Epilepsy and the Status Epilepticus in Connexion with Pregnancy and Labour, with Illustrative Cases.*

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In *most* works on midwifery the subject of epilepsy is discussed in a few sentences of general remarks. Again, in some books on epilepsy, the effect of pregnancy and labour on the condition is hardly mentioned. It is thus difficult to gain information on the matter.

In his recently issued book on epilepsy, Dr. W. Aldren Turner gives his experience of the influence of pregnancy, the puerperium, and lactation in 41 epileptic women, with a history of 61 pregnancies. He tabulates the results in the following way:

Quickening induced a relapse in	7	cases.
Pregnancy was the original cause in	2	„
„ induced relapse in	14	„
„ was temporarily beneficial in	6	„
„ made no difference in.....	1	„
Accouchement was the original cause in	5	„
„ induced a relapse in.....	17	„
Lactation was the original cause in	3	„
„ induced a relapse in	6	„
Total pregnancies	61	

41 cases. Of 25 of these cases the family or other history bearing upon heredity was studied, and 13 cases, or 52 per cent., gave a history of family epilepsy or alcoholism, a percentage which corresponds with that ascertained as the relative proportion of a family predisposition amongst epileptics in general.

In the two cases where *pregnancy* was given as the original cause, the women were both young and pregnant for the first time. In one of the cases where there was freedom from attack in one pregnancy, the fits become more frequent in a 2nd pregnancy. He says: "There are undoubted cases on record in which fits have been permanently arrested by pregnancy, and others in which a temporary remission has been observed; but it will be seen from the figures here given,

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that it is more common to find a relapse of the attacks, or the conversion of a minor type of the disease unto the combined major and minor type. Nerrlinger's figures on this subject show that of 92 women with 157 pregnancies, 28 per cent. showed complete cessation of the fits during pregnancy, and 35 per cent were made distinctly worse."

In regard to *accouchement* and the *puerperium*, he says: "There were 5 cases in which the disease clearly originated at this time, and 17 in which it led to a serious relapse. Of the first series, the onset was in the form of serial epilepsy, or the status epilepticus (puerperal eclampsia), and the disease continued in a chronic form for many years afterwards. In one case it commenced during the fourth confinement, and in three others during the first. These cases are particularly interesting, as they argue strongly in support of the view of Féré, that puerperal eclampsia, like many other 'eclampsias,' is merely epilepsy in an acute form, and that the disease, once started in this way, may persist for years. Two cases were illustrative of this, by the fact that after the original eclamptic attack the further continuance of the malady was in the form of minor seizures over a period of eighteen and ten years respectively.

"Of the second series—those cases in which a relapse was caused by confinement—there were two, in which a remission of 12 and 18 months respectively, was broken by the eclamptic seizures of the puerperium. In the others they merely formed an incident in the course of the confirmed disease.

"It was not uncommon, in cases of already existing epilepsy, for puerperal convulsions to be delayed until the later pregnancies.

"The incidence of serial epilepsy, at or immediately succeeding parturition, is therefore a common feature in epileptic women, and raises the question as to the diagnosis of some forms of puerperal eclampsia. A history of pre-existing attacks would determine the diagnosis of epilepsy; while the existence of a neuropathic family history, or the presence of stigmata of degeneration, would point to eclamptic attacks as being of epileptic nature. The presence of albuminuria does not of necessity form the main element in the differential diagnosis, as albumen has been found in the post-paroxysmal urine of epileptics (Voisin and Péron) although it is not of common occurrence.

"It is therefore clear that many cases of puerperal eclampsia are really examples of serial epilepsy, or the status epilepticus, induced during the puerperium in predisposed and neuropathic persons."

I have gives these long quotations from Dr. Turner, as they represent the views of a man who has had a very large experience in epilepsy. With most of his statements I am in agreement, but I cannot agree with the statement that many cases of puerperal eclampsia are really examples of serial epilepsy, or the status epilepticus. I

have now seen considerably over 100 cases of puerperal eclampsia, and, before the present series, there were only two which I considered were really epileptic cases. In one of them there was no albumen in the urine, and in the other there was a considerable quantity, but in the latter case there was a distinct history of previous epileptic seizures. The two conditions resemble each other so closely that it is difficult to distinguish between them, but there are so few cases of eclampsia which subsequently have fits, even in connection with pregnancy, that, I think, we are justified in concluding that a true case of puerperal eclampsia is not one of epilepsy. It is impossible to follow up the subsequent history of Hospital cases, but in my private cases I have not met with one in which epilepsy has developed. In this connexion it will be interesting to hear the experience of others, and in that way we may get the results of a fairly large number of cases.

Case I. Mrs. T., æt. 27, iii-para, full time, was admitted to the Glasgow Maternity Hospital on February 27th, 1907, at 7-30 a.m., with a history of having had 12 fits since 4-25 a.m.

The patient's father died in an asylum, where he was confined apparently on account of melancholia. Her mother is alive and well. She had had seven sisters, one of whom died at the age of 20, after having had epileptic fits for six years. The other sisters are quite healthy but one is very rickety. She had no brothers. She had always been a healthy woman and had never had any serious illness. There was no history of any injury and no evidence of syphilis. She had never had a fit prior to the morning of admission.

The patient was married on February 6th, 1903; her first child was born in October of the same year, and the second in June, 1905. Both children were delivered with forceps at the patient's home. She had had no miscarriages.

During the present pregnancy the patient's health had been good. There had been no swelling of the face or legs, and no headache until the evening before admission. On that evening she had complained of headache, and had not felt well, but there was no other symptom until she took the first fit at 4-25 a.m.

On admission, the patient was quite unconscious, and had several fits in the reception room before she was removed to the labour ward. The fits were of the ordinary eclamptic type. There was no cry at any time. The pulse was full and bounding, and the respirations were hurried.

I saw the patient at 10 a.m., and up to that time she had had 21 fits. She was at once put under chloroform and 16 fl. oz. of blood were drawn from a vein in the right arm and 3 pints of saline solution (1 dr. of sodium chloride and acetate to each pint) were infused. While this was being done, I found that the os was about half dilated, so I finished the dilatation manually and delivered the

child by version. The uterus retracted well, but there was considerable bleeding from the cervix, which had been lacerated bilaterally. After I had allowed her to lose a good deal of blood I stitched the cervix with catgut. The pulse remained fairly full and strong.

The delivery was finished at 10-45 a.m., and the patient was free from fits until 6-20 p.m. (*i.e.*, an interval of 8 hours). During this time she had been able to swallow 6 fl. oz. of milk, 3 fl. oz. of imperial drink, gr. viii. of calomel, and later a dose of magn. sulph., and although she was not sufficiently conscious to answer questions she could be roused when spoken to. From 6-20 p.m. to 8-20 p.m., she had 14 fits. A hot pack was given at 8 p.m., and from 10 p.m. on the 27th until 3 a.m. on the 28th (*i.e.*, for 5 hours) she had no fits. At 11-15 p.m. an intravenous injection of two pints of the saline solution was given into the left arm. After midnight the patient perspired freely. The pulse rate had varied from 96 to 116, the temperature from 100·2° to 101·4°F., and the respirations were 28. The urine was found to be quite normal without a trace of albumen.

February 28th. The fits began again at 3 a.m., and she had from four to seven per hour until 10-30 a.m. At 3-15 a.m. and 6-30 a.m., 30 grains of chloral and 60 grains of potassium bromide were given per rectum. Part of the first injection was returned, but the second one was retained. At 10-30 a.m., an intra-cellular injection of 2 pints of the usual saline solution was given beneath the right breast, and the fits became less numerous. At 12-45 an attempt was made to draw off some cerebro-spinal fluid, but none could be obtained, although the needle was inserted four times. Venesection was now performed in the right arm and 9 fl. oz. of blood were withdrawn, and 2 pints of saline solution, with double the quantity of sodium acetate, were infused. The fits remained infrequent until 3 p.m., when they again became more numerous and gradually increased in number until she had 13 between 8 and 9 p.m. In the afternoon, between 3 and 5 p.m., $\frac{3}{4}$ of a grain of morphia was given hypodermically in 3 doses without the least apparent effect on the fits. At 9 p.m., 30 gr. of chloral and 60 gr. of potassium bromide were given by the rectum, but the fits continued at the rate of 12 per hour. At 10-30 p.m., lumbar puncture was again tried, and at this time a little over a fl. oz. of cerebro-spinal fluid was withdrawn. The fluid was very slightly opalescent, but did not contain more blood than could be accounted for by the puncture. A slight deposit settled from the fluid and this was found to consist of polymorphonuclear leucocytes with a larger number of lymphocytes (small mononuclear leucocytes). The withdrawal of the cerebro-spinal fluid did not produce any change in the patient's condition. The fluid escaped from the needle by drops except during the fits, when it ran freely. Between the fits there did not seem to be any increase in the tension.

After the fluid was withdrawn a dose of stovaine was injected, and almost immediately there was a lessening of movements in the legs during the fits, but the fits continued to recur about every five minutes. In a few minutes there was absolute paralysis of the lower limbs. The paralysing effect spread up to the arms, and movement in them became much less marked. The diaphragm was not affected. Shortly after the stovaine had been administered, the movements, which had all along been much more marked on the right side, now became confined to the left side. In a short time the fits ceased to affect the left side and returned to the right. The patient became extremely collapsed, and the pulse disappeared entirely from the wrist. Strychnine ($^1/_{30}$ gr.) was given hypodermically, and a pint of hot milk and 3 fl. oz. of brandy were injected into the stomach through a tube. The pulse rapidly returned to the wrist, but the patient remained collapsed for a considerable time.

March 1st. The patient remained in much the same condition all day. The pulse kept remarkably good, the skin acted well, the bowels moved freely, and plenty of urine was passed, partly by catheter and partly unconsciously in bed. The patient was fed at intervals with milk, water, and brandy by the stomach tube. At 5-50 p.m. 2 pints of the usual saline solution were infused into the abdominal walls, and at night $\frac{1}{4}$ gr. of morphia was injected subcutaneously. The fits occurred at the rate of from 6 to 14 per hour.

March 2nd. At 8 a.m. the fits became much more frequent, and she had 20 between 8 and 9 a.m., and 30 between 9 and 10 a.m.; the fits were slighter than on the previous days, but the patient passed almost immediately from one to another. The pulse was still remarkably good, but the patient's general appearance was bad. The fits hardly affected the legs, and the movements of the arms were not nearly so marked as during the first two days. An attempt was made to examine the fundi oculi with the ophthalmoscope, but nothing abnormal could be detected except that the discs looked rather too red. No optic neuritis could be detected. The superficial layers of the corneæ were beginning to show evidence of destruction, and this, with the continuous movements of the head, made the examination very difficult. Between 12 noon and 1 p.m. the patient had the greatest number of fits during any hour, *viz.* 32.

At this time Mr. Hogarth Pringle kindly saw the patient with me and we discussed the advisability of trephining, but concluded that it would be of no avail.

From 6 p.m. the patient gradually sank and she died at 7-40 p.m. without any other change taking place in her condition. The rectal temperature taken immediately after death was 104·2°F. The number of fits recorded was 774.

The fits, though so numerous, did not appear to be very exhausting. All along they were much more marked on the right side than on the

left, except for a short time after the injection of the stovaine. As a rule, they commenced with twitchings of both upper eyelids, especially of the right, followed by conjugate deviation of the eyes to the right, the right arm and right leg were then affected and the left side only very slightly. During the tonic stage the patient did not become so cyanosed as is ordinarily seen in epileptic or eclamptic seizures, and she never gave a cry. The fits lasted from ½ a minute to 2 minutes, and the tonic and clonic stages were of much the same duration.

For the first two days no albumen could be detected in the urine, and even later there was never more than a very faint trace. Towards the end some finely granular tube-casts were found on centrifugalizing the urine. At no time did the patient vomit, not even after the stomach tube had been passed. There was some retching after a few of the fits, but that was all. There was no jaundice. The knee-jerks were absent. The highest temperature recorded was that taken, per rectum, immediately after death, viz., 104·2°F. The skin acted well during the whole of the last three days and abundance of urine was excreted. There was no œdema at any time.

The child, a male, was delivered by podalic version. It weighed 7¾ lbs. At birth it was asphyxiated, but was easily resuscitated. It remained, however, extremely blue during the whole of the 27th Feb., and the colour never became quite satisfactory, especially in the arms and legs. It lived three days, and during that time its body was more or less in a state of rigidity, the stiffness becoming much more marked at intervals. It had many of these fits of rigidity, although there were no convulsive attacks. At times the body assumed a position of opisthotonos, and it could be lifted by placing one hand under the head and another under the heels. When these fits of rigidity came on it gave a peculiarly distressing cry and then became very cyanosed. It was very restless and cried a great deal. Its urine contained a considerable amount of albumen. Chloral hydrate was given to it in grain doses every hour at first, and finally every half-hour, and it gradually grew quieter, but whether this was due to the action of the chloral or to the increasing weakness it was impossible to say.

A post mortem examination was performed on both bodies by Dr. Carstairs Douglas.

The Mother. On exposing the dura mater a number of small hæmorrhages were seen, some punctiform and others slightly larger, due to rupture of venules within the membrane. The whole venous system of the membrane was engorged to a striking degree, the engorgement being very evident in the vicinity of the superior longitudinal sinus. The engorgement was much more marked on the left side of the brain. The meninges stripped off quite easily, and there was no evidence of any thickening or of inflammatory adhesions.

The brain substance was somewhat firmer than usual. No lesion could be detected. About an inch of the upper end of the spinal cord was removed and it appeared to be quite normal.

Microscopic examination of sections from the motor area showed that the nerve cells were normal. A small aneurismal dilatation was observed on one of the minute arteries.

Thorax. Both lungs were adherent and showed evidence of old pleurisy. The lung tissue was fairly normal. The heart was normal except that the tricuspid valve was dilated.

Abdomen. The liver showed a slight nutmeg condition. The spleen was congested, pulpy and enlarged. The kidneys were both smaller than normal. The capsules stripped off readily. The substance was pale and firm. The cortical area in both was diminished in size. The uterus, ovaries and tubes were normal.

Microscopic examination of sections of the kidneys showed evidence of acute nephritis. There were some hæmorrhages and blocking of the tubules with débris.

THE CHILD. The body was plump and well nourished. The skull was harder than usual. The meninges were healthy. The brain was soft and pulpy, and showed a fair amount of vascularity, but not quite as marked as in the case of the mother. There were no indications of hæmorrhage in any part.

Abdomen. The liver was of the usual size; a little pale and spotty in parts and engorged along the margin. The spleen was of ordinary size and consistence. The kidneys were lobulated and normal in appearance, but somewhat smaller than usual.

Microscopic examination of the kidneys showed evidence of congestion, but not so marked as in the mother's. There were some hæmorrhages and some of the tubules showed exudate.

Remarks. The differential diagnosis between epilepsy and eclampsia is very difficult, and some alienists seem to think the two conditions are identical. If there is a history of previous epileptic seizures I think one would be justified in looking upon the case as epileptic. The condition of the urine will not be of much assistance unless there is a large quantity of albumen in it. I have never yet seen a case of eclampsia in which there was no albumen in the urine. I am aware that such cases have been recorded, but may not these have been cases of epilepsy? In the case just recorded there was no albumen in the urine at the onset. At first I looked upon the case as an ordinary eclamptic, but when I found there was no albumen in the urine, and the fits began to recur, I concluded that we were dealing with a case of epilepsy. Towards the end of the case there was a trace of albumen in the urine, but that was what was to be expected.

The status epilepticus is a condition seen frequently enough in asylums. During a seizure the number of fits may be very great. Dr. Turner says that in a case of Leroy's there were 488 fits in

24 hours and 1,000 in three days, and in a case of Parsons's there were 1,400 in four weeks. Turner has seen 2,080 in 8 weeks, 673 in 10 days, 820 in 5 days, and as many as 289 in 24 hours. In my case there were 774 fits in the 88 hours from the onset until death, but during 14 of these hours the patient was free from fits, so that the 774 fits occurred in 74 hours, an average of over 10 per hour, and in one hour there were 32. In the last 24 hours there were 407, and in the last 12 hours no fewer than 261. It seems almost incredible that any constitution could stand such a terrible strain for so long.

In regard to treatment, drugs seem to have no effect. It will be noticed that the fits ceased for eight hours after bleeding, saline infusion, and delivery. After a hot pack they also ceased for one hour, and again for five hours after a cellular transfusion. After the intra-cellular infusion and the second bleeding and third (intra-venous) infusion, they lessened, but did not entirely cease. The final intra-cellular infusion seemed to have no effect. Eleven pints of saline solution was used, seven of them directly into the vein. In the third infusion I doubled the quantity of sodium acetate. I did this in the hopes of neutralizing any lactic acid which might have formed in the blood. Lactic acid has been found to be present in the blood of eclamptics, and it has been suggested that the efficiency of my solution is due to the neutralizing effect of the sodium acetate. The patient's blood must have been well diluted, and as the kidneys and skin were acting so well one would have expected that if a toxin were present it would have been flushed out.

The removal of the cerebro-spinal fluid did no good. The tension did not seem to be raised except during the fits. I tried the injection of stovaine but the effect was very alarming and for a time I thought the heart would be paralysed. The injection of hot milk and brandy into the stomach and $^1/_{30}$ of a grain of strychnine hypodermically had a splendid effect upon the heart and soon brought the pulse back to the wrists. If I should ever have another such case to treat I shall be inclined to try the effect of an injection of potassium bromide.

By a curious coincidence at the date on which I read this paper I had under my care in Hospital two cases of epilepsy and I also admitted a third one about 10 days later. I shall give short notes of these three cases.

Case II. A., æt. 34, ii-para, was admitted to Hospital on April 28th, 1907, under the care of Dr. Munro Kerr, and she came under my care on May 1st, when I took up duty.

The patient's first child was born four years ago, and died in November, 1906, with cerebral symptoms following a discharge from the ear. Previous to her first pregnancy the patient had never had any convulsions, but when the pregnancy was 3 or 4 months advanced, fits had commenced to occur, and at that time as many as 50 had

occurred in 24 hours. Since this pregnancy she had had attacks of *petit mal* every few weeks. These attacks had become less frequent of late.

On April 24th the patient complained of headache, and on the 25th she had one fit, on the 26th 4 fits, on the 27th 8 fits, and on the 28th 9 fits.

On admission, the patient was conscious between the fits, and, though dazed, she was able to answer questions intelligently. She was $7\frac{1}{2}$ months pregnant. There was no indication of labour. There was a very faint trace of albumen in the urine, but no œdema.

At 5 p.m., $2\frac{1}{2}$ pints of saline solution were infused into the median basilic vein, and 7 grains of calomel followed by magnesium sulphate were given and $^1/_6$ gr. of morphia hypodermically. Four fits occurred before midnight.

April 29th. Hypodermic injections of $^1/_6$gr. of morphia were given at 1-30 a.m. and 4-50 a.m. and also 30 gr. chloral and 60 gr. of potassium bromide per rectum at 3 p.m., but the fits continued. Strychnine $^1/_{60}$ gr. was given 4-hourly. Twenty-three fits occurred during the day. At 8 p.m., Dr. Kerr performed vaginal Cæsarean section and emptied the uterus.

April 30th. The patient had 42 fits.

May 1st. The patient had 9 fits.

May 2nd. The patient had 2 fits.

May 3rd. The fits had ceased, but the patient was very delirious. The restlessness and delirium continued for three days, and after that recovery was rapid.

It will be noticed that the greatest number of fits occurred on the day after delivery. There were 102 fits in all. The case might be termed one of the status epilepticus. The fits first began during pregnancy, so that according to Turner's classification pregnancy would be given as the original cause.

Case III. Mrs. M.S., æt. 21, iii-para, was transferred to the Maternity Hospital from the Royal Infirmary, where she had been under treatment for epilepsy for some weeks.

Her mother was eight years ago confined for some time in an asylum. There is no other history of mental disease in the family. The patient's two children are alive and healthy.

The patient states that as a girl she enjoyed good health, but her parents have told her that she had convulsions when she was 12 years old. She has no recollection of them. She was married four years ago, and her first child was born in June, 1904. When six months pregnant she began to take fits, and as pregnancy advanced they became more frequent, 2 or 3 a week. Just before labour they became much more frequent. She says she was unconscious during the labour and for a couple of hours afterwards. She had about two fits a week during the puerperium. She could not nurse her baby.

During the second pregnancy she had about three fits per week. The child was born in December 1905, at full term. The patient was again unconscious during the labour. As on the first occasion a midwife attended.

The third, present, pregnancy began about 7 months ago. Between the second and third pregnancies she had had about 3 fits a week. About 7 weeks before admission to the Maternity Hospital the fits became more frequent, and she fell and injured her face during an attack. She was admitted to the Royal Infirmary, and after 4 weeks' treatment in the Infirmary the fits ceased. She had 4 fits on the day of admission. She had never had any treatment for the fits prior to this. She was transferred to the Maternity Hospital as a slight vaginal discharge of blood had commenced.

There was no indication of labour; the urine contained a trace of albumen; the bromide mixture of 15 gr. doses thrice daily was continued.

Two days after admission labour came on and a premature female child (3 lbs.) was born alive, but only lived 5 hours. There were no fits during the labour and there was only a very slight one in the puerperium, on the first day she was allowed out of bed.

The patient stated that she never had any warning of an attack, and she was usually unconscious for some hours after a seizure.

In this case there is the history of convulsions at the age of 12, so that there was a predisposition, but pregnancy was the exciting cause. In both of these cases there was a trace of albumen in the urine.

Case iv. Mrs. B., v-para, æt. 26.

The patient's mother committed suicide, after a former unsuccessful attempt, by eating rat poison. Of her father's brothers one died in an asylum two years ago, and another is in an asylum at present.

At the time of her mother's death she was ten months old, and was being nursed by her mother. Shortly afterwards she is said to have begun to take convulsions, and she can remember having had convulsions as a child. After the age of 13, the attacks were not always of a convulsive nature. She sometimes lost consciousness, but did not struggle. Her friends informed her that in some of the attacks she acted and spoke in an unusual way, but she has no recollection of this.

She was married 7 years ago, and since marriage the attacks have never been convulsive, but have consisted of loss of consciousness with strange actions or remarks. She is now pregnant for the fifth time. One of the previous pregnancies was a twin pregnancy. One of the twins died at the age of $3\frac{1}{2}$ months, but all the other children are alive, and in good health, except one which is rickety. Her labours have been slow but natural. During pregnancy the attacks

have always been much less frequent than at other times. When she was nursing, the attacks were most frequent—about once a fortnight.

The labour was natural, and the patient had no attacks while in hospital.

In this case pregnancy seems to have had a beneficial effect on the attacks, but it will be noticed that the attacks were most frequent during lactation. In this case there would be a strong hereditary predisposition. So far the children show no tendency to neurotic attacks.

I am indebted to my residents, Drs. Nielson and Walker, for the careful notes of these cases, and to Dr. Carstairs Douglas for the post mortem reports.