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CAESAREAN SECTION SCAR SAFETY

BY

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After a caesarean section, the question "Once or always?" still remains most pertinent, the answer to which verges on gambling. There is no clinical test for the estimation of scar safety in a subsequent pregnancy or labour short of the practical experience. Radiology is capable of providing guidance in some cases.

A recent specimen of a uterus previously subjected to a lower-segment caesarean section and later removed because of fibroids shows quite clearly the typical appearance of a healed transverse incision in the lower segment (Fig. 1). On opening all uteri which have previously been subjected to lower-segment caesarean section a larger or smaller depressed scar will always be seen. That this scar is at the site of the previous caesarean section can be confirmed by inspecting Fig. 2. A hystero-gram prior to opening this particular uterus shows a sound in the external scar and the typical internal depression at the same level (Fig. 3). Histopathological evidence (Fig. 4) adds further confirmation

FIG. 1.—Arrow points to the typically seen scar of a previous lower-segment caesarean section.

in that this thinned area, with both an external and an internal depression, consists entirely of fibrous tissue.

Now this finding, far from being unusual, is present after all lower-segment caesarean sections. Poidevin and Bockner (1958) previously reported the results of a

FIG. 2.—Arrow points to the external scar of a previous lower-segment caesarean section shown at the same level as the internal scar.

FIG. 3.—Hystero-gram of same uterus, as in Fig. 2, before opening. The sound is in the external scar, and the internal depression is shown by a typical wedge-type deformity.

FIG. 4.—Section of scar area from same uterus as in Figs. 2 and 3.

hystero-graphic study of uteri after caesarean sections and compared the findings between 13 control subjects who had vaginal deliveries and 43 who had caesarean section deliveries. The lateral x-ray view was used, and is again stressed because of the position of the lower-segment wound, which would not be expected to show up in an antero-posterior view. All the control cases showed a smooth anterior lower-uterine outline (Fig. 5). All the 43 section cases showed some deformity. The deformities usually took the form of a wedge depression (Fig. 6), but occasionally a larger irregular deficiency was shown (Figs. 7 and 8).

Since this report, further cases have been examined hystero-graphically by Bockner, and again all previously sectioned uteri showed some deformity. (All figures used here are from the new series.)

The only other reported work on this subject that a previous search revealed was that of Baker (1955). His study concentrated on a digital palpation of the previous section area immediately following subsequent vaginal delivery, and found that 53 out of 64 examined had sound scars. In 24 he carried out hystero-graphs, but was able to demonstrate a deformity in the lower anterior uterine wall in only five cases.

Significance of the Deformity

It is not permissible to label all these deformities as defects. There is no proof that they are all defective. In fact, there is much proof to the contrary in the abundant clinical evidence of subsequent safe vaginal delivery in many cases. Further investigations and follow-up of our cases tend to confirm the view that the small wedge deformities—that is, those of 5 mm. or less in depth—are those on which reliance can be placed. On many occasions now, at repeat caesarean section in such cases, no deformity has been found in the lower segment except the white scar line. No doubt as the lower segment is stretched out in late pregnancy over the enlarging presenting part this deformity, involving scar tissue, is pulled out and holds quite well.

One of the cases used in the previous report showed an irregular deformity high in the cervix. The patient subsequently passed uneventfully through an easy vaginal delivery, and then in the next labour suffered an extensive rupture from the site of this deformity in the cervix. The details of this case form the subject of another publication at present being prepared by Bockner.

FIG. 5.—Hystero-graph six months after a normal vaginal delivery. Note smooth contour of anterior uterine and cervical walls.

In the search for the real significance of these x-ray findings this case seems to emphasize that deformities, other than the acceptable wedge type, are to be distrusted, and in the present state of our knowledge should serve as an indication for repeat caesarean sections. Further, in these irregular deformities the integrity of the scar should be suspect during the whole pregnancy.

FIG. 6.—Hystero-graph nine months after a lower-segment caesarean section. Note wedge-type deformity on lower anterior uterine wall at site of scar. This is a typical finding.

FIG. 7.—Hystero-graph 11 months after lower-segment caesarean section. Note irregular deformity at site of scar.

FIG. 8.—Same uterus as in Fig. 7, showing antero-posterior view. This demonstrates a lateral pouching.

Suggested Reasons for the Uterine Deformity

The lower uterine segment is composed of smooth muscle, and this tissue is known to heal by scar-tissue union (Schwarz *et al.*, 1938 ; Siegel, 1952). The original length of the uterine wound is approximately 3 in. (7.5 cm.) at the time of its surgical suture. Within the next few days scar tissue is laid down by fibroblastic activity, while concurrently a gradual and relatively rapid shrinkage of the whole uterus is occurring. Thus the scar tissue which sets out to heal a 3-in. (7.5-cm.) long wound is reduced, after a few weeks, to a $\frac{1}{2}$ -in. (1.3-cm.) long wound. This is an extraordinary situation which is found only in such an organ as the rapidly involuting uterus. Thus some redundancy of scar tissue is understandable in the resting state. Also, the union is by scar tissue rather than muscle regeneration, so that a narrowing of this part of the uterine wall (Fig. 4) is understandable. The wedge-shaped deformity is thus explained.

The larger and more irregular defects are most likely due to an initial failure of scar-tissue union. This could follow a technical defect, such as the giving way of the suture material, or the failure to close completely a torn angle wound or even haematoma formation. Could it also possibly be related to a residual inherent hypercorticism of pregnancy ?

Defective union of wounds, due to a failure of fibroblastic reaction, is well known in such conditions as Cushing's syndrome and during cortisone or corticotrophin administration, both experimentally in animals and therapeutically in humans (Creditor *et al.*, 1950 ; Alrich *et al.*, 1951 ; Baxter *et al.*, 1951 ; Conway and Stark, 1951). In all these conditions we are able to attribute the failure of wound healing directly to the increased amount of circulating corticosteroids.

Pregnancy itself is another condition of marked adrenal cortical hyperactivity leading to increased amounts of circulating corticosteroids (Venning, 1946 ; Gemzell, 1953 ; Robinson *et al.*, 1955 ; Jailer, 1956 ; Gold, 1957 ; McKay *et al.*, 1957). This rise in steroid blood levels persists into the puerperium for a questionable time, Robinson *et al.* (1955) stating that six weeks is required before a return to normal levels is achieved. Could it be that, in some women, this inherent hypercorticism of pregnancy, which continues into the puerperium, causes a failure of fibroblastic reaction and thus poor healing of caesarean section wounds ?

Practical Considerations

The problem obviously is to find a measure by which the integrity of each caesarean section scar can be judged. In the absence of such a measure there are those who may legitimately believe and practise the old dictum, "Once a caesarean section, always a caesarean section." This should avoid any possibility of uterine rupture in labour, but will not prevent the rare rupture during pregnancy.

On the other hand, there are those who would argue that uterine rupture is so rare after the modern lower-uterine-segment operation that repeat caesarean section is unjustifiable when the original indication does not recur. They support their argument with statistics. This line of argument was suggested in the previous report (Poidevin and Bockner, 1958) when in the preceding five-year period, during 17,000 deliveries, there had not been a case of uterine rupture. Of 216

women who had previous caesarean sections, 83 were selected for subsequent vaginal delivery and safely conducted through, while 133 had repeat sections. Since then there have been two cases of uterine rupture in the clinic service of our teaching hospitals, both having had previous caesarean sections. One was the case referred to previously with the grossly deformed upper cervix on hystero-graphy, the other an emergency admission on whom no hystero-graphy had been done. Thus the statistics have changed from no cases of rupture in 17,000 deliveries to 2 cases in 21,000 deliveries.

There are many reports of scar weaknesses and even scar ruptures found at the time of elective repeat caesarean sections ; conversely, there are many reports of large numbers of cases safely conducted through vaginal deliveries subsequent to caesarean sections. What, therefore, is the correct scientific approach to the individual under one's own care ?

I believe most of us who have been satisfied with the technical details and subsequent post-operative course of the previous caesarean section would trust that wound to a future vaginal delivery. In spite of this faith, however, some large defects have been shown by hystero-graphy in a series of cases that were each the responsibility of a specialist obstetrician.

X-ray evidence has been demonstrated as a means of visualizing the deformity in the uterus after caesarean section. From this an opinion can be given on whether the deformity is within the normal acceptable wedge type range or whether it is an irregular pouching.

We therefore have the alternatives of firstly judging the safety of caesarean section scars after an appraisal of the details of the previous operation and puerperium, or, secondly, of requesting any x-ray evidence of scar weakness as judged by an irregular deformity. Reliance on the former has been found insecure on occasion, whilst the small amount of x-ray evidence to date suggests that it may be safe to rely on the integrity of the commonly seen wedge deformity but not on large irregular ones. Both methods should be used in cases of doubt even if not practicable in all cases. So often new cases coming under one's care are already pregnant, giving one no opportunity to use hystero-graphy. However, in the non-pregnant state, when evidence of scar safety is sought, hystero-graphy has a definite place.

Conclusion

Hystero-graphy is an advance in our search for evidence of the safety of lower-segment caesarean section scars. Experience strongly suggests that the usually seen wedge-shaped deformities may be considered within acceptable limits of normal healing, and most reliance can be placed in them. The irregular deformities, of any size, should be suspect, and indicate subsequent caesarean section rather than vaginal delivery.

It is suggested that the inherent raised blood corticosteroid levels of pregnancy may play a part in defective union of caesarean section wounds of the uterus, although technical failures must accept the major blame.

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DIABETES INSIPIDUS ASSOCIATED WITH TUBERCULOUS MENINGITIS

BY

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Diabetes insipidus is a rare syndrome of water-balance disturbance characterized by the excretion of excessive quantities of urine with resultant polydipsia.

Apart from heredity, the common causes of diabetes insipidus are trauma, encephalitis, brain tumours (such as gliomata, craniopharyngiomata, meningiomata, or chromophobe adenomata of the pituitary gland), syphilis, Hand-Schüller-Christian disease or xanthoma, and cerebrovascular accidents (Fink, 1926, 1928; Warkany and Mitchell, 1939; Biggart, 1935; Wyllie, 1943; Jones, 1944; Soffer, 1956). Meningitis is an extremely rare cause. In Fink's (1928) series tuberculoma and tuberculous meningitis comprised 5% of cases, with non-syphilitic inflammatory conditions 9%. Soffer (1956), in a review of 160 cases collected from the literature, states that inflammatory diseases (that is, encephalitis, meningitis, tuberculosis, and syphilis) accounted for 31 cases (20%). Jones (1944), observing 42 cases, mentions only one associated with meningitis; and Hagenbach (1883) reported one case of diabetes insipidus resulting from tuberculous meningitis. Healy (1935) gave an account of a case occurring in a child of 2½ years with miliary tuberculosis and basal meningitis. Acheson and Smith (1958) described a case of tuberculous meningitis in which diabetes insipidus occurred five years after the onset of the infection. Lorber (1958) reported a similar association, and briefly reviewed 11 other cases which appeared mainly in the European literature.

In view of the rare association of diabetes insipidus with meningitis, a further case is here presented.

Case Report

A 5-year-old Bantu girl was admitted to the paediatric unit, Coronation Hospital, on October 12, 1956, with the complaint of headache for one month, a painful and stiff neck for three weeks, prominence of the eyes, and thirst and polyuria associated with marked weight loss. There had been no previous illnesses and the family history was non-contributory. The child's developmental history was normal.

Examination revealed a thin, undernourished child lying curled up in bed, very irritable and uncooperative. Stiffness of neck and back was present, and a marked proptosis of both eyes with bilateral sixth-nerve palsies was noted. Fundoscopic examination revealed temporal pallor of both

disks. Reflexes were equal but accentuated, and the plantar response was normal on both sides. Tone was normal. The pulse rate was 110, respirations 28, temperature 99° F. (37.2° C.), and blood pressure 100/60 mm. Hg.

The cerebrospinal fluid showed numerous pus cells and lymphocytes, but no bacteria were detected on direct or cultural examination; protein, 230 mg./100 ml.; chloride (as NaCl), 640 mg./100 ml.; sugar, 27 mg./100 ml. The leucocyte count was 11,000/c.mm. Urinalysis was normal. Patch test for tuberculosis was negative (activity equal to 0.0001 ml. (0.1 mg.) of Old Tuberculin). Radiological examination of the heart and skull showed nothing abnormal; the pituitary fossa and sinuses were normal; the lung films showed hilar adenopathy with perifocal pneumonia (Fig. 1). While awaiting the cultural examination of the C.S.F. the patient was treated with soluble penicillin, 250,000 units, and sulphadiazine, 0.5 g. every six hours.

She remained febrile, with temperatures rising to 102° F. (38.9° C.). Polyuria and polydipsia continued, with daily variation in quantity. Re-examination of the C.S.F. showed 60 polymorphonuclears and 45 lymphocytes per c.mm. and protein 1,700 mg./100 ml., with insufficient fluid for sugar and chloride estimations. As the patient's condition had deteriorated, with persistence of the pyrexia, sulphadiazine was stopped on October 19 and tetracycline, 62.5 mg., was given every six hours. Thereafter the proptosis receded and the temperature started to settle. The C.S.F., however, showed no essential change.

On November 11 a Mantoux test with purified protein derivative (second strength) was positive and treatment was altered to intramuscular streptomycin sulphate, 0.5 g. daily, and oral isoniazid, 0.1 g. three times daily. Thereafter the child showed steady improvement, with gradual return of the C.S.F. to normal. The bilateral sixth-nerve palsies disappeared, but the polyuria and polydipsia continued. On December 8 the patient became dehydrated and required intravenous fluid therapy. Aqueous "pitressin," 0.25 ml. eight-hourly, was then started with dramatic decrease in her fluid intake and urinary output, which fell to near-normal values. She rapidly began to gain weight. Aqueous pitressin was changed to pitressin tannate in oil, 2.5 units daily, on January 16, 1957. She was finally stabilized on alternating daily doses of 2.5 and 5 units. Pitressin therapy was discontinued on March 6, 1957. The fluid intake and urinary output immediately rose.

Further Investigations

Weight 32½ lb. (14.6 kg.); height 38 in. (96.5 cm.); span 39 in. (99 cm.); lower segment 18 in. (45.5 cm.), ratio upper to lower segments 1:0.9; head circumference 20 in. (51 cm.); chest circumference 21½ in. (54.5 cm.); abdominal circumference 23½ in. (59.5 cm.). Blood urea, 25 mg./100 ml.; plasma chloride, 103 mEq/l.; CO₂ combining power, 21 mEq/l.; serum calcium, 5 mEq/l.; serum sodium,

FIG. 1.—Radiograph of chest (right lateral) on October 12, 1956, showing hilar adenopathy and perifocal pneumonia in the right lower lobe.