidence of puerperal sepsis. Arteries healthy in VII VIII and IX - condition of vessels not noted in other cases.

Albuminuria was present in three cases VII, IX XII and is likely to have been present in XI. Was not present in I, IV, V, VI and X, and the condition of the urine was not given in II III and VIII.
The onset of the paralysis varies greatly. In Case I it appeared in the later months of pregnancy, while in Case VI it appeared 57 days after parturition. It was most common in the first 3 weeks after labour; eight cases out of the twelve occurred during that period.

Age: Seven cases occurred between 30-40: five cases between 20-30. The average age being 32.

Number of Confinement: Two cases occurred at the first, five at the second, the other five occurred from the 4th to the 9th.

Seven were affected with left hemiplegia and five with right hemiplegia and aphasia. In five with left hemiplegia and three with right hemiplegia, the onset of the paralysis was quick if not sudden, and in two with left and two with right hemiplegia the onset was slow and gradual.

Character of Labour: Seven labours are described as easy or normal: four as difficult and in one the character of the labour is not mentioned.

Condition of Child: In three cases the child is described as poorly nourished, of which one was still born; four were healthy children, and in five the condition of the child is not noted.
Condition of patient during pregnancy: Three had marked sickness and vomiting during pregnancy, of whom one had influenza. There is no mention of this feature in the other nine.

Morbid Anatomy: Of the twelve cases three died and autopsies were held. Nine recovered of whom four made a complete recovery and five incomplete.

Autopsy - Case II. A large embolus blocking left middle meningeal artery at its origin and large clot in left lateral ventricle and posterior part of 3rd left frontal convolution transformed into red diffuent mass. Other organs healthy.

Autopsy - Case III. Brain only. Clots in veins of right middle meningeal artery and right middle cerebral artery. These clots were distinctly ante-mortem and adherent to the vessel. No signs of thrombi in the sinuses or extravasations of blood in other parts of brain. Brain substance free from morbid appearance.

Autopsy - Case IV. Thrombosis of veins of Galen and straight sinus also of right temporosphenoidal vein and other cerebral veins. Extravasation of blood into the optic thalamus right and left, and on the right side extending as far as the internal capsule.
Suppurative nephritis in right kidney.

Cystitis present. Heart and uterus healthy.
Consideration of the various factors observed and inferred in the Etiology and Pathology of Paralysis of Central Origin occurring during pregnancy and the Puerperium.

The history of the subject, the observations set forth in the foregoing cases, together with evidence from collateral subjects, afford a basis on which arguments may rest for the discussion of the etiological and pathological factors.

The ancients as well as the older physicians recognised a distinct causal relationship between paralysis and the pregnant and puerperal states. Many of the cases cited to prove this were doubtlessly coincidences and due to heart and vascular disease apart from pregnancy. Others were due to indirect causes secondary to pregnancy, such as puerperal sepsis, sometimes resulting in septic endocarditis. The remainder required other explanations, such as reflex action or the altered state of the blood in pregnancy. These latter are direct or primary causes arising out of the pregnant state.
Paralyses arising from either the primary or secondary causes must fall under the title of this thesis, and I shall attempt to deal with the primary causes first. The older theories of reflex action, and the altered state of the blood in pregnancy still hold the field but as will be seen in a somewhat different form.

The proximate causes of the paralysis are vascular lesions and any vascular lesion in the brain or elsewhere depends on two main factors (1) The state of the blood as to pressure, chemical constitution, rate of flow. (2) The state of the heart and blood vessels whether healthy or diseased.

In the cases already considered there is no evidence of diseased heart or blood vessels. If there were any such evidence, the explanations of the paralysis would be simple and would refer to that disease rather than to the pregnant or puerperal state. This goes to show that the state of the blood is the important factor in producing the vascular lesions which in time produce the paralysis here considered.

Barnes fifty years ago said: "The condition of the blood in pregnant and lying in women if thoroughly understood would furnish
46.

the key to much of the pathology of child bed diseases."

The blood in pregnancy according to Marshall 11 who relies chiefly on German observations is not hydraemic as was formerly believed and the number of red corpuscles is not appreciably altered in normal pregnancy. The haemoglobin is slightly increased towards the end of pregnancy, and there is an increase in the number of leucocytes, due it is supposed to the increase of lymphatic glands in the neighbourhood of the genital organs. The total amount of blood is increased - at least in the dog - during pregnancy from 7.8 per cent to 9 per cent.

It is also stated that the alkalinity of the blood is increased during pregnancy but this needs confirmation. The molecular concentration of the blood shows no changes in pregnancy.

Kermauner 12 states that in normal blood the fibrinogen amounts to .31 per cent apparently arising as a by-product of the destruction of leucocytes. It is destroyed in the lungs.

"Zerstört wird es in den Lungen." In healthy pregnant women the fibrinogen increases corresponding to the leucocytosis to .45 per cent. In eclampsia after the attacks the average is .53 per cent while during the attacks it rises to .61 per cent and more especially when the organ of
elimination - the lung, is damaged. In his article Kermauner assigns definitely a new and important function to the lungs, namely, the destruction or elimination of fibrinogen. If this be true then it follows that the blood in the pulmonary veins must be less coagulable than the blood in the pulmonary arteries. This is an important inference when considered from the point of view of this thesis.

Dienst 13 who has made a special study of the bio-chemistry of the blood of pregnant women, especially with the view of elucidating the cause of eclampsia says that fibrin ferment is not normally a constituent of the blood but in pregnancy both fibrinogen and fibrin ferment are increased and if these are not neutralised destroyed or eliminated then there is a precipitation of fibrin and an excess of fibrin ferment in the blood. Fibrin ferment, he says, is formed from 'thrombogen" a product of the red corpuscles. This 'thrombogen' is as a rule neutralised by a product of the liver cells known as 'anti-thrombin.' In pregnancy owing to congestion of the liver there is a deficiency in anti-thrombin so that 'thrombogen' in excess unites with the 'thrombokinase'(also a product of the breaking down of blood corpuscles)
to form 'prothrombin' and this latter is converted into fibrin ferment in the presence of calcium salts.

During pregnancy large numbers of blood cells are being used up in the placenta for the nourishment of the embryo, thus there is an increase in the fibrin forming elements, and the coagulability of the placental blood is increased. The venous blood of the uterus is poor in calcium but this factor is supplied when the blood in the right heart is mixed with that from the portal veins. If this blood be deficient in 'anti thrombin' as it often is in pregnancy then fibrin ferment is formed and fibrin precipitated. Further if the kidneys are impaired the excretion of this toxic ferment is hindered and leads to the convulsions known as eclampsia.

These theories of Kermauner and Dienst explain how the blood in pregnancy is more coagulable than normal and they further explain the occurrence of thromboses in various parts, especially in the veins as in 'white leg' or the cerebral veins.

Russell 14 regards thrombi once formed as difficult things to get rid of, and this may be true of thrombi in diseased and sclerosed vessels but in pregnancy Abderhalden's recent researches
(as quoted by Ballantyne\textsuperscript{15}) shew that in the maternal blood there are protective enzymes which digest unusual components of the circulating blood such as proteins fats carbo hydrates and even protein derivatives of the placenta.

Thus the increased coagulability of the blood with the tendency to thrombosis is compensated for by these digestive enzymes.

With this greatly increased coagulability of the blood in pregnancy and during the puerperium a sound basis of fact is established on which to explain the occlusion of the cerebral vessels which is the immediate cause of the paralysis. Any disease of the heart or blood vessels would greatly increase this tendency to occlusion but where no evidence of such disease is present, the main factor of increased coagulability must be considered sufficient.

The pathological evidence in cases II, III, IV shew that in case II occlusion was due to an embolus of the left middle cerebral artery, but in III IV the cerebral veins were occluded. The haemorrhages reported are evidently secondary conditions.

The clinical evidence in the other nine cases points to cerebral embolism or cerebral thrombosis.
The slow onset in several of the cases excludes embolism and haemorrhage, while the subsequent history of recovery and long life in many of the cases of sudden onset excludes haemorrhage. Cerebral haemorrhage as an accompaniment of eclampsia will be referred to later.

**Site of Formation of Cerebral Emboli in the Puerperium.**

It is important to consider the site of the formation of cerebral emboli in the puerperium.

Taylor and Osler consider that the left auricle is the site of these emboli, especially the left auricular appendix, but in the healthy active hearts present in the foregoing cases some other more passive site must be sought.

Bearing in mind Kermanner's theory as to the function of the lungs in destroying fibrinogen it is not difficult to see that if this function be defective then there must be a tendency to thrombosis in the pulmonary veins. Thrombi formed in these veins would vary in size and the greater number would undergo resolution but fragments of clot greater or less might easily be swept into the circulation and so into the systemic arteries causing pain in the limbs or paralysis, if the cerebral vessels are obstructed.
The ordinary explanation of 'white leg' or 'white arm' as being due to septic processes is not entirely satisfactory in a great many cases and obstruction to the circulation by emboli from the lung is well worth considering. If the cerebral circulation be obstructed by emboli then paralysis or some arrest of cerebral function results perhaps leading to inhibition of the higher centres with resultant insanity.

In all cases recovery is due to the resolution of the clot by the digestive enzymes of the blood and by the establishment of the collateral circulation. Another source of cerebral emboli has been mentioned by Taylor. He does not admit that in cases of puerperal sepsis thrombi from the pelvis could pass the pulmonary capillaries, but he thinks that in the rare cases of foramen ovale it is possible for thrombi of pelvic origin to pass into the left heart and so into the systematic circulation. Such cases must be very uncommon for according to Carpentier an open foramen ovale usually occurs with other heart affections such as atresia or stenosis of the pulmonary artery, which are readily diagnosable.

None of the foregoing cases belong to this group. Slight patency of the foramen ovale is common enough, but is not diagnosable during life,
and until post mortem evidence is forthcoming of
the association between patency of the foramen
ovale and cerebral embolism in puerperal cases
this explanation must be regarded as an hypothet-
ical possibility.

Cerebral Thrombosis.

Two of the autopsies revealed thrombosis of
the cerebral veins and the history of the cases
shewed a slow onset of paralysis. Cases I and
IX had also slow onset and the condition may be
referred to thrombosis of the cerebral veins.
Primary thrombosis of healthy cerebral arteries
must be regarded as non-existent even in the
puerperal state, but secondary thromboses
following embolism from the lungs must be ad-
mitted and probably many of the cases quoted in
the middle of last century were of the nature
of secondary thrombosis following embolism.
Primary thrombosis of the cerebral veins is only
second in importance to embolism and is much more
dangerous, for once the thrombotic process is
started it is difficult to see why it should be
arrested. Apparently the defensive mechanism of
the blood is often sufficient even to arrest
thrombosis of the cerebral veins.

Angiospasm.

In the more transient cases such as V VI Gillies ruled out embolism and haemorrhage and considered they might be due to spasm of the cerebral vessels. This is the theory of reflex action advocated by the older writers to explain cases otherwise inexplicable. Brown-Sequard fifty years ago attempted to explain some forms of paraplegia by a theory of vascular spasm and he believed that peripheral excitation set up a contraction of the vessels of the pia mater of the cord and so lowered its nutrition. In modern times this theory is strongly advocated by Russell and supported by Brunton and others and it certainly offers a temptingly easy explanation of transient paralysis of central origin. But in cases V and VI there was complete paralyses of the left side for three days and recovery was not complete until a month had passed. The loss of function for so long a period points to something more than a spasm which is an affair of minutes rather than days.

The rational explanation in these transient cases is embolism from the pulmonary veins pro-
bably a loose clot which rapidly resolves under the digestive action of the maternal blood.

**Cerebral Haemorrhage.**

Cerebral haemorrhage as a cause of puerperal paralysis must be rare except in cases of eclampsia when such cases as a rule end fatally. When the stress of labour and the hypertrophied heart of pregnant women is considered, it is to be wondered at that cerebral haemorrhage is not more common. Safety must lie in healthy elastic vessels yielding to the strain.

Kermadner \(^{12}\) and Dienst \(^{13}\) have shewn that in cases of eclampsia, the fibrinogen of the blood is greatly increased and it might be expected that in cases of albuminuria the onset of paralysis would be more frequent than in normal cases, and Galabin \(^{18}\) has stated this risk in addition to eclampsia as following albuminuria. Although only four of the twelve cases given here had a history of albuminuria yet the records are incomplete from this point of view in other three cases. In five it is definitely stated that there was no albuminuria.

The history of the subject (Churchill and Imbert Gourbeyre) shews that albuminuria was often associated with paralysis and although it is not
essential yet if a very large number of cases were thoroughly studied one may safely assert that albuminuria is an important factor both theoretically and practically in the production of puerperal paralysis.


When puerperal sepsis was more rife than it is to-day, it is not difficult to see that small septic emboli might pass from the pelvis through the capillaries of the lung, and set up septic endocarditis.

This would be the puerperal endocarditis of Kirkes and Simpson. Vegetations from the heart valves and endocardium would then be swept into the systemic circulation blocking various arteries and among these the cerebral arteries.

Such cases would be necessarily fatal, and no case of this type has been met with by the writer of this thesis.
A Classification of Puerperal Paralysis of Central Origin based on the foregoing considerations would be as follows:

In order to express the increased coagulability of the blood of pregnancy in one word the term 'Fibrinogenosis' might be used.

Thus

A Primary

I Fibrinogenosis.

   a Cerebral embolism from Pulmonary veins.

   b Cerebral venous thrombosis.

   c Cerebral embolism from pelvic veins via patent foramen ovale.

II Cerebral Haemorrhage - Eclampsia.

B Secondary

I Puerperal Sepsis

   " Cerebral embolism from septic endocarditis.
PROGNOSIS.

The prognosis varies in accordance with the nature of the lesion and its extent.

In simple embolism even when complete hemiplegia and aphasia result the prognosis is good both as regards life and recovery if good nursing is available. Of the 8 cases of embolism, one died, two cases have still some paralysis of extensors of arm and leg, while 5 cases recovered completely from the paralysis of which one is still partially aphasic.

In venous thrombosis the prognosis is more grave both as regards life and recovery. Of the four cases of venous thrombosis two died one has marked deformity and one in which the condition appeared during pregnancy, made a complete recovery after parturition.

Case IX one of the cases of venous thrombosis has had three confinements since the onset of the paralysis without untoward results and Case XI - one of the embolic cases - has had two confinements after her attack without recurrence. These two cases, so far as they go, do not bear out the statement of Edgar 19 that a repetition of pregnancy would likely cause a relapse of the
paralysis.

TREATMENT.

Prophylactic: The increase of fibrinogen and fibrin ferment in the blood during pregnancy being itself a salutary condition nothing should be done to diminish it unless there is evidence of some abnormal increase. Premonitory symptoms of thrombosis, such as severe and persistent pains in the head or the presence of albuminuria point to an excess of fibrinogen. The treatment must be similar to that for preventing eclampsia. Rest to diminish metabolic activity; diet largely free from protein, and fruit, especially fruit juices such as lemon juice with copious draughts of water. Milk is especially valuable. Its value in the prophylactic treatment of eclampsia receives the general assent of the profession, and when it is considered that milk is in itself a product of pregnancy much of its therapeutic value may lie in the possession of protective enzymes which neutralize or eliminate the excess of fibrinogen or fibrin ferment. Should this be so, then milk from a healthy recent parturient cow should prove
the most effectual, as in this milk these protective enzymes would be most abundant. Alkalis such as potassium or ammonium carbonate should also be given with the view of increasing the alkalinity of the blood and so fixing the excess of protein matter in the form of alkali-protein.

Therapeutic: This must be in accordance with the extent of the paralysis. If complete hemiplegia and aphasia result suddenly from embolism then the resulting shock must be combated by strychnine\(^1\) grain hypodermically. Warmth, careful and attentive nursing are essential, as is also a water bed in the worst cases.

Fluid food, such as milk, Benger’s food and gruel to be given with a feeding cup in small quantities and at short intervals. Thrice daily a little alcohol 2-4 drams of whisky or brandy may be given to act as a sedative and to help sleep. The distressing restlessness of the non-paralyzed side must be met by the soothing attentions of a good nurse and a quiet room.

Special attention must be paid to the paralyzed limbs to see that they are in good position, and if swelling occur in them they must be swathed in a layer of gamgee tissue.
The bowels should be moved with gentle purgatives, and if these are ineffective then simple enemata should be used. On no account should strong purgatives be given.

At first there may be complete loss of sphincter control, but as improvement takes place there may be retention of urine requiring the use of the catheter.

Daily watch should be kept for any movement of the limbs, and if no movement takes place at the end of a fortnight gentle massage may be applied night and morning for a period of five or ten minutes at a time. Whenever voluntary movement appears this movement should be practised as far as patient's strength allows.

As improvement goes on eggs and minced chicken may be added to the dietary, and if the patient is anaemic then peptonate of iron and arsenic may be administered with advantage.

As soon as patient's strength allows her she must be helped out of bed and encouraged to walk a little.

Aphasia is a distressing condition but great patience must be exercised and no pressure but on the patient to exercise such powers of speech as remain until her general strength has improved.
The aphasia is generally of the motor type, but in one of the cases Case VII there was a considerable amount of word deafness and word blindness together with loss of articulate speech. Bearing in mind the diaschisis theory of Von Monakow which lays stress on the secondary effects of a cerebral lesion which produces aphasia. These secondary effects proceed from a functional shock like condition of uninjured areas dynamically connected with area of the initial lesion. Efforts of will to revive the memories of these temporarily damaged areas inhibits the power to express these memories either in speech or writing. The indication of this theory for the treatment of aphasia is to allow the patient to practise writing and speaking in a natural and unforced manner.

After the patient has been up and out in a bath chair or carriage Liq Strychninae in 5 minim doses thrice daily before food greatly improves the general tone and stimulates the nervous system.

Cases of thrombosis suffer much less from shock than the embolic cases. The treatment is on the same lines and special care must be taken to avoid violent purgation.
The nutrition of any group of muscles which seem to be specially affected with paralysis should be kept up by electricity and massage.

The Serum treatment of morbid coagulability of the blood in pregnancy and the puerperium occurs to the mind especially when horse serum has been given empirically with benefit in many conditions. The serum from healthy mares advanced in pregnancy or in the puerperal state might be tried in some of these cases with advantage.
Paralyses of Peripheral Origin.

In his study Charpentier divides puerperal paraplegia into 4 classes.

(1) Organic paraplegia with well-defined lesions.
(2) Reflex paraplegia.
(3) Paraplegia from morbid conditions of the blood.
(4) Traumatic paraplegia.

In an examination of the cases quoted by Charpentier to establish his first group no real causal connection can be assigned either to pregnancy or the puerperium. Some of these cases were due to Pott's disease, others to myelitis, which in a few cases may have arisen from puerperal sepsis.

There is no evidence to shew that any lesion of the spinal cord arises directly from pregnancy or the puerperium.

The other three groups were based on cases which were undoubtedly due to lesions of the peripheral nerves or else to some temporary functional loss without evident lesion.

With the exception of Galabin all writers are agreed that paraplegia is much less frequent
than hemiplegia in the puerperium, and even if other forms of peripheral paralysis are included it will be found that paralysis of peripheral origin is less frequent than paralysis of central origin.

I am only able to quote five cases of paralysis of peripheral origin from the literature available, with an authentic reference to a sixth case, but I am able to add two cases which have been met with in my own experience.

**Case I. Handford**

M.K. aged 43. Married 7 years. 3 children.

Has always been a stout florid woman. Her husband keeps a public house and is an habitual drunkard. No evidence of drinking habits in patient.

Three days after confinement, she lost power in her legs completely, and at same time felt sensations of pins and needles in the arms. This was soon followed by paralysis. She had been able to walk up to the day of her confinement. The general character and course was that of an alcoholic multiple neuritis. There was loss of muscular sense impaired cutaneous sensibility, but greatly increased deep sensibility, muscular wasting, and loss of knee jerks and
presence of reaction of degeneration in muscles of arms and legs. Eyes normal.

She was able to walk a little in 6 months and in twelve months was comparatively well.

Case II Handford.²¹

This case was also wife of public house keeper. Age 34. Paralysis of lower limbs came on immediately after confinement. There was complete paralysis of extensors of toes and flexors of ankle with weakness in most other muscles of the leg. The ankle joint was habitually extended and the toes pointed. There was some hyperaesthesia both superficial and deep and some oedematosus swelling. Some weeks later there was considerable muscular wasting extending as high as the right gluteal muscles. Six months later she could walk without a stick and eventually she completely recovered except that the affected muscles remained small.

Case III. Handford²¹

E.S age 31. Married 6 years. 3 children.

Difficulty in walking and weakness in arms came on immediately after her confinement two
years ago. In the house she had to assist herself by holding on to the furniture, and in the street she had to walk along the wall. When admitted to the General Hospital at Nottingham she could not walk. The knee jerks were totally absent as were also the superficial reflexes. There was double ptosis with divergent strabismus and dilated pupils moving neither to light or accommodation - complete paralysis of third nerves. Sight good and discs normal. There is inco-ordination and loss of muscular sense.

She was under observation for five years after the onset of the paralysis and her condition was if anything worse than before. There was no rigidity of the legs. Thighs were well nourished but there was considerable wasting in the muscles below the knee. She could not stand and had to spend her time entirely in bed. She was free from pain.

Case IV. Thomas

Mrs. G. age 26. 1st confinement normal; 2nd confinement membranes ruptured at 7 a.m. Labour pains not strong began at 1 p.m. Between 3 and 4 p.m. she began to have severe pain at back of right leg between knee and hip. At 9 p.m. with aid of chloroform and forceps a large child was
delivered which died soon after.

Patient slept during the night but in the morning complained of pain in right leg which was paralyzed and felt numb.

Puerperium was normal and patient got up on the 10th day and walked with help of a stick. When examined it was found that there was complete paralysis of the muscles supplied by the right external popliteal nerve with weakness of muscles of calf and flexors of knee.

Continuous electrical treatment improved leg but after a year there was still complete paralysis of flexors of ankle.

Dr. Whitridge Williams examined the pelvis and reported it to be normal and there was no inflammatory condition present.

Case V. Thomas 22

Mrs. C. age 25. 1st confinement. Was quite well during pregnancy except some pains at lower part of abdomen and front of thighs. Labour began at 4 a.m. Doctor examined her at 10 a.m. and thought everything satisfactory. During day pains passed off. At 6 p.m. chloroform and forceps were used to complete delivery. Difficult
case, forceps had to be reapplied six or seven times. Patient was unconscious for two hours. After this she complained of pains in her legs below knee. Morphia was given and she slept. Next morning both legs were paralyzed but the weakness affected the left more than the right and the pain was severe and localised below the knee.

Puerperium normal. Up on 10th day but could not walk for three days. After three days improvement began in right leg and progressed favourably. Left leg also improved but not so much as right.

She walks with flapping gait of double foot drop and waddles from side to side.

On both sides it was found by electrical examination that the muscles paralyzed were those supplied by the external popliteal nerves, and the paralysis was complete on the left side and incomplete on the right. Weakness of the glutei gave the waddling gait.

Six months after she was greatly improved by massage and galvanic treatment.

Dr. Whitridge Williams reported pelvis generally contracted. The child weighed 12 1/3 lbs.
Case VI. Manson.

While doing locum tenens for a doctor at North Shields shortly after graduation in 1906 one of the cases attended by the writer was a multipara, age 35, of spare physique, who had had a normal confinement a week previously. She complained of loss of power and numbness and tingling in both legs. There was an absence of knee jerks and a considerable amount of foot drop. The diagnosis was peripheral neuritis and the condition seemed to be getting worse during the period of attendance.

On the doctor's return, the writer enquired as to the possibility of an alcoholic cause but the doctor knew the patient well and would not admit this possibility. He was puzzled about the case. Enquiries were made a year later and it was stated that she was all right.

Case VII.

Mrs. D. age 27. 1st Confinement.

During pregnancy she felt quite well until a day or two before confinement she had some sharp shooting pains on the right side of the face. The labour was difficult requiring chloroform and forceps.
On the day following the confinement she complained at not being able to drink properly. When she drank the fluid ran out of her mouth. It was then noticed that the right side of her face was paralyzed and on the following morning the paralysis was worse and the right eye could not be shut.

Improvement began to shew itself at the end of the week, and in a month the condition was completely better.

Case VIIa

Handford mentions in his article that Gowers quoted the case of a lady who had neuritis of the ulnar nerve with paralysis and wasting twice after child birth.
ETIOLOGY and PATHOLOGY.

In the foregoing cases there is definite evidence of peripheral nerve lesions causing various forms of paralysis, and all are closely associated with pregnancy, parturition or the puerperium without any co-existing disease or intoxication likely to cause these lesions.

A comparison and study of these cases shew that they vary in onset, site and extent, and that they cannot have a common cause.

In Cases IV and V the paralysis came on immediately after parturition and reached its maximum at once. The muscles chiefly affected were those supplied by the external popliteal nerve, although others reaching as high as the glutei were also affected. Improvement began shortly after the patient got out of bed and continued, although even after a year there was still some weakness. Case II presents exactly similar features and all three may be set down to a similar cause.

Thomas describes the labours in his two cases and they were evidently difficult labours due to large size of child and one case had in addition a generally contracted pelvis. Handford
does not describe the nature of the labour in his cases, but the sudden onset after labour combined with a maximum of paralysis which shortly after began to improve, all point to traumatism of the sacral nerves during labour. These two cases are examples of the paraplegie traumatique of Charpentier who quoted several cases from the literature of fifty years ago.

That traumatic paraplegia does not occur oftener in difficult labours is to be wondered at, and there must be some peculiar circumstances in any particular case where it occurs.

Hunermann's conclusion is that it only occurs in a generally contracted pelvis with a large child.

Thomas who has further analyzed its etiology accepts this conclusion and extends the explanation on a basis of Bordeen's analysis of the sacral plexus. Thomas points out that the upper roots of the sacral plexus do not lie on the pyriform muscle but against the bony wall of the pelvis, and are thus exposed to injury from pressure during difficult labour. It is the dorsal offsets of these roots which lie against the wall and which receive the chief injury. The external popliteal nerve is made up chiefly of these dorsal
offsets, and therefore the paralysis is chiefly in the distribution of this nerve.

The traumatic origin of paralysis of this type although recognised long ago by Burns 1839 and other writers, and although all the facts are satisfactorily explained by it, is not fully accepted by Lloyd 23 who believes that septic inflammation from periuterine cellulitis or metritis is the chief cause.

In Cases I, III and VI the paralysis is of wider extent involving the arms and cranial nerves and of slower onset than in three cases already mentioned. Here the question of traumatism cannot arise because of the gradual onset and of the involvement of the arms and third nerve. In all these cases there was absence of knee jerks, foot drop and paraesthesia which together led to the diagnosis of peripheral neuritis.

Cases I and VI made a complete recovery in about a year, but case III where the paralysis was much more extensive did not recover, and remained a helpless cripple.

In Cases I and III the character of the labour and puerperium was not stated but in Case VI the labour and puerperium were normal except for the onset of the paralysis.
The state of the urine is not known in any of the cases, and this is important for Galabin\(^{18}\) states that paraplegia often occurs after the albuminuria of pregnancy.

Charpentier\(^4\) was not able to give very much evidence in favour of the association of paraplegia and albuminuria and states that Imbert-Gourbeyre who favoured the association of albuminuria and hemiplegia did not quote a single case.

Other explanations of puerperal paraplegia without evident lesion of the cord were given by Brown-Sequard and Jacoud. The former believed that peripheral excitation set up a contraction of the vessels of the pia mater of the cord and so lowered its nutrition. The latter proposed a theory of exhaustion of the nerves due to ischaemia or a dyscrasia of the blood.

The onset of facial paralysis in Case VII a few hours after parturition may seem quite an accidental coincidence, but as Charpentier\(^4\) quotes 9 cases of facial paralysis of which 5 were undoubtedly of peripheral origin the coincidence may be more than accidental. In two of the five cases there was albuminuria and the other three were said to have been due to puerperal rheumatism - a condition recognised in the middle of last century.
In Case VIIa there is further evidence of a localised neuritis with paralysis and wasting and this occurred twice after child birth.

These five cases illustrate the occurrence of peripheral neuritis with paralysis in close association with pregnancy and the puerperium.

The neuritis is not of traumatic origin nor is there any evidence of puerperal sepsis or other toxaemia. Handford in discussing his cases raises the question whether the influence of parturition is not simply indirect in allowing other poisons such as alcohol to act on nerve tissue. Bury in discussing the etiology of multiple neuritis argues conversely as to the action of alcohol for he holds with Parkes Weber that alcohol is only indirectly a factor by lowering tissue resistance, and so permitting the successful attack by toxins.

The most powerful peripheral nerve poison known is the toxin of diphtheria and it is possible that in some cases of puerperal paraplegia a mild unrecognised attack of diphtheria might co-exist with the latter months of pregnancy. In other cases some autogenous toxin arising from defective diet or aberration of metabolism during pregnancy may act and destroy
nerve tissue. Until further evidence is available both the etiology and pathology must remain obscure. As a clinical entity the condition of puerperal neuritis must be recognised.

On the Continent Moebius Eulenberg and Winscheidt have directed particular attention to it and the latter has divided puerperal neuritis into four classes:—

I Certain cases which develop during pregnancy and persist after confinement. The clinical picture is that of a pure motor neuritis, gradual weakness of different nerves with atrophy of muscles.

II Neuritis from puerperal infection.

III Neuritis from traumatic injury during labour.

IV Puerperal neuritis proper arising after normal pregnancy and confinement without existing disease.

a Localised form. In arm ulnar or median nerves affected.

In leg crural nerve.

b Generalised form. Many nerves affected often after the manner of Landry's paralysis - an acute ascending neuritis. The cranial nerves may be affected.
PROGNOSIS.

The prognosis is fairly good in all except the more generalised forms of neuritis.

In the traumatic cases recovery goes steadily on but it is likely that some weakness of the limbs may remain and a diminution in the size of the muscles.

In the toxic cases recovery is slow but usually complete in the limited forms of paralysis, while in the more generalised form the paralysis may remain, or gradually grow worse until the muscles of respiration are affected, death then resulting.

TREATMENT.

Prophylactic: In cases of difficult labour due to large size of child and a generally contracted pelvis full anaesthesia and axis-traction forceps should be used to complete delivery with all possible speed. The cause of the toxic cases being yet unknown no directions can be given to prevent these cases. Cases of mild sore throat during pregnancy should be investigated bacteriologically with the view of discovering mild diphtheritic attack.

After the paralysis has appeared then good nourishing food and attentive nursing are essential.
At first gentle massage and later electricity both faradic and galvanic may be used to stimulate the nerves and keep up their nutrition. Strychnine to be given internally. In the limited forms the baby may be suckled, but not in the more generalised cases.

SUMMARY and CONCLUSIONS.

I Apart from existing disease pregnancy and more especially the puerperium give rise to various forms of paralysis.

II These paralyses are best classified as follows:

Paralyses of Central Origin.

A. Primary.

I. Fibrinogenosis.

a. cerebral embolism from pulmonary veins.

b. cerebral venous thrombosis.

c. cerebral embolism from pelvic veins via patent foramen ovale.

II Cerebral Haemorrhage - In eclampsia.

B. Secondary

I. Puerperal Sepsis.

a. Cerebral embolism from septic endocarditis.
Paralyses of Peripheral origin.

I. Traumatic Neuritis from difficult labour.

II. Septic Neuritis from puerperal infection.

III. Toxic Neuritis.
   a Localised form
   b Generalised form.

III. Primary central paralyses of the puerperium most commonly arise from the blocking of one of the cerebral arteries by a fibrinous clot derived from the pulmonary veins.

IV. These clots form because of the inactivity of the lungs in destroying the excess of fibrinogen present in the blood of pregnant women, and especially in those with albuminuria.

V. Thrombosis in the cerebral veins occurs in those cases where there is some deficiency in these protective enzymes which have been found present in the blood of pregnant women.

VI. Albuminuria in pregnancy points to excess of fibrinogen and to a diminution of the protective enzymes and so predisposes to the onset of paralysis.

VII. Cerebral haemorrhage is a rare cause of paralysis in pregnancy and the puerperium, except as a complication of eclampsia.
VIII. Puerperal sepsis may cause paralysis of both central and peripheral origin, but it should be clearly distinguished as a secondary or indirect cause, and not a direct or primary cause inseparable from pregnancy or the puerperium.

IX. Traumatic neuritis from difficult labour causing paralysis must be clearly distinguished from toxic forms of neuritis which appear in the puerperium.

X. There is no evidence that the spinal cord is affected by any of the primary or secondary causes already mentioned.

XI. An important physiological enquiry arising out of these considerations is that of the action of the lung in destroying the excess of fibrinogen in the blood. If so the coagulability of the blood in the pulmonary veins must be less than that in the pulmonary artery. In other words venous blood is more coagulable than arterial blood. It is assumed in the foregoing pages that this is so, but there is need of experimental proof.
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