histologists have in many instances subjected the apparently normal tube on the affected side to serial histological examinations and in the end have been compelled to concede it normal upon the basis of a complete absence of chorionic tissue.

Curtis (1941) suggested that in certain cases the ovum, after becoming fertilized in the Fallopian tube, may "slide downwards to the gaping recently ruptured ovary, there to develop." Other writers, like Mann *et al.* (1941) believe that "the ovum becomes fertilized while remaining in the follicle and develops its sac from contiguous ovarian tissue." Each of these views has the merit of plausibility, and it is not improbable that both processes may occur.

In view of the unfavourable nature of the implantation site it might be supposed that ovarian pregnancies generally would come to an abrupt and early termination. Statistics, however, do not support this view, and Dale (1947) collected from the literature 38 cases in which the pregnancy had proceeded beyond seven months, and from which 12 living infants had been produced.

Lithopaedion formation has been recorded in several cases of ovarian pregnancy, and De Lee (1943) reports an ovarian hydatidiform mole. The coincidence of ovarian pregnancy and eclampsia reported by Pride and Rucker (1942) may, however, claim distinction as a rarity of rarities.

Many writers have stressed the relatively slight danger of alarming haemorrhage in ovarian pregnancy as compared with the tubal variety, and the above case in this respect conforms to type. Indeed, it stands out in my experience as one of the very few cases of ectopic pregnancy in which the administration of a blood transfusion would have been completely without justification.

Summary

The relative rarity of primary ovarian pregnancy is stated, with statistical support.

Spiegelberg's criteria of primary ovarian pregnancy are quoted and a case is reported which seems to satisfy these criteria.

Various views on the aetiology of primary ovarian pregnancy are mentioned, with brief comment.

The unexpectedly high proportion of viable infants resulting from ovarian pregnancy is mentioned.

The relative infrequency of severe haemorrhage attendant upon this type of ectopic gestation as compared with tubal pregnancy is stressed.

I am indebted to Mr. Douglas M. Lindsay for permission to report this case, which occurred in his unit at Beckenham Hospital.

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DELAYED CHLOROFORM POISONING IN OBSTETRIC PRACTICE

BY

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Cases of delayed chloroform poisoning in obstetric practice have been reported by numerous writers. The outcome in many of the cases was fatal. Nine of the 10 cases reviewed by Gibberd (1935) were fatal. Stander (1932) reports a mortality of 80% in five cases. This figure is regarded as too pessimistic by Sheehan (1940), since his last six cases recovered and two of them were very severe. Clinical histories of three patients who developed delayed chloroform poisoning are here briefly described.

Case 1

The patient, aged 24, was admitted to hospital on January 10, 1947, following a difficult forceps extraction under chloroform anaesthesia at home 48 hours previously. This was her first confinement and followed a normal antenatal period. The duration of labour was 83 hours. The food and fluid intake had been insufficient during this time, and the patient had experienced troublesome vomiting.

On admission she was drowsy and slightly jaundiced. There was no vomiting. Her blood pressure was 130/85 and her temperature 99.8° F. (37.7° C.). Prophylactic penicillin had been given since delivery. There was no liver tenderness or enlargement. Shortly after admission she became very restless and cried out as if in pain. This was associated with muscle-twitching. Periods of extreme restlessness occurred during the next three days, and on occasion she became almost maniacal. The jaundice deepened during this time. No vomiting occurred and sips of fluid were taken. Her condition then gradually improved, and a slow, steady recovery was made.

Retention of urine before and after admission into hospital necessitated catheterization. The urine output was always satisfactory. A bacteriological examination of the urine showed a *Bact. coli* infection, and the cervical swab yielded a growth of haemolytic streptococci. The blood culture was negative. Pyrexia persisted for several days after admission, but responded to penicillin and sulphonamide therapy.

Intravenous dextrose therapy was given on admission and continued for five days. In each 24 hours two litres of 5% dextrose and one litre of 4.3% dextrose in 0.18% sodium chloride were given. Five units of soluble insulin twice a day, 20 ml. of 10% calcium gluconate intravenously, and vitamin B by injections were given each day. Intramuscular paraldehyde was administered when necessary to control the restlessness.

Laboratory Investigations.—On January 11 (third day after delivery) an immediate positive van den Bergh reaction was given by the serum. The indirect van den Bergh reaction was 5.8 units; this increased to a maximum of 10.3 units on January 13. The alkaline phosphatase was 21.4 King-Armstrong units on January 11 and 22.3 on the 15th; it then slowly returned to a normal level. At all times the colloidal gold test showed no precipitation (0). The Takata-Ara reaction did not show a positive result, and the serum thymol turbidity varied between 0 and 3.2 units. Other blood chemistry estimations on January 11 showed the alkali reserve 34 vols. CO₂ per 100 ml., with the chlorides and total protein within normal limits. The blood urea was 108 mg. per 100 ml., and remained raised for several days. On January 15 the blood urea was 104 mg. per 100 ml., and then returned to a normal level. The urine contained bilirubin and urobilin. The Rothera test was negative on all occasions before and after the beginning of intravenous dextrose therapy. The blood-sugar level of 135 mg. per 100 ml. of blood on January 13 was probably influenced by the dextrose therapy.

Case 2

A primigravida aged 23 started in labour on April 29, 1951. Prior to this the pregnancy had been normal. Further details regarding her labour and delivery were obtained from her doctor. A manual rotation and forceps extraction for a right occipito-posterior presentation after 28 hours' labour had been carried out under chloroform anaesthesia, the duration of which was approximately 20 minutes. Two hours later chloroform anaesthesia was again used for manual removal of the placenta. The history did not indicate any vomiting or undue maternal fatigue before delivery. Vomiting occurred only once after the second anaesthesia. Jaundice appeared about 24 hours after the forceps extraction, increased for two days, and then gradually faded. The patient did not have any symptoms other than nausea and general weakness. She was able to take light diet from the time of delivery, and liberal glucose drinks were given.

She was admitted into hospital on May 10. On examination the skin showed slight icterus, but otherwise her condition was satisfactory. Prophylactic penicillin had been given throughout the lying-in period. Pyrexia had not occurred. Complete recovery was made in hospital without any special treatment. There were no relevant laboratory investigations to report.

Case 3

A primigravida aged 35, following a normal antenatal period, had her confinement at home under the care of her own doctor. On September 3, 1951, after 36 hours' labour, twin males were delivered, the first by a forceps extraction and the second by breech extraction. The patient was under chloroform anaesthesia for approximately 25 minutes. Four and a quarter hours later, chloroform was again administered, and manual removal of the placenta was performed. Adequate sedation was given prior to delivery and only slight post-anaesthetic vomiting occurred. Jaundice developed on the second day after delivery. The patient was sent into hospital on September 7.

On admission she was semi-comatose and deeply jaundiced. There was abdominal tenderness over the liver area, but no liver enlargement was detected. The blood pressure was 130/95 and the temperature 98.4° F. (36.9° C.). Slight oedema of the legs was present. The patient remained comatose for 24 hours. Hiccup was troublesome. During the next four days slight improvement took place, but there was extreme drowsiness, with mental confusion and periods of restlessness. No vomiting occurred and the swallowing reflex was present, enabling small quantities of fluid to be taken by mouth. The urine output was satisfactory, but catheterization was necessary for several days owing to retention. Five days after admission a rigor occurred, due to a *Bact. coli* urinary infection, which responded to streptomycin therapy.

Treatment in this case followed similar lines to that in Case 1. Intravenous dextrose and saline therapy was continued for four days after admission. A slow but steady recovery was made.

Laboratory Investigations.—On September 7 (fourth day after delivery) blood chemistry estimations showed: alkali reserve, 54 vols. CO₂ per 100 ml.; chlorides, 570 mg. per 100 ml.; total protein, 5.3%; blood urea, 50 mg. per 100 ml.; and sugar 52 mg. per 100 ml. On September 8 the alkali reserve was 45 vols. CO₂ per 100 ml.; blood urea, 24 mg. per 100 ml.; and blood sugar, 120 mg. per 100 ml. Thereafter the above estimations were within normal limits.

On September 7 the direct van den Bergh reaction was positive, and the indirect reaction 14.7 units. The alkaline phosphatase was 21.2 King-Armstrong units, serum thymol turbidity 2.2 units, and the thymol flocculation positive. The colloidal gold test showed no precipitation (0). On admission the urine contained a trace of albumin, bilirubin, and excess urobilin. The Rothera test was negative on all occasions.

Comment

Only in Case 1 had the labour produced notable maternal fatigue and been accompanied by much vomiting before delivery. Exhaustion due to a long labour, starvation with dehydration from vomiting, and poor food intake, and also sepsis, are considered by Sheehan (1940) to be the most important factors predisposing to possible poisoning when chloroform anaesthesia is used. Uterine sepsis was present in Case 1. The urinary infections in Cases 1 and 3 occurred after the appearance of the jaundice.

Chloroform is favoured in domiciliary practice, since by its use induction of anaesthesia is easy and rapid; also, by reason of its safety, in rooms where open fires are present. Stander (1932) states that if chloroform is used it should be given in very small quantities for the "crowning" of the head, and at other times it should be avoided if some other anaesthetic is available. There is no doubt that great caution is necessary in using it, and that it is contraindicated in labour cases in which exhaustion is evident, owing to the factors previously mentioned, or in which there is a history of toxæmia.

When delayed chloroform poisoning occurs it may follow one induction or two inductions separated by a short interval of time, as in Cases 2 and 3. Kerr and Moir (1949) point out the danger of repeated administration of chloroform at short intervals of time or for longer than 15 minutes at one time. The total amount of anaesthetic used varies very much in every case, and usually the exact quantity is not known. In cases in which two inductions are given, it is probably the total quantity of anaesthetic administered that is the important factor, rather than the fact that the anaesthetic has been given in repeated doses.

The cases reported all conformed to the usual clinical picture found in cases of delayed chloroform poisoning. The jaundice appeared during the two days after the administration of the anaesthetic, vomiting being followed by drowsiness, restlessness, and perhaps coma. Vomiting did not occur in the cases above, but in those reviewed by Gibberd (1935) vomiting was the first symptom in most cases.

The liver tests performed in Cases 1 and 3 indicated some degree of liver derangement. Such tests, however, as is well known, give very variable results, and may show no change even when the liver damage is severe. Again, in Case 1 the alkali reserve was found to be low and the blood urea raised during the worst phase of the illness. There was less disturbance of these values in Case 3. If liver damage is extremely severe the blood urea may not increase, but in some fatal cases very high levels have been reported, as in a case recorded by Gibberd in which the blood urea reached 300 mg. per 100 ml.

Summary

Three cases of delayed chloroform poisoning in obstetric practice are recorded. All the patients recovered.

Stress is laid on the danger of chloroform anaesthesia when used in certain types of obstetric cases, particularly when there has been a long labour associated with dehydration and starvation acidosis.

A brief account of the treatment given in these cases is described. The early administration of intravenous dextrose was carried out.

I would like to express my thanks to Mr. F. E. Wheeler, under whose care these patients were admitted, and to Dr. J. Mills, in whose department at the Royal Berkshire Hospital, Reading, the pathological investigations were carried out.

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