THE VALUE OF CAESAREAN SECTION IN PRE-ECLAMPTIC TOXAEMIA

BY

MARGARET M. BASDEN, M.D.LOND.

F.R.C.S.ENG., F.C.O.G.

OBSTETRIC SURGEON, MOTHERS' HOSPITAL, CLAPTON; SURGEON,
SOUTH LONDON HOSPITAL FOR WOMEN

The object of this communication is to suggest that, although Caesarean section should never play anything but a small part in the treatment of pre-eclamptic toxæmia, it might, nevertheless, be used with advantage more frequently in the most serious cases. In my experience, the high mortality which has been shown to follow Caesarean section when eclampsia has actually supervened¹ does not occur when the operation is performed in the pre-eclamptic stage. The following is a short series of thirty cases of pre-eclamptic toxæmia treated by Caesarean section with no maternal deaths and no subsequent development of eclampsia. I have done my best to include all my cases so treated, but it is possible that one or two may have been overlooked, owing to difficulty in tracing the earlier records. I can state definitely, however, that I have never had a maternal death in a patient treated by Caesarean section for pre-eclamptic toxæmia, and the same applies in cases of Caesarean section performed for other reasons, but in which a mild degree of toxæmia was present, too slight to include in this series.

Caesarean section for toxæmia does not condemn the patient to delivery by this method in subsequent pregnancies. Since it is from eclampsia that the patient dies, and not from pre-eclamptic toxæmia, I seldom operate unless eclampsia appears to be so imminent that there is no time to induce labour. My usual practice in severe

cases is to keep the patient on water only, or water, glucose, and lemonade, for twenty-four hours. The response to this treatment is generally satisfactory, but if at the end of that time there is only a slight improvement, I induce labour, while if the condition is the same, or worse, I perform Caesarean section. I should have operated sooner in some of the following cases had they come under my care earlier. I have not found that toxæmic patients stand the operation less well than others, and I am forced to the conclusion that the risk of operative procedure in this condition has been over-estimated.

Vaginal Caesarean section was performed in one case; in all the others the classical operation was carried out. The anaesthetic employed was usually ether, sometimes preceded by ethyl chloride on an open mask. I should prefer spinal anaesthesia, or gas and oxygen, but, since the operation need not take more than a quarter of an hour, I do not think the choice of the anaesthetic is of very great importance. This may prove a practical point in an emergency operation.

Eclampsia following Induction

That induction, even when followed by the immediate onset of labour, is too slow a method to be relied upon to prevent eclampsia in severe cases is illustrated by the following examples.

Case 1.—Mrs. A., a primipara, was under my care for her confinement. When thirty-eight weeks pregnant, her husband, a doctor, reported a trace of albumin for the first time. Her blood pressure, which on a previous occasion had been 135, was 150. There was practically no oedema, and she felt quite well. As she had a slightly contracted pelvis, and induction had already been considered for this reason, it was decided to induce labour, although neither the toxæmia nor the pelvic contraction alone seemed sufficient to indicate this. She went

Case No.	Date of Admission	Age	Para	Duration of Pregnancy (weeks)	Condition on Admission and till Operation	Reason for Operation and Date	Date of Discharge and Result	
							To Mother	To Child
1	September 19th, 1924	25	1	40	Blood pressure 145; much oedema of abdominal wall and legs. No albumin, although this had been present in antenatal department	September 24th. Despite treatment oedema increased; blood pressure rose to 170, and urine was loaded with albumin. Labour was induced with a stomach tube, but on following day blood pressure was 175, and albumin had still further increased. Labour was progressing very slowly	October 13th. Febrile puerperium. Finally did well	Good
2	May 1st, 1925	26	1	40	In labour. Membranes ruptured; heavy cloud of albumin. Blood pressure 152. (Oedema of face, feet, and abdominal wall had been noted four days earlier in antenatal department, but notes do not say whether this was present on admission)	May 1st. Very slow progress of labour owing to disproportion. General condition getting worse. Temperature 101.2°	July 2nd. Febrile puerperium; Finally did well	Good
3	November 22nd, 1925	30	1	40	Admitted with "show." No pains; no toxic symptoms. Following day developed very severe headache, nausea; trace of albumin. Blood pressure 160. Drug induction failed	December 4th. Extended breech which would not descend with good pains. Toxic symptoms were still present on December 2nd, but were getting less. Condition on day of operation is not stated in notes	December 23rd. Good	Good
4	June 4th, 1926	39	2	40	Oedema of feet, legs, and abdominal wall. Blood pressure 130. Passing very little urine; trace of albumin. All symptoms improved, but she later relapsed	June 22nd. Almost complete anuria; what there was was loaded with albumin. Very marked oedema; blood pressure 148. Very anxious for a living child; twins had been diagnosed and it was feared they would succumb to the toxæmia	July 23rd. Good	Triples; all did well
5	April 1st, 1927	25	1	32	Very marked oedema. Urine loaded with albumin; had been vomiting. Blood pressure 170	April 2nd. Worse, despite being on water only. Urine nearly solid on boiling. Blood pressure 184	April 28th. Good	Good
6	May 13th, 1927	23	1	40	Much oedema, and albuminuria. Blood pressure 160	May 14th. After water only for 24 hours, urine was nearly solid on boiling; only 16 oz. passed. Blood pressure still 160	Good	Good
7	May 27rd, 1927	29	1	36	Oedema of feet and legs; much albumin. Blood pressure 160. Temporary improvement followed by relapse despite medical treatment	May 29th. Blood pressure 190. Much albumin; headache	June 18th. Good	Good
8	September 30th, 1927	38	1	38	Much albumin; oedema of feet. Headache and eye symptoms. Blood pressure 144. Temporary improvement under treatment, then oedema, albumin, and blood pressure increased. On October 6th blood pressure was 164	October 7th. Tending to get worse despite treatment, although on actual day of operation there was improvement, and blood pressure was 128	October Good	Good
9	November 4th, 1927	24	1	40	Looked ill. Puffiness of face; very marked oedema of vulva and much oedema of legs. Much albumin. Blood pressure 140	November 4th. Risk of eclampsia supervening seemed too great to allow time for medical treatment	November Good	Good
10	January 20th, 1928	24	1	34	Much albumin and oedema. Blood pressure 140. After water only for 24 hours and magnesium sulphate, albumin was the same, oedema less. Blood pressure 170. Following day, face puffy, albumin same; had vomited. Much oedema of abdominal wall. Blood pressure 150	January 22nd. Failure of response to medical treatment. Fear that eclampsia might supervene during induction	February Good	Good

TABLE (continued)

Case No.	Date of Admission	Age	Para	Duration of Pregnancy (weeks)	Condition on Admission and till Operation	Reason for Operation and Date	Date of Discharge and Result	
							To Mother	To Child
11	July 4th, 1928	25	1	36	Blood pressure 172 (which rose to 180) and albuminuria. Under treatment blood pressure dropped to 135 and albumin disappeared			
12	Re-admitted August 11th, 1928				When re-admitted blood pressure was 210. Eye symptoms present; albuminuria. Attempt to induce labour by bougies, followed by "intensive" drug treatment, unsuccessful. Toxaemic symptoms improved temporarily, then began to increase again	August 28th. Recurrence of toxaemic symptoms after temporary improvement. Failure to induce labour	September 17th. Good	Stillborn
13	September 29th, 1928	24	1	? 38	Vomiting; slight jaundice. Albuminuria and oedema. Blood pressure 115	September 30th. After strict medical treatment condition became worse. Blood pressure 118. Restlessness, headache, epigastric pain, and blurred vision. Albumin and bile in urine. Marked oedema	October 26th. Good; febrile puerperium, but developed active tuberculosis	Twins; both did well
14	February 8th, 1929	25	1	40	Much albumin. Blood pressure 185	February 9th. After 24 hours on water only blood pressure was 180	March 2nd. Eventually good; convalescence delayed by bronchitis	Good
15	April 5th, 1929	27	1	33	Albuminuria; much oedema. Blood pressure 152	April 6th. After 24 hours' medical treatment blood pressure 172	June 10th. Eventually good; convalescence delayed by phlebitis	Good
16	May 13th, 1929	27	1	? 38	Much oedema and albumin. Blood pressure 165. (In the ante-natal department a high blood pressure and oedema had preceded the albuminuria)	May 14th. After 24 hours' medical treatment blood pressure was 160. (She was apparently either worse, or no better, but the notes of this and of the two preceding cases are deficient)	June 11th. Good	Good
17	April 4th, 1930	27	2	34	Much albumin; blood pressure 165. Despite treatment blood pressure rose next day to 200, dropping to 190. Albumin less.	April 6th. Blood pressure 195. Vomiting; face puffy. Worse, despite medical treatment	April 28. Eventually good; convalescence delayed by bronchitis	Good
18	July 7th, 1930	36	1	38	Blood pressure 178. Nausea, headache, and vomiting. Much albumin and oedema of legs	July 8th. After water and barley sugar only for 24 hours blood pressure was 175; passing very little urine. The patient had already been advised Caesarean section for contracted pelvis	July 28th. Good	Good
19	August 28th, 1930	30	1	40	Much oedema, headache, and vomiting. Blood pressure 135. No albumin on admission, but this appeared next day	September 21st. Temporary improvement under medical treatment, followed by relapse. Blood pressure now 170; much albumin	October 10th. Good	Good
20	October 17th, 1930	27	1	40	Oedema and headache; albumin absent or trace only. Blood pressure 150. Admitted for drug induction. Oedema and high blood pressure (170) had preceded albuminuria in ante-natal department	October 19th. No result from drug induction. Trace of albumin; headache. Blood pressure 170. Getting worse, despite medical treatment	November 8th. Good	Good
21	October 19th, 1930	25	1	36-38	? In labour. Abdominal pain and vomiting. Oedema of feet and legs; blood pressure 150. There is no note as to condition of urine on admission, but there had been a faint trace of albumin in ante-natal department, preceded by oedema and high blood pressure	October 20th (12.30 a.m.). Blood pressure rose to 200 after admission, and eclampsia seemed imminent	November 8th. Good	Good
22	January 29th, 1931	21	1	30	Blood pressure 195. Oedema; much albumin (oedema, headaches, and high blood pressure had preceded albuminuria in ante-natal department). Two attempts to induce labour by insertion of bag failed	February 3rd. (Vaginal Caesarean section.) Persistent high blood pressure and albuminuria, despite strict medical treatment. Blood pressure on day of operation was 175, but it varied between 160 and 195	February Good	23rd. Stillborn
23	January 30th, 1931	20	1	34	Oedema of feet and much albumin. Blood pressure 180. After 24 hours on water only blood pressure was 150	February 2nd. Blood pressure rose again to 170. Severe headache; oedema increasing	February Good	20th. Good
24	March 20th, 1931	25	3	34	Severe headaches and eye symptoms. Blood pressure 150. Much albumin. (Had albuminuria in ante-natal department for more than three weeks, despite medical treatment)	March 20th. Had albuminuria and high blood pressure with both previous pregnancies; the first was induced at 6½ months. The present operation was performed both to prevent onset of eclampsia and to sterilize	May 8th. Good. The long convalescence was due to delayed healing of wound	Died day after birth
25	April 6th, 1931	31	2	—	Albuminuria. Oedema of legs and feet and enormous oedema of vulva; two pints of fluid let out by tapping. Blood pressure 145	April 12th. Chief toxaemic symptom was great oedema of vulva, which reappeared after tapping	May 4th. Good	Twins; both did well
26	June 23rd, 1931	26	2	26	Much albumin; oedema. Blood pressure 190. After water only for 24 hours blood pressure was 210; highest reading was 220, fell before operation to 210	June 27th. To prevent eclampsia and to sterilize. She had severe toxaemia with previous child, which was induced at 26th week, but which lived and did well	July 8th. Good	Died
27	September 21st, 1931	24	1	32	Oedema; trace of albumin. Blood pressure 145	October 4th. Getting worse despite treatment. Drug induction failed. Blood pressure 180; headache and eye symptoms	October Good	29th. Twins (dead); born alive
28	November 18th, 1931	34	5	34	Oedema; cloud of albumin. Blood pressure 235. (Previous pregnancies said to have been uncomplicated)	November 18th. Ten hours later blood pressure 220, despite water, glucose, etc., only. Fear of impending eclampsia	Jan. 2nd. Good. Prolonged convalescence owing to phlebitis	Good
29	April 1st, 1932	—	1	38	Much oedema and albumin. Blood pressure 165	April 4th. Improved at first on water only; blood pressure fell to 120, then rose to 150. Headache	April 25th. Good	Good
30	November 1st, 1932	26	1	40	Oedema of legs, hands, and face. Blood pressure 132. Heavy cloud of albumin. (Trace of albumin noted in ante-natal department four days previously)	November 5th. Albuminuria gradually disappeared under strict medical treatment, and the oedema diminished, but the blood pressure continued to rise, and on day of operation it was 174	November Good	25th. Good
31	December 9th, 1932	25	2	40	Blood pressure 172. Much albumin. Had had trace of albumin in ante-natal department on three separate occasions, the first being in September. It always disappeared on dieting, and the blood pressure varied from 110 to 140	December 9th. She was admitted for operation the same day, both to prevent eclampsia and because she had had toxaemia in her previous pregnancy and a stillborn child. She was kept on water only until the operation, but the blood pressure continued to rise slightly (to 176) and the urine became almost solid with albumin on boiling	Good	Good

into the nursing home, and her urine was then found to be albumin-free. Later in the day labour was induced by rupturing the membranes.² Pains started about two hours later, but eleven hours after the membranes had been ruptured she had a fit, and the urine was found to be almost solid with albumin.

Case 2.—Mrs. W. was admitted into a maternity home for treatment for severe toxæmia. The urine was loaded with albumin, her legs were very oedematous, and her blood pressure was between 180 and 190. After she had been in bed on a restricted diet the oedema diminished and the albumin almost disappeared; the blood pressure remained the same. About five days after admission the oedema began to increase again, the blood pressure remaining as before. The doctor then decided to induce labour; the operation was very successful, labour starting in four hours. The child was born twenty-four hours later, but by then the mother had had an eclamptic fit, and I had been asked to see her in consultation.

In both cases the mother and child did well and the induction probably diminished the severity of the disease. It gave time, however, for the relatively safe pre-eclamptic condition to pass over into the very dangerous one of eclampsia, although in both cases there was a minimum of delay between the induction of labour and the birth of the child. The second case illustrates the prognostic value of the blood pressure compared with other symptoms, and this is the symptom on which I chiefly rely in estimating the severity of the case. I have also found that when the high blood pressure and oedema appear

before the albuminuria the toxæmia is particularly apt to be severe. This happened four times in my series.

Figures are not available to show what percentage of cases of pre-eclamptic toxæmia develop eclampsia, but it has recently been suggested that 6.4 per cent. of late pregnancy toxæmia cases develop this condition.³ It is reasonable to assume that the percentage, when only the most severe cases are considered, would be higher, and therefore that some of the cases in this series would certainly have developed eclampsia but for the operation.

It has been argued that this method of treatment is contrary to the interests of the child, but in my experience the child is more likely to die as the result of prolonged toxæmia in the mother than from prematurity, and I sometimes use this as an argument to the parents in advising the operation. The figures in this series show that the actual foetal mortality was 17.1, six children having died of the thirty-five that were born. This included three sets of twins, of whom one set died, and one of triplets. Five out of the thirty mothers, or 16.6 per cent., lost their children, and twenty-three of them, or 76.6 per cent., were primiparae.

REFERENCES

- ¹ FitzGibbon, G.: *Journ. Obstet. and Gynaecol. of the British Empire*, 1931, xxxviii, 495.
- ² *Proc. Roy. Soc. Med.*, 1922-3, xvi (Sect. Obst.), 1-11.
- ³ Young, J.: *Proc. Roy. Soc. Med.*, June, 1932.

Memoranda

MEDICAL, SURGICAL, OBSTETRICAL

A CASE OF POST-VACCINAL ENCEPHALITIS

The following brief account of post-vaccinal encephalitis occurring in an adult and resulting in recovery seems worthy of record.

Miss N. B., a robust, healthy girl, 19 years of age, was primarily vaccinated with fresh glycerinated calf lymph on November 15th, 1932. Two vaccination marks were made on the left calf.

On November 28th the patient attended surgery, complaining of malaise, lassitude, and diffuse headache. On examination she had normal pock-marks with no inflammatory areolae. The same evening all symptoms were accentuated and vomiting occurred once. The headache had become occipital; there was no stiffness of the neck; and all reflexes were normal, except for an indefinite Babinski reflex on the left. Temperature 101° F., pulse 90, respiration 24.

Early on November 29th, after a night of alternating restlessness and drowsiness with occasional vomiting, she was apathetic and answered questions briefly. Stiffness of the neck was marked, with no retraction. Pupils and retinae were normal, except for slight lateral nystagmus. The lower abdominal reflexes, the left knee-jerk, and both ankle-jerks were absent. The upper abdominal reflexes and right knee-jerk were diminished. There were bilateral Babinski and Kernig reflexes (left ++, right +). At midday the patient was comatose, with urinary incontinence. Lumbar puncture produced 32 c.cm. of clear fluid, and 10 c.cm. of anti-meningococcal serum was substituted intrathecally. The leucocyte count showed 18,125 cells per c.mm., 80 per cent. being polymorphonuclears. At midnight the patient was unconscious, with Cheyne-Stokes respiration. She was then given an intravenous transfusion of 30 c.cm. citrated blood from her mother, who had been vaccinated twenty years previously, and 23 c.cm. of cerebro-spinal fluid was withdrawn by lumbar puncture. One hour after transfusion generalized skeletal spasm occurred, which ceased after subcutaneous injection of adrenaline.

Early on November 30th the patient was still comatose and incontinent of urine and faeces, but Cheyne-Stokes breathing

had ceased. Consciousness gradually returned, and towards evening she became restless. At midnight 30 c.cm. of serum from a Group IV donor, who had been vaccinated three and a half years previously, was given intravenously, and 20 c.cm. of cerebro-spinal fluid was withdrawn by lumbar puncture.

On December 1st the comatose state gave way to a lethargic state. Sphincter control was restored, but occasional enuresis persisted. During December 2nd, 3rd, and 4th the following reflexes were still absent: left upper and both lower abdominal, both knee- and ankle-jerks. Plantar responses extensor.

On December 14th all abdominal reflexes and both knee-jerks were present; the left plantar response was still extensor and the right flexor. Neither pain nor paresis was observed throughout. Further recovery was uneventful.

EXTRACT FROM CEREbro-SPINAL FLUID EXAMINATION
(November 29th, 1932)

Culture—sterile.
Cells—233 per c.mm. 1/3 small lymphocytes.
1/3 polymorphonuclears.
1/3 large endothelial.

Globulin—increased.
Protein—45 mg. per 100 c.cm.
Sugar—57.7 mg. per 100 c.cm.

Bognor Regis.

JULES F. REY.

INTESTINAL POLYPUS AND INTUSSUSCEPTION

It may be of some interest to record the following case.

The patient, a boy aged 6, was sent to me last summer with a history of vague abdominal pains with occasional vomiting. The bowels were somewhat constipated, with intermittent passage of bright red blood. On examination the general condition was fairly good. The abdomen was somewhat distended and distinctly tender over the appendicular area. Nothing abnormal was made out on rectal examination. A barium meal examination did not help in any way. A diagnosis of appendicitis was made. It was thought that an intestinal polypus might also be present.

At operation the appendix, which was subacutely inflamed, was removed. A search was then made along the large intestine for a polypus, and about the middle of the transverse colon a rounded mass, about three-quarters of an inch in diameter, was found. It felt like a piece of faeces, and on attempting to milk it in a downward direction I noticed that an invagina-