The capacity of stellate ganglion block to produce dilatation of cerebral vessels has been a further matter for dispute. Naffziger and Adams (1950) reported the use of this manoeuvre in a series of 155 patients suffering from acute cerebrovascular accidents together with 70 other cases suffering from a variety of cerebral disorders. Whilst admitting the difficulty of assessing the effectiveness of this measure in a disorder with such a variable response without special treatment, they conclude that their results were sufficiently gratifying to encourage its use in cases in which cerebrovascular spasm could be inferred. Leriche (1952) and Walsh (1954) have strongly advocated stellate ganglion block in the treatment of recent and late cases of cerebral thrombosis. Our experience in this case has shown that sympathetic denervation can effectively dilate the retinal arteries and by inference can dilate other cerebral arteries under control of the stellate ganglion. As a corollary it is suggested that stellate ganglion block might prove of considerable value in the treatment of other forms of retinal vascular constriction, and especially in the treatment of thrombosis of the central retinal vein and of embolism of the central retinal artery. Cotson (1951) reports a case in which stellate ganglion block was done 5½ hours after the onset of sudden blindness due to an embolus of the central retinal artery. Within fifteen minutes there was a marked dilatation of the entire retinal tree and the embolus was seen to pass into the periphery of the fundus. Within a few days vision was restored.

The effect of quinine on the foetus is worthy of comment. In our case the patient aborted spontaneously; more often the foetus is unharmed and the pregnancy proceeds normally. Professor A. M. Claye, in a personal communication, has suggested that if a large dose of quinine did not produce abortion the foetus would escape serious harm. Nevertheless, it has seemed possible to us, in view of the wide range of sensitivity to this drug, that some unexplained examples of congenital blindness and/or deafness might be due to maternal ingestion of quinine for medicinal or abortifacient reasons.

Finally, it would appear desirable that quinine should be supplied to the public only on a doctor's prescription. Chemists with whom we have discussed this matter have assured us that quinine is rarely purchased nowadays, and only in small quantities, usually for the manufacture of home tonics and wines. Against this view is the fact that our own patient informed us that she had been advised to take quinine by a friend who had herself successfully aborted twice after taking ½ oz. (15.5 g.) of quinine on two separate occasions. Furthermore, a case of quinine poisoning was admitted under the care of one of us (L.G.) at the Royal Halifax Infirmary in December, 1952. This woman took an ounce (31 g.) of quinine sulphate by weight, having purchased it without difficulty at a multiple chemist's shop. She felt ill as soon as she had swallowed the powder, and vomited violently within the first five to ten minutes. She was seen within half an hour by her doctor, who rushed her into the Royal Halifax Infirmary, where the stomach was washed out. She never became completely blind, and her vision recovered fully within three weeks. Whilst writing this paper a further case of attempted abortion by quinine has come to our notice. This woman took an amount of quinine the "size of a sixpence" once weekly for the second two months of her pregnancy. She then gave it up and the foetus developed normally to term. It seems unlikely that our experience is unique. We have wondered whether this dangerous practice is not more common than is realized by the profession or by the pharmacist.

Summary

A case is recorded of complete blindness, of approximately forty hours' duration, due to the ingestion of quinine for abortifacient reasons. Drug therapy was ineffectual, but stellate ganglion block produced an immediate return of central vision and has probably favourably affected the degree of residual loss of peripheral vision. It is suggested that this treatment should

be carried out immediately in all cases of retinal vascular spasm and especially in the treatment of thrombosis of the central retinal vein and embolism of the central retinal artery. The possible dangers to the foetus and the prevalence of the practice are discussed. It is recommended that restriction on the sale of quinine be imposed.

We wish to record our thanks to Mr. R. W. Hendry for his surgical assistance, to Mr. P. M. Wood for the ophthalmic assessments, and to Mr. Norman Emblin, who supervised the gynaecological aspects of the case.

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RUPTURE OF UTERUS FOLLOWING ACCIDENTAL HAEMORRHAGE

BY

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The incidence of rupture of the uterus is difficult to determine with any accuracy. It is particularly difficult to discover the incidence in Great Britain, as few large-scale reviews are reported, and the standard textbooks refer to series mainly from the United States of America. Brierton (1950) analysed eight large American series which reviewed 368 cases of uterine rupture occurring in 734,932 deliveries, an incidence of 1 in 1,997 cases. Watt (1950), from Canada, reported an incidence of 1 in 1,163 cases out of 17,455 deliveries. Simons (1950), from the Walton Hospital in Liverpool, recorded an incidence of 1 in 2,233 cases out of 33,492 deliveries, whilst the Rotunda Hospital (1952) figures were 1 in 1,333 deliveries. These figures seem to indicate that uterine rupture occurs once in every 1,500 to 2,000 deliveries, as is usually stated. All these figures are for hospital-managed cases. One wonders how truly representative these figures are, for as Titmus (1939) suggested, many cases of rupture are 'probably never diagnosed, being registered as deaths due to haemorrhage or shock.

Analysing 30 maternal deaths, Gordon and Rosenthal (1943) found at necropsy that 11 had a ruptured uterus, the diagnosis not having been made before death. Delfs and Eastman (1945) reviewed 17 cases of uterine rupture occurring in the absence of any uterine scar; in 23.6% the diagnosis was made post mortem. Sheehan (1948) reviewed 147 cases of obstetric shock: rupture of the uterus was found to be the cause in 20 cases.

The possibility of accidental haemorrhage causing rupture of the uterus is not considered by the majority of authors, although Powers and Gerard (1944) mention that in advanced pregnancy uterine rupture may be mistaken for premature separation of the placenta. Fraser (1954) describes two such cases in which a wrong diagnosis of concealed accidental haemorrhage was made, uterine rupture being discovered when a laparotomy was performed after the patients had failed to respond to conservative treatment. The presence of subperitoneal uterine haemorrhage following accidental haemorrhage has been noted many times. Uterine rupture may result when this process progresses a stage

further, especially in a uterus that has already been subjected to repeated caesarean sections.

A search through the index literature reveals only two cases of rupture of the uterus associated with accidental haemorrhage: Gibberd (1933) described a spontaneous complete rupture of the uterus occurring at the 28th week of a tenth pregnancy, the outcome being fatal. Gwillim (1933) described a similar case occurring at the 37th week of a second pregnancy; the patient survived. Gibberd was in no doubt that the sudden and increasing distension of the uterus was the cause of the rupture, although the multiparity of the patient and the thinness of the lower uterine segment must have been predisposing factors. No cause was found in the second case.

Case Report

A married woman aged 28 had previously had four lower-segment caesarean sections performed on account of diabetes mellitus. She was first found to have diabetes mellitus when she was 14 years old and had attended the hospital clinic for the past 11 years. The diabetic condition was well controlled on a 200-g. calorie diet and 20 units of soluble insulin morning and evening.

The first pregnancy was uneventful, lower segment caesarean section being performed at 36 weeks. The baby weighed 5 lb. 14 oz. (2.7 kg.) and died four hours after delivery, following a cyanotic attack. Post-mortem examination revealed atelectasis, but no foetal abnormalities.

Thirteen months later another caesarean section was performed at 36½ weeks. Some pre-eclampsia occurred in this pregnancy; the patient was in hospital for two weeks before delivery. The baby weighed 7 lb. 7 oz. (3.3 kg.) and caused no anxiety at any time.

The third caesarean section was performed 19 months later, at 36 weeks, following an uneventful pregnancy. The baby weighed 5 lb. 3 oz. (2.4 kg.) and died following a cyanotic attack 14 hours after delivery; no post-mortem examination was allowed.

The fourth caesarean section was performed at 36 weeks, the pregnancy having been complicated by a mild degree of pre-eclampsia. The presentation was a breech, and a type 1 placenta praevia was discovered at operation. The baby weighed 7 lb. (3.2 kg.) and at no time caused anxiety. After each operation the patient had a normal puerperium.

The present pregnancy had been uneventful except for a slight rise of blood pressure which had been noted at the clinic one week previously. By dates she was 34 weeks pregnant, there being no evidence of hydramnios.

The patient was admitted as an emergency, semi-comatose, shocked, and bleeding per vaginam. A history could not be obtained from her, but her husband gave a very full account of the happenings prior to admission. About 2 a.m. he awoke and noticed that his wife was sweating, and found that she was difficult to arouse. After taking a glucose drink she slept until 5 a.m., when again she was given a glucose drink as she was sweaty and drowsy. At 6 a.m. he gave her the usual dose of insulin. She did not have breakfast, but during the morning had two more glucose drinks. About midday he became more anxious because his wife was more drowsy. While dressing her he discovered that she was losing enough bright-red blood to produce a large stain in the bed. She did not complain of pain, but this was in all probability masked by the hypoglycaemic coma that was developing. Immediately after finding the bleeding the husband arranged for his wife to be admitted to hospital.

On admission to hospital on June 21, 1954, at 2.15 p.m., the patient was semi-comatose, extremely pale, and shocked: pulse rate 100, temperature 97° F. (36.1° C.), respirations 22, blood pressure 122/88. Abdominal palpation revealed a tense, woody-hard uterus, enlarged to the size of a 34-week pregnancy. The outline of the uterus was regular; no

bulging or tenderness was discovered over the lower segment. The lie of the foetus was longitudinal; the presentation was not identified, nor was the foetal heart heard. Some bright-red blood was seen at the vulva. A diagnosis of hypoglycaemic coma and accidental haemorrhage was made. The blood sugar was 50 mg. per 100 ml., the haemoglobin was only 52%, and a catheter specimen of urine contained albumin.

While blood was being cross-matched a transfusion of "plasmosan" was started and 10 g. of glucose was given intravenously. Because at 4 p.m. the patient was becoming more restless she was given "omnophon," 1/3 gr. (22 mg.) intramuscularly, at the same time the blood transfusion was started. Two hours later she was much improved, her pulse was stronger and slower, and she had a drink containing 20 g. of glucose. The improvement continued until 9.30 p.m., when it was noted that her pulse rate was rising, and that she was becoming restless despite having had a further injection of omnophon, 1/3 gr. (22 mg.) intramuscularly. The slight bright-red blood loss was persisting, and the uterus was felt to be a little larger, softer, and much more tender, but still of regular outline, with no excess of tenderness over the lower segment. The blood pressure at this time was 140/100. It was thought that the uterus was becoming distended with blood from continued accidental haemorrhage.

By midnight the patient had become much more restless, and her pulse had risen to 120 beats a minute. Abdominal examination revealed a completely different picture. The uterus had previously been enlarged almost up to the xiphisternum, but now it was felt as a firm mass just below the umbilicus. The abdomen was seen to be distended, and everywhere was extremely tender. A pelvic examination revealed a slight blood loss; the cervix was closed. A diagnosis of rupture of the uterus was made, and the patient was prepared for immediate operation.

Caesarean Supravaginal Hysterectomy.—Under general anaesthesia the abdomen was opened, and immediately a large quantity of fresh blood was encountered. A complete rupture across the lower uterine segment at the site of the previous caesarean sections (see photograph) had allowed the foetus and placenta to escape into the peritoneal cavity. The foetus and placenta were removed from the right epigastric region together with a large blood clot and at least a pint (570 ml.) of fresh blood. Supravaginal hysterectomy was performed. At the end of the operation the patient's condition was satisfactory: blood pressure 122/88, pulse 120.

After an uninterrupted recovery she was discharged from hospital, on the 14th day. Subsequent examination of the uterus showed extensive infiltration of the uterine wall by a blood clot at the fundus in the region of the placental

Photograph of the ruptured uterus.

attachment. Blood extended through the uterine wall into the subsera, giving the typical appearance of a Couvelaire uterus. There was a complete rupture through the lower uterine segment at the site of the previous lower-segment caesarean section operations.

Discussion

Rupture of the uterus is a comparatively rare accident, and because of this it is often not considered as a diagnosis until the patient is moribund. Gordon and Rosenthal (1943) stressed that the failure to think of rupture of the uterus as a cause for cessation of contractions, absence of foetal heart sounds, shock during labour, or unaccountable shock after delivery may result in maternal death. Daniel and Inman (1951) advised exploration of the uterus in every case of persistent bleeding or shock following any major vaginal procedure, in order to eliminate a ruptured uterus. Watt (1950) emphasized that the diagnosis of complete rupture is usually made earlier than that of the incomplete variety. Incomplete rupture may present as a spreading haematoma, which may be unnoticed if it is posterior.

Cases of uterine rupture are divisible into three main categories, due to: (1) manipulative trauma, (2) rupture of a uterine or cervical scar, and (3) spontaneous rupture. In the first group inadequate antenatal care, misdiagnosis during labour, and, finally, unwise obstetrical manoeuvres are largely responsible for the disasters. Cases of the second group mainly concern ruptured caesarean section scars, in particular those of classical and vertical lower-segment operations. A cervical tear in a previous labour may subsequently extend: Harris and Angawa (1951) report three fatalities due to high cervical tears. In the third group one finds that occasionally rupture occurs for no very good reason other than age, multiparity, and possibly dietetic deficiencies, as suggested by the experiments of Gillman *et al.* (1947), who noted that rats fed on certain deficient diets became more liable to rupture of the uterus.

Of recent years the vast improvements that have come about in the realms of anaesthesia, chemotherapy, and antibiotics have made caesarean section much safer. This has had the effect of increasing the number of caesarean operations performed, whilst at the same time tending to reduce the number of major obstetrical manoeuvres undertaken. The effects of this swing away from heroic obstetrics in favour of caesarean section has shown itself to some degree when the causation of uterine rupture is considered. Improved obstetrical practice and fewer grand multiparae have reduced the number of uterine ruptures due to traumatic interference, but the increasing number of caesarean sections being performed does counterbalance this to some degree. The incidence of rupture of the scar following the classical operation is probably in the neighbourhood of 4%, as against 0.25% for the lower-segment operation (Wetterwald, 1926; Gellé, 1932).

Although the lower-segment operation is comparatively safe, in particular with reference to uterine rupture, there is a limit to the number of pregnancies that can be safely embarked upon in a uterus that contains an incision. The danger of the scar rupturing must increase with succeeding pregnancies, and more so in a uterus subjected to repeated sections.

Just how many pregnancies should be allowed is a very debatable point. Posner *et al.* (1951) state that tubal ligation is proposed to all their patients after two previous caesarean sections. Any statistics to illustrate the safety of pregnancy or the incidence of uterine rupture following caesarean section must obviously take this aspect into consideration. The incidence of hydramnios, pre-eclampsia, and foetal abnormalities in diabetic pregnancy is manifestly greater than in other pregnancies, and these complications can all be factors in the aetiology of uterine rupture.

These facts being evident, the wisdom of allowing a diabetic patient to have repeated pregnancies terminating in caesarean section must be doubted, as the risk of uterine rupture must be increased.

In the present case the fifth pregnancy was allowed to continue as the patient had only two living children from her four previous pregnancies, all the sections having been performed at the 36th week under ideal conditions and all the post-operative periods having been uneventful.

Reading through the literature one is impressed by the great variation in the way in which these cases present. Equally impressive is the manner in which some cases present with the minimum of symptoms and have been known to walk into hospital (Kerr and Moir, 1949). Walker (1950) described a case of lithopaedion formation following expulsion of the foetus into the peritoneal cavity after rupture of the uterus in labour.

After an accidental haemorrhage of even a small amount the general condition of the patient is usually poor. The case described presented just such a picture, complicated by hypoglycaemic coma. In hospital, with a blood transfusion and under sedation, the patient improved, and only some hours later, when her general condition was noticed to be deteriorating, was it discovered that the uterus had ruptured, there being no dramatic change in her condition.

Summary

A case of diabetic pregnancy complicated by accidental haemorrhage associated with spontaneous rupture of the uterus is described.

The difficulties of diagnosis are noted, with particular reference to that of distinguishing between accidental haemorrhage and uterine rupture.

Accidental haemorrhage is mentioned as being an aetiological factor in the causation of rupture of the uterus.

The diagnosis of shock following delivery is a dangerous one to make and should not be entertained without first exploring the lower uterine segment.

The correct diagnosis of uterine rupture will be made more often and more rapidly if the condition is kept in mind.

The danger of repeated caesarean section is discussed, with special reference to added complications of the diabetic pregnancy.

Early diagnosis, timely blood transfusion, prompt surgery, skilled anaesthesia, and the use of antibiotics should make the condition become less fatal than it is at the present time.

With the wider use of caesarean section, rupture of the uterus following internal version should become a great rarity.

The comparative rarity of grand multiparae, and the fewer classical caesarean sections that are performed, should reduce the incidence of uterine rupture.

I am indebted to Mr. J. H. Peel for permission to publish this case, and to Mr. W. Smith, of the photographic department of King's College Hospital, for the photograph.

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