THESIS.
PROFESSIONAL EXAMINATION FOR
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1906
HAEMORRHAGE into the SPINAL CORD-
complicating
PREGNANCY and LABOUR

The following case—which I had the opportunity of closely observing while House-Physician to Dr Alex. Bruce in the Royal Infirmary Edinburgh, whose permission I have to use the notes I made at the time has many points of interest bearing on various debated subjects. It shows the need of following cases when possible to the postmortem-room, and the necessity of very thorough examination of them there; both as regards obvious morbid appearances and under the microscope to show the exact nature of the lesions and the correct conclusions to draw from them. It also shows the value which can be placed on similar cases, recorded as primary Haematomyelia, but not supported by post-mortem evidence.

From an Obstetrical point of view it also raises some interesting questions, bearing on the nervous mechanism of labour, which I will mention later.
NOTES OF CASE

NAME- Mrs. Mitchell
AGE.- 31
ADDRESS- 177 Canongate, Edinburgh.
OCCUPATION- Hawker.
ADMITTED- Nov. 1, 1903.
COMPLAINT- Paralysis of lower limbs and weakness of right arm.

FAMILY HISTORY- Father died of concussion of the brain. Mother died of uterine Disease.

Has had 7 children. Of these-

4 are alive and healthy.
2 died of pulmonary disease.
1 died from result of accident.

No history of nervous disease in family.

GENERAL SURROUNDINGS- Patient has to be out in all weathers. She has to carry a heavy pack on her back, selling fruit, and often does not go home to her dinner till she has earned it.

PREVIOUS ILLNESSES- Usual children's diseases.

She has always had severe pain in her back during the first three months of her pregnancies.
She considers herself about six months pregnant now, and has suffered from severe vomiting since lost the beginning, and has lost weight in consequence.

PRESENT ILLNESS- Last Thursday (Oct. 21.) at 9 am. patient felt sick and vomited, and while vomiting she felt something "twist round and then tear and give way"
on the right side at the level of her waist. She fell to the floor, having intense pain at intervals, and feeling very "dizzy." Her arms and legs shook spasmodically, and then she found that she was unable to move either of her legs, and that her right arm was also partially paralysed.

Dr Hardie, of the Cowgate Dispensary, was sent for and gave her a hypodermic injection of morphia to relieve the pain—chiefly complained of in the arms. The paralysis of the right arm lasted during that night. Next day (Nov. 1) the patient states she had pain all over the body and especially between the shoulders and was very sick all day.

On ADMISSION to Ward 27 the same evening I found her in a very collapsed condition, retching continually and perspiring profusely.

ON EXAMINATION I found that there was complete anaesthesia of the skin of both legs and trunk as a line drawn from a point 2½ inches above the tip of the Xyphisternum, and extending through the lower borders of the areolae of the nipples to the 7th Dorsal spine behind. Her right hand and arm were still weaker than the left, but patient states that she is left-handed. Pupils rather small. Patient drowsy from the effects of the morphia. Uterus 1½ inches below Umbilicus. No foetal heart sounds audible.

ALIMENTARY SYSTEM—Patient has suffered from sickness all through the present pregnancy. Her bowels have
Area of anaesthesia, in black.
VASOMOTOR and NUTRITIVE FUNCTIONS - There is oedema of both legs and trunk, especially in the interscapular region. Perspiration is profuse, and the face is flushed.

LOCOMOTOR SYSTEM - There is flaccid paralysis of the muscles in the anaesthetic area, and tenderness on percussion over 6 and 7 Dorsal Spines.

PROGRESS of CASE.

NOV. 5. - Patient spent a restless night, was sick several times. Urine drawn off at 6am. Patient drowsy, pupils normal. She complains of slight tingling in the right arm but the power is good. There is stiffness and pain in the back and sides of the neck, and between the scapulae. Left knee jerk - present. Right " " " absent.

Plantar reflexes both visible on strong stimulation.

Tablespoonful of Castor-oil given.

NOV. 4. Bowels moved unconsciously. The upper limit of anaesthesia seems to have descended, but patient is very lethargic and not certain in her answers. She is very much troubled with hiccupping and the distension of the abdomen with flatus.

NOV. 5. - There is still retention of urine. The knee jerks are both absent. Plantar reflex very slight. Patient complains of feeling cold. The skin is cold and clammy and covered with a profuse perspiration. There is a motor restlessness of the hands and arms. She is twitching of the tendons on the back of the arms. She complains of an itching of the skin.
and a feeling as of electricity running her arms.

Breathing is easier when patient is propped up in bed.

Nov.7—patient feels better to-day, and is more comfortable. Perspiration still very profuse. Retching continues. There is still tingling in right arm and hand. Knee reflexes cannot be elicited.

Nov.8—The area of anaesthesia seems lower down than before; a bedsore has developed over the sacrum, so patient has been turned on her side. Bowels have not moved for two days.

Without entering into further details of daily progress, I may state that up to the end of November, patient in some ways improved; both knee jerks re-appearing on Nov.13, and once she stated she had a feeling as of electricity in her legs. Her hand-grip became more powerful. Unfortunately the bedsore over the sacrum increased to an alarming extent, and another developed over her great trocanter on the left side, while yet another—apparently independent of pressure—appeared in the perineum outside the right labium major. On Dec.1 patient began to suffer from Bronchitis and had great difficulty in getting up the expectoration. A tent and steam kettle were rigged up and this, along with a mixture containing Digitalis and Ammon. Carb., gave some relief. The Temperature rose to 106 Degs. and she became very distressed and breathless. This condition continued for a fortnight; patient
gradually losing ground, and giving up hope.

At 4am on Dec. 14, I was rung up by the night nurse, who explained that when she had occasion to change the patient's sheets, she noticed a gush of water coming from the vagina. On examination I found a foetus born except for the head which was in the vagina. Patient had not complained of pain, and was not aware that she was in labour until informed of the fact. Not having had experience of such a case before, I injected Ergot hypodermically into the buttock, and got hot douches ready for emergencies, before doing anything else. I then extracted the foetus, which was dead; and on examining further, I found that there was another presenting. At this time the Uterus could be felt through the abdominal walls, small and firmly contracted in an empty condition. The second foetus was expelled by supra-pubic pressure; the placenta came with it, the membranes being intact. There was to my relief little or no haemorrhage. The patient felt nothing, and did not assist labour by any expulsive efforts.

The parts were thoroughly cleansed with Corrosive Sublimate Lotion, and a douche of the same given, the parts being well powdered with Boracic Acid. A foul sloughing sore encroached on the right Labium, so that it seemed impossible to prevent puerperal septic infection. Both foetuses were dead, but were apparently healthy, and between the 6th and 7th month.

Next day (Dec. 15) Patient seemed relieved by the emptying of her Uterus.
DEC. 20.—Bed-sores rapidly going from bad to worse, in spite of every care. Patient can now only gain comparative comfort, by sitting up in bed, leaning forward and supporting herself on her elbows. This has caused bed-sores to develop on both elbows, and sores are threatening to form also on the heels.

FURTHER PROGRESS—Patient lingered on getting weaker and delirious. The bed-sores increased in depth and size, and there were frequent distressing attacks of breathlessness. She collapsed while being cleaned up and died on JAN. 6 at 1 pm. nine weeks after the onset of the paralysis.

Before relating the condition found at the post-mortem, let me discuss the diagnosis.

The salient features of the case seemed to be - Sudden onset, during vomiting, of paralysis of sensation and motion below a well defined line, in a woman, who, although pregnant, and suffering from excessive vomiting, had apparently been previously quite healthy; at least neither the patient nor her friends had noticed anything which would lead us to suspect any existing lesion of the central nervous system.

THE DIFFERENTIAL DIAGNOSIS then seemed to be between

(1) Fulminating haemorrhagic Myelitis.
(2) Extra-Medullary Haemorrhage.
(3) Haematomyelitis.

(1) Fulminating haemorrhagic Myelitis—ie-Myelitis coming on so suddenly as to suggest Haemorrhage into the Spinal Cord; indeed in some cases there is haemorrhage,
but it is the result of the inflammation, not the cause. Gowers in "Diseases of the Nervous System" page 259 states that the onset may occupy only a few minutes. "The legs are found suddenly to be heavy and tingling; the sufferer sits down in a chair for a quarter of an hour, and then finds he cannot stand. Such rapid onset resembles that of Spinal Haemorrhage; and it is probable that in most of these cases there is Haemorrhage in addition to inflammation. Such cases are however sufficiently rare not to interfere with the diagnostic rule that a sudden onset means a vascular lesion." In a footnote he adds "Indeed it is possible that these cases constitute no exception to the rule, the Myelitis being set up by vascular obstruction." The question is a difficult one to settle, because unless death occurs at once (and this is rare) changes ensue in the part, which mask the real state of affairs. "Naked eye examination of the Cord post-mortem is of no value, because so much blood may be effused into the tissue, that it looks as if there had been an actual haemorrhage.

(2) Extra-Medullary Haemorrhage. This is a rare affection, except as the result of an injury. If the Haemorrhage is large and sudden it is practically impossible to distinguish it from Haemorrhage into the substance of the Cord. The distinctive features are those of meningeal irritation—severe pain in the back and general convulsive movements of the limbs. These appreciably precede the onset of paralysis."
Primary Haemorrhage into the Spinal Cord or Haematomyelia.

This disease is characterised by a sudden onset of paralysis without any premonitory symptoms. Gowers says "We are not justified in regarding as primary which Haemorrhage, any case in which premonitory symptoms existed for more than a few minutes; unless such symptoms were so pronounced and sudden in onset, that they might have been due to a definite extravisation, afterwards increasing." Pain is not an important symptom, but is usually present. There may be tenderness on pressure over the affected spot.

The case I have described was therefore provisionally diagnosed as Haemorrhage into the Spinal cord, apparently primary, but as this was known to be a very rare affection, the post-mortem was looked forward to with great interest. But here a very common difficulty presented itself. The deceased's friends, who were Roman Catholics, refused absolutely to grant permission for a post-mortem. Even the advice and threats of the Priest failed to move them. So much so that the possibility of foul play suggested itself. This idea was strengthened by the fact that the patient, who was a hawker, and lived in a low part of the Canongate, was illegitimately pregnant, and had no history of Spinal disease, before the onset of the paralysis. The case was therefore reported to the Fiscal, who ordered a post-mortem examination, with the result which I will state
It has been (and is) a much debated point as to whether such a condition as Primary Haematomyelgia really exists, and no doubt many case like this one, have been observed and recorded as such without having been fully investigated post mortem. Cowers says "it is more rare than is suggested by cases recorded as such". Strictly speaking, Primary Haematomyelgia means Haemorrhage into the tissues of the Spinal Cord which were previously healthy. This definition ought not, I think, to include cases which seem to be the direct result of a traumatism, and in discussing recorded case later, I will not refer to such; nor do I think that cases caused by sudden and severe alteration in atmospheric pressure, should be included under this heading.

Since the beginning of last century authorities have written and differed on this subject; and here I would like to point out that, considering the comparatively recent development of exact pathological methods—especially where the microscopical examination of the central nervous system is concerned—those cases with autopsies, recorded in the early part of last century, cannot be accepted as authoritatively proving anything. As early as 1808 Cautier de Claubry published a case of Spinal Haemorrhage, apparently spontaneous. Hayem, in "Des Hemorrhagies intra-Rachidiennes" criticizing this case says it was evidently one of inflammatory softening with secondary haemorrhage. In 1827 Ollivier D'Angier used the name Haematomyelia and next
year, Hutin described a case of Haematomyelia in a healthy subject, with a post-mortem.

Koster in 1869, in "De pathogenie der apoplexia medullae spinalis", reviews all the cases he could find up to that date, and gave it as his opinion that, in all of them, the haemorrhage was secondary to the inflammatory condition. The question as to whether miliary aneurisms are found in the cord, is a debated subject. Liouville described a case in 1872, where he observed miliary aneurisms. Gowers states shortly that "miliary aneurisms are not found in the Spinal Cord".

Hayem, in "des Hemorrhagies intra-rachidiennes", dwells on Liouville's case, and though he allows that he saw the aneurisms in question, he looks upon it as an unique case, and a coincidence, the haemorrhage being, he is certain, secondary to a myelitis which was present.

Hayem's paper was written to prove that Primary Haematomyelia did not exist; he explains all the cases he could collect, rather monotonously, I think, as being secondary to myelitis, but Liouville's case, even although it was a genuine case of Haematomyelia, is a solitary example of its kind, and is therefore of very little clinical value.

The most modern and comprehensive work on Haematomyelia is that of Lépine, published in 1890. He recognises primary haemorrhage into the Spinal Cord, and divides Haematomyelia into primary, and secondary. He says secondary Haematomyelia may be due to

(1) Acute myelitis.
(2) Chronic myelitis.
(3) Syringomyelia.

(4) Tumours.

He divides primary haemorrhages into those caused by

(1) Traumatism.

(2) Sudden and severe alterations in atmospheric pressure.

(3) Spontaneous.

There is no doubt at all that primary haemorrhages occur under sections (1) and (2), but as the question of spontaneous haemorrhages is a more debateable point I will dwell on it, and follow out a certain number of the cases Lépine quotes; only giving serious consideration to those, in which, the result of the autopsy is fully and clearly given.

Under the heading of cases caused by vascular congestion of the Cord, he quotes cases of sudden paralysis coming on after lifting heavy weights, or in men who were working in constrained positions; but these cases are not supported by post-mortem evidence, and of course the exertion may only have been the exiting cause in a Cord already diseased. (2) Straining at stool—this might quite well have been included under the heading of vascular congestion. It is supported by a case recorded by page in the Lancet, 1850, p445, of a child who died suddenly while straining at stool. But this child had had symptoms pointing to disease of the Spinal Cord previously, and the post-mortem report does not give any explanation of these symptoms, — pain referred to the spine,— and the child was ill for four days before
days before the onset of the paralysis. Altogether it is not a satisfactory case to use as an argument for spontaneous Haematomyelia.

(3) Whooping Cough, also causing, I should say, vascular congestion. Lépine cites three cases, but they are not accompanied by post-mortem evidence.

(4) Convulsions, as in (a) Epilepsy.

(b) Strychnine poisoning.

(c) Tetanus.

I have carefully investigated the cases recorded under these headings, and I not convinced that any of them deserve to be included under the heading of Spontaneous Haematomyelia. One case is recorded by Wille in Arch. f. Psychiatria Ed. 31. 1898. p535. of an alcoholic, who died shortly after an epileptic fit. The author explains that the haemorrhage in this case was due to forcible flexion of the head on the neck during an attack, when congestion of the cerebral and spinal vessels predisposed to rupture. This case then should be said to be due to traumatism, rather than be called spontaneous. So might the cases of Strychnine poisoning, but none of them have pathological evidence in support of there title.

The case of Tetanus which Lépine quotes, showed a latent Gloma which sufficiently explained the haemorrhage as being secondary.

(5) Arrest of Menstruation. Two cases are recorded under this heading, but here, again, I think neither of them, on close examination, are satisfactory examples of Spontaneous Haematomyelia. The first, recorded by Levier
in his Thèse de Berne, 1864, is of a patient who was ill for six days with violent pains in the lumbar and hypogastric regions, and I do not think that it is proved that the haemorrhage was not secondary to a myelitis. The second case is recorded by Eichhorst in 1874, who is very positive that the haemorrhage was a "capillary apoplexy", the blood vessels showing sacciform and fusiform dilations, the rupture of which was the source of the extravasated blood. He mentions specially that the surrounding tissues do not show signs of inflammation. The only criticism I would venture to suggest in this case is, that the onset was not sufficiently sudden as to suggest haemorrhage, and the case might quite well have been explained clinically as one of acute Myelitis. The author allows that the Cord, on first examination, seemed to him to be a case of acute Myelitis, but his opinion is that the microscope proved the case to be "apoplexie capillaire primitive".

It seems to me that the suppression of the menses in these two cases was just as likely to be the result and not the predisposing cause of the illness.

(6) Severe cold.-

Haemorrhage into the Spinal Cord has been produced experimentally in animals, by placing liquid carbon-dioxide on the spine for twenty minutes, but I do not think that this proves anything of clinical importance. There is a case recorded by Boinet "Hemorragie Primitive de la Moelle" 1891; Lépine says of this case, that
the section showed a true Haematomyelia, without a trace of myelitis. The case was one of a man, aged 53, who remained bathing in the sea for two hours. It was in July, in France, and the water cannot have been very cold. There were distinct premonitory symptoms,—tingling and pain in the limbs,—and he managed to walk home feeling some stiffness in his ankles. It was during the night following that he became paralysed, and there was considerable constitutional disturbance. If we apply the rule laid down by Gowers, which I have already quoted, concerning the onset of symptoms in Haematomyelia, to this case, we find that it cannot satisfactorily be included in the category of true primary Haematomyelias.

(7) In haemorrhagic Diseases, such as Pernicious Anaemia, Haemophylia, Purpura, haemorrhages may take place in the Spinal Cord as much as in any other part of the body. They are usually small, and do not often cause symptoms during life.

(8) Vascular lesions; As I have already said, it is very doubtful if miliary aneurisms are ever found in the Spinal Cord. I have quoted the case recorded by Louville, but in spite of it Gowers says that miliary aneurisms are never found in the cord. The reason of this I will give later. Whether vascular degeneration in syphilis and alcoholism can cause by itself haemorrhage into the Cord is, I think, open to doubt; but no one will deny that such vascular disease predisposes to haemorrhage under favorable conditions.
Before leaving this subject it is instructive to note that minute hemorrhages are often found in the Cord, after from diseases which interfere with respiration or cause venous congestion; and they are specially frequent in maladies, which at the same time cause functional excitement of the Cord, such as Tetanus and all severe convulsions. They cause no symptoms and are probably produced during the last moments of life. They have been termed accessory.

It is interesting to note, in connection with the foregoing remarks, that Lépine has collected 126 cases of Haematomyelia followed by post-mortems. Of these, only 45 are claimed to be spontaneous. Of these 45 he says that 10 show vascular alteration.

9 " Haemorrhagic disease.
10 " congestion from effort.
while 16 are without apparent cause.

From the review I have made of many of these cases, it will be seen that no great value can be placed on such statistics.

It would be ridiculous, with the amount of experience and literature at my disposal, to assert that Spontaneous Haematomyelia did not exist; but I have said enough to show that at least it is very rare, and more so than is apparent from a superficial acceptance of the records of some writers.

The rarity of its occurrence is accounted for by the following facts,—The main arterial supply to the Cord
is from the spinal branches of the Vertebral arteries. These, in comparison to their great length, are very slender vessels, and since the blood flow meets with considerable mechanical obstruction, high pressure — which is the cause of degeneration and rupture of the cerebral arteries—is prevented.

What was the result then, of the post-mortem examination in the case I had the opportunity of observing? — a case in which the onset of paralysis was sudden, without any premonitory symptoms, in a comparatively young and healthy woman. Surely a case in which to expect that one would find a true primary haemorrhage, and be able to add another to the lengthening list of cases of Spontaneous Haematomyelia.

POST-MORTEM REPORT.

This was limited to the spinal cord, and revealed a high degree of thrombosis, and dilatation of the vessels in the pia-mater on the posterior aspect of the cord in the dorsal region. Through the whole of the dorsal region the cord was of a soft consistence, and somewhat darker in colour than normally. At one or two points in the upper dorsal and lower cervical regions what appeared to be a small extravasation of blood projected beneath the pia in the posterior columns.

ON section of the cord, by Dr Alex. Bruce, after it had been hardened with 10% Formalin, a tubular haemorrhage was found throughout a great length of the Cord, extending from the 6th cervical segment to the 1st Lumbar, but presenting very different features at different
levels. In the mid-dorsal region the haemorrhage occupied nearly the whole of the interior of the cord, being covered merely by a thin layer of white matter. The haemorrhage was of a rusty colour in parts. In the lower half of the dorsal region the haemorrhage showed in its interior a peculiar tubular structure of small size, capable of admitting a fine pin. This structure was more or less circular in outline, had pale, somewhat thick walls, and it appeared to contain blood. This tubular structure persisted to the lower end of the dorsal region; but as successive sections were made from above downwards, the surrounding haemorrhage gradually diminished, and disappeared at a higher level than this tubular structure itself. In the cervical and upper dorsal regions the haemorrhage became somewhat rapidly lessened in size and limited entirely to the grey matter. In the upper dorsal and eighth cervical segments it occupied the anterior and posterior horns and the central grey matter, having somewhat the outline of a capital H. At the level of the seventh cervical the haemorrhage became limited to the posterior cornua alone. At the fifth cervical segment on the posterior aspect of the left side of the cord, a small tumour-like mass was found, which extended from the pia mater inwards, nearly to the central canal. It was independent of the haemorrhage above described, was very vascular, and of a yellowish appearance in the centre.

On making microscopic sections at various levels the haemorrhagic nature of the lesion was quite apparent.
In the mid-dorsal region the tissue of the cord was almost completely, especially in its anterior portion, torn up by the haemorrhage. The vessels which persisted were engorged with blood, and surrounded with numerous round cells. In the lower dorsal region the fine tubular structure already referred to became evident in the midst of the haemorrhage. It was situated behind the central canal, for the most part in the anterior portion of the posterior column, partly in the mesial plane, partly somewhat to one side. The tube had walls of condensed neuroglial tissue, which were fairly vascular, being occupied by numerous small capillaries. An extensive series of sections failed to reveal any endothelial lining. At its upper extremity the tube became converted into a solid cord. It gradually passed forwards from the posterior column into one posterior cornu somewhat to one side of the mesial plane, and could be traced upwards until it came into contact with the neuroglia of the wall of the central canal. The lower end of the tube was also closed, but terminated in the posterior columns, and had no nearer relation to the central canal. The source of the haemorrhage into this tube was found in a rupture of its wall, and a communication with the haemorrhagic area outside. A careful examination seemed to indicate that the haemorrhage had arisen outside this tube, and had burst through its wall. In addition to this structure there was found in the mid-dorsal region two small angio-gliomatous masses, partly connected.
with the pia mater, and partly involving the white matter underneath. The vessels of this tumour were much dilated, and had very thin walls. Further, there scattered throughout the, in parts near to, but not destroyed by, the haemorrhage, several small areas in which the vessels were massed in close proximity to each other, but with very little other tissue between. These were evidently minute angio-gliomata. Similar masses also appeared to be situated in the midst of the haemorrhage, where this had not so completely torn up the tissue. The source of the large tubular haemorrhage appears to have been one or more of these very dilated vessels. The tumour in the fifth cervical region of the cord had a similar structure, but with much larger vessels, apparently dilated veins as well as dilated capillaries. This tumour had apparently begun to degenerate in its centre, but no haemorrhage had taken place into it. In the cerebellum there was a small angio-gliomatous tumour similar to that just described, and situated in the left lateral lobe.

The above post mortem report is Dr. Alex. Bruce's description of what he found on making a careful examination of the Cord; he is of the opinion that the tubular structure, above referred to, anti-dated the haemorrhage and was due to a malformation of the central canal. Such tubular structures are frequently found in spinal haemorrhage, and they probably point to imperfect vascular development being the cause of the haemorrhage.
The following diagrams are copies of photographs of sections of the spinal cord at different levels, made by Dr Alex. Bruce.

Fig. (1) shows an angio-glioma from the posterior column of the 5th cervical segment. The clear spaces are dilated vessels, with very thin walls, the fragile nature of which, was the primary cause of the haemorrhage.

Fig. (1)

Fig. (2) shows a section of the 4th Dorsal segment. The oval laminated area to the left of the middle line is the upper end of the tubular structure, found in the lower dorsal region. The vessels are shown filled with thrombi in the substance and membranes of the cord. The tissue of the cord is destroyed by the haemorrhage and undergoing a process of softening.

Fig. (2)
Fig. (3) shows a section of the 6th Dorsal segment. It shows the left anterior cornua torn up by the haemorrhage and the tubular structure, its walls torn, and filled filled with blood.

Fig. (4) represents a section of the 10th Dorsal segment, showing the dilated tubular structure, with its thick laminated walls. It is situated in the anterior part of the posterior columns, immediately behind the central canal of the cord. The walls are of condensed neuro-glial tissue, and there is an absence of any endothelial lining.
Fig. (5) shows two angio-gliomata in the membranes of the cord. The tubular structure is again seen, the blood has been washed out of it in the process.

Fig. (6) is a representation of a section of the 1st Lumbar segment. The closed lower end of the tubular structure is seen in the left posterior column, in its anterior part. It is evidently of long standing and is not the product of the haemorrhage.
This report shows that what was apparently a primary haemorrhage, was really one secondary to a glioma with fragile vessels, the giving way of which, during an act of vomiting, caused the sudden paralysis. It is probable that this case, had it been examined by an inexperienced observer, would have been classed as primary, and would have swelled the ranks of primary cases, supported by post mortem evidence.

The state of the reflexes in this case is interesting. Usually when the cord is completely destroyed above the Lumbar enlargement—although there may be at first an initial inhibitory loss of reflex action, the reflexes return in the course of a few hours; and subsequently the action becomes excessive.

Dr Charles Biaston avers, that if the cord is completely destroyed, the reflexes are completely abolished.

In this case, the right knee jerk was present the day after the onset of the paralysis; the other was recorded first on the fourth day. As the haemorrhage reached as far down as the first Lumbar segment, its proximity to the segments concerned in the production of the knee reflex (2&3 Lumbar) explains the irregular results.

Their condition was so irregular, that I am unable to draw any conclusions from observing them. There was certainly no exaggeration of them as one would have expected with the cord destroyed above the Lumbar enlargement; but probably secondary myelitis, which would develope in the neighbourhood of the haemorrhage, would
account for this. The centres for the vesical reflex, being low down in the Lumbar enlargement, (4 & 5 sacral) escaped, and the usual phenomena were observed, namely, retention at first, followed by reflex overflow.

There was very obstinate constipation, but when feces, or enemata reached the lower bowel, they were expelled reflexly and unconsciously. The formation of bed sores was very characteristic of a serious transverse lesion of the cord. They were of the acute trophic variety; a gangrenous condition of the skin following pressure on the boney points, and one very deep one on the inner side of the thigh, away from pressure, no doubt due to escaping urine and feces. As to the causation of these acute bedsores, evidently pressure is a secondary consideration. Charcot thinks that they are not due to "absence of nerve action" but to spinal irritation. Experimentally in animals, the development of gangrenous ulcerations over the sacrum do not supervene on ordinary sections of the cord, but only in cases where inflammation has been set up in the neighbourhood of the spinal lesion. There are said to most frequent where the lesion occupies the central regions of the cord, and this seems to designate the grey substance as playing a predominant part in this respect, not the grey matter of the anterior cornua - for in infantile paralysis acute bedsores are rare - but the grey substance surrounding the central canal.

These acute bedsores, if death does not ensue from other causes, sometimes are the source of septic pulmonary
Rough sketch, from life, showing the various bedsores described in the notes of the case.
emboli, or cause general pyemia.

And now if we look at the case from an obstetrical point of view, several points of interest arise. It is evident from this case, and from several others recorded, that labour can take place and be successfully ended without cerebral control or consciousness. It is evidently a reflex act, the centre being probably in the Lumbar enlargement. And yet it is quite certain that the progress of a normal labour is partially under the control of, or at least influenced by, cerebral centres. It is a matter of every day observation, that emotion may entirely arrest uterine contractions. A woman in labour has the power, to a certain but very limited extent, to hasten and augment the uterine contractions. How often the arrival of the doctor causes the pains to disappear, and become feeble; and how certainly they will recur with redoubled violence when he leaves the house. Experiments have shown, that the uterus, like the heart, can contract automatically when removed from the body, if kept warm; and it is probable that the uterus is able automatically to expel its contents as far as the genital canal, even when deprived of spinal influence. Sir J. Y. Sympton performed several interesting experiments on sows to prove this. He removed, or destroyed the Dorsal and Lumbar Cord a day or two before labour was due. In some, labour progressed regularly, in each case all the foetuses were born except the last one. That is to say, each except the last, was expelled from the uterus into the vagina automatically, and each
in turn was forced from the vagina by the one following it. The last one remained as there was no abdominal reflex working. Sir J. Y. Simpson says "though uterine contractions are no doubt guided by brain and Spinal Cord, they are essentially independent of these organs. The nerve power probably comes through the ganglionic system of nerves. For, he adds, although the uterus will contract out of the body, we do not believe it would contract regularly unless governed by some nervous supply." In the case I have described, what particularly struck me was, that although the first foetus was not fully born when I arrived, the uterus was felt firmly contracted and quite empty, by palpation through the abdominal wall. The patient was absolutely unconscious of what was going on—suffering no pain—and in no way aiding labour by expulsive efforts. I do not think that in this case there was any evidence of any reflex expulsion of the foetuses after they had been ejected from the uterus. Had there been any reflex action, they could easily have been expelled, as they were small and soft. The first of the twins was no doubt pushed out and partially born, by the pressure of the uterus, transmitted through the second foetus, as in Sir J. Y. Simpson's experiments. Several cases have been published of labour occurring in paraplegic women. Dr. Routh, in the London Obstetrical Transactions, 1897, publishes the case of a woman, seven months pregnant, who, through an accident was paralysed below the sixth Dorsal segment. She went on to full term, and was delivered successfully
The first stage lasted 10 hours, the second, $2\frac{1}{2}$, the third 5 hours. The patient felt a tight feeling at the epi-
gastrium during the "pains", and as the head passed the
vulva, the patient cried out; but it seemed to be a reflex
act, there was no pain. He quotes six other cases, and
concludes from them that, if the lesion destroys the re-
flex centre, the uterus may automatically expel its
contents - a certain distance at least - but it cannot
complete the labour. If the reflex centre is intact,
the labour progresses through the second stage reflex-
ly, and without voluntary assistance.

One subject remains a mystery, although we may be quite
certain as to the mechanism of labour once it is start-
ed, we are yet entirely ignorant as to the exit ing
cause, and why it should take place at a definite date.
It can be explained by some substances gradually accum-
ulating in the maternal blood as the foetus approaches
and arrives at maturity. These substances, of the nature
of chemical irritants, by means of the blood, act on the
motor centres of the uterus, and bring it into a state
of activity. Spiegelberg asserts that there is a motor
centre for the uterus in the Medulla Oblongata, very
near the vaso-motor centre, having connection with the
uterus by means of the Spinal Cord and sacral nerves,
but chiefly through the aortic plexus and lumbar sym-
pathetic ganglia and nerves. Once set going, the act-
ivity of the uterus is maintained, by the ganglia and
nerves in its walls. It is thought that the normal seq-
ueence of movements is due to the anatomical arrange-
ment of the fibres of the Uterus, and not to special nervous co-ordination.

An interesting discussion on the occurrence of "painless labour" has been appearing in the last few numbers of the British Medical Journal. The origin of it was a statement by Dr Peter Horrocks, in an address on the "Midwifery of the present day". In it, after dwelling on the reflex nature of the act of parturition, he emphasizes the fact that, while labour can be helped or hindered by voluntary muscular efforts on the part of the woman, such help is not absolutely essential, as is proved by delivery being effected in cases in which there is complete severance of the will power through a lesion of the Spinal Cord. On the other hand, he says, hindrance is limited for no woman could prevent delivery taking place at term by mere voluntary efforts.

But it was his remark that "there is no such thing as a painless labour" (in a normal case) that prompted several Practitioners to write giving their experiences of apparently healthy women, who had had perfectly painless labours. No attempt is made to explain these cases, and no detailed account of any examination of the nervous system in any of the cases is given.

One fact I have not drawn attention to, and that is the exciting cause of the Haemorrhage, in the I have described, ie, the excessive vomiting. This vomiting which was severe all through the pregnancy, did not cease after the onset of the paralysis, even though all sensation and reflex action were abolished. The exact cause of this
vomiting is unknown, but many writers speak of it as a reflex act, caused by displacements or abnormal conditions of the uterus. Now in this case, unless the nervous mechanism concerned was totally supplied by the sympathetic system, the vomiting cannot have been due to the impressions conveyed to the vomiting centre through the medium of the Spinal Cord. I have not been able to find any case recorded, where this point was noticed or explained. Of course there may be a fallacy in this, brought about by the fact that no doubt the constant vomiting, which the patient had before the onset of the paralysis, must have caused a gastric catarrh, with great irritability of the mucous membrane, and the subsequent vomiting may have been due to this.