Summary

Of 2028 pregnant women, 5·5% were found to have bacilluria. Acute pyelonephritis developed during pregnancy in 19 (36%) of 52 women with untreated bacilluria, but in only 3 (5%) of 57 women with treated bacilluria. Of 1916 women who had sterile urine early in pregnancy, only 9 (0·4%) had acute pyelonephritis later.

These results confirm that pyelonephritis of pregnancy is usually preventable.

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SURGICAL TREATMENT OF EXTRAHEPATIC PORTAL HYPERTENSION IN YOUNG PATIENTS

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EXTRAHEPATIC portal hypertension chiefly affects children and young adults, and often gives rise to severe hæmorrhages from œsophageal varices. Conservative treatment consists in the insertion of a Sengstaken tube (if this is feasible) and blood-transfusion. Surgical treatment may take the form of portacaval anastomosis, ligation of the œsophageal varices, or resection of the œsophagus. In most cases, extrahepatic portal hypertension is caused by thrombosis of the portal vein. Portacaval anastomosis is effective only when the thrombosis is confined to the peripheral portion of the portal vein. Mostly, however, the portal vein is entirely obstructed by the thrombosis and surrounded by a cluster of thin-walled collateral vessels, unsuitable for anastomosis.

Shaldon and Sherlock (1962) reported their discouraging experience in these cases with procedures other than portacaval anastomosis. If portacaval anastomosis is impossible, they believe that conservative treatment is to be preferred to splenorenal anastomosis. They attached relatively little importance to the risk of fatal hæmorrhage, because none of their patients died as a result of bleeding. On the other hand, Milnes Walker (1962) believed that the risk of hæmorrhage was grave, and that a child under five years, bleeding from œsophageal varices, was unlikely to reach adulthood without surgical intervention: in his first series of 28 patients there were 7 deaths from hæmorrhage. Hallenbeck and Adson (1961) reported favourable results from splenorenal anastomosis, but took the view that in children under eight years of age the splenic vein was still too small for establishing an adequate anastomosis.

In view of these reports we have reviewed our own series of patients treated by procedures other than portacaval anastomosis.

Patients and Methods

17 young patients with extrahepatic portal hypertension were treated in the Utrecht University surgical clinic: 11 of them had a history of having undergone splenectomy. Splenoportography had been performed during laparotomy in the remaining 6 patients. A splenorenal anastomosis was found possible in 5 of these; in 1 this was impossible owing to thrombosis of the splenic vein. Since the portal vein and the superior mesenteric vein were also thrombosed, portacaval or mesenterico-caval anastomosis was likewise impossible; consequently the operation was confined to a splenectomy. Unfortunately, the œsophageal varices were not ligated. During the five subsequent years this patient repeatedly needed blood-transfusions to control severe hæmorrhages. The other 5 patients underwent splenorenal anastomosis; in 3, the œsophageal varices were ligated at the same time.

The operative approach was a thoracolaparotomy in the tenth intercostal space. After splenectomy, an end-to-side anastomosis was first performed. The splenic vein was cut obliquely to ensure a wide anastomosis. The œsophagus was then opened, working through the same incision, and groups of varices (as a rule three) were treated by means of multiple-site ligation (Boerema 1949). The œsophagus was closed with three rows of knotted linen sutures, and a nasal tube remained in situ for five days.

Results

Of these 5 patients only 1 had severe recurrent hæmorrhage a year after the operation. Although the operation had included ligation of the œsophageal varices, re-operation revealed two groups of varices in the œsophagus. The pressure in the superior mesenteric vein was barely increased (17 cm. water), and certainly remained below the 30 cm. indicated as the critical pressure in the portal system for the occurrence of hæmorrhage. No dilated veins were found below the diaphragm. We concluded that the ligation of varices had been incomplete, and that the child had bled despite an adequately functioning splenorenal anastomosis. The remaining varices were ligated, and there was no further bleeding during the next seven months. The other 4 patients have not bled for periods of one, three, five, and seven years, respectively. Consequently we are so satisfied with the results of splenorenal anastomosis that we intend to continue with this.

Much greater difficulties were encountered in treating the 11 previously splenectomised patients. A splenorenal anastomosis is impossible in these cases, because the splenic vein usually becomes thrombosed after splenectomy. Nor was portacaval anastomosis feasible in these cases, because the entire portal vein was thrombosed. In such cases, the operation described by Santy and Marion (1953) is resorted to in the Utrecht surgical clinic. The vena cava is ligated above the bifurcation and severed; it is then anastomosed end-to-side with the superior mesenteric vein. This procedure was used in 5 patients; in 2 of these, the œsophageal varices were ligated at the same time and they have not bled for four and five years respectively. All 3 patients who underwent only mesentericocaval anastomosis bled after a year: 2 therefore had their varices ligated a second time and have had no further hæmorrhage for four years. The 3rd of these patients was conservatively treated (elsewhere), and three years later, despite blood-transfusions, succumbed to a fatal hæmorrhage.

In only 2 patients could portacaval anastomosis be carried out, and their varices were ligated at the same time: 1 patient died as a result of leakage of the œsophageal suture; the other has remained free of hæmorrhage for two years.

In 1 patient the superior mesenteric vein was anastomosed to the left ovarian vein. In the seven years since then, the patient has bled slightly only when she was pregnant.

The last 3 patients in this group were treated exclusively by means of ligation of the œsophageal varices. 1 was a woman with severe hæmolytic jaundice which had required corticosteroid therapy after splenectomy: another hæmolytic crisis set in postoperatively; in addition severe varicella developed and the patient died. The 2 other patients subsequently had a number of mild hæmorrhages. Some of these cases were reported by Zaayer (1957).

Summary and Conclusions

In 11 patients with extrahepatic portal hypertension who had undergone splenectomy there were 2 postoperative deaths, of which one was a direct consequence of the operation. 1 patient died of hæmorrhage at a later date.

Judged by our experience with 17 patients an adequate portacaval or mesenterico-caval anastomosis, combined with ligation of œsophageal varices provides adequate protection against hæmorrhages; the same applies to the patients treated by means of splenorenal anastomosis. Patients treated exclusively by ligation of œsophageal varices, or by anastomosis not combined with ligation of varices, invariably had more or less severe recurrent hæmorrhages in subsequent years.

In patients with extrahepatic portal hypertension without a history of previous splenectomy, splenorenal anastomosis combined with ligation of varices prevents recurrent hæmorrhages.

After successful anastomosis, even when combined with ligation of varices, there can still be bleeding from remaining varices.

In previously splenectomised patients, mesenterico-caval anastomosis (or sometimes portacaval anastomosis), combined with ligation of varices, prevents recurrent hæmorrhages.

Ligation of varices alone affords no protection against recurrent hæmorrhages.

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" . . . One of the most puzzling problems of pregnancy is why the fœtus is not immunologically rejected. The tissues of the fœtus are foreign to the mother in that they contain some of the genetic material from the father. Skin, for example, taken from a daughter and grafted on to her mother would, after about fourteen days, wither and be sloughed off by the process known as the homograft reaction. Yet during pregnancy these self-same tissues were tolerated for nine months, or in the case of the mouse, for 19 days, without showing any signs of immunological attack. Such an attack should, of course, be manifest in the placenta, where fœtal and maternal tissue come into intimate contact with one another. Many hypotheses have been put forward to account for the immunological success of the fœtus but all have failed to withstand experimental verification."—Dr. D. R. S. KIRBY. *New Scientist*, March 4, 1965, p. 563.

HÆMODIALYSIS OF THREE CHILDREN AND ONE INFANT WITH A HÆMOLYTIC-URÆMIC SYNDROME

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IN 1955 Gasser et al. first described a syndrome characterised by severe hæmolytic anæmia with the appearance of abnormal red cells in the peripheral blood, thrombocytopenia, central-nervous-system manifestations, and renal-cortical necrosis with uræmia. The syndrome is not always severe enough to require other than conservative therapy (Aherne 1957, Allison 1957). In the past year four patients in whom conservative therapy had proved inadequate were referred to the regional artificial kidney unit for hæmodialysis. All were anuric on admission. Twelve dialyses were performed. Three of the patients died; but the fourth, an infant of 6 months, is alive and well, though hypertensive, 9 months later. We report these four cases because young children present special problems during dialysis.

Case-reports

Case 1

This was a boy of 5 who had diabetes mellitus for which he had been receiving soluble insulin for 2 years. For 3 days he had had diarrhœa, vomiting, and dark urine. On admission to hospital the urine was scanty and contained blood and albumin. There was mental confusion, which eventually progressed to unconsciousness. The blood-urea rose steadily, being recorded elsewhere as 900 mg. per 100 ml. The blood-sugar was satisfactorily controlled throughout the illness by soluble insulin. On the 11th day of his illness the child was transferred to the artificial kidney unit.

On arrival at the unit the child was deeply unconscious, pale, and twitching, with frequent hiccup. Hydration was normal, blood-pressure was 130/85 mm. Hg, and temperature 98·2°F.

Laboratory investigations (*fig. 1*).—Blood-urea 640 mg. per 100 ml., serum-potassium 5·0 mEq., sodium 147 mEq., chlorine 81 mEq., alkali reserve 24·4 mEq., blood-sugar 312 mg. per 100 ml., hæmoglobin 5·7 g. per 100 ml., platelets 70,000 per c.mm., white cells 20,000 per c.mm. (polymorphs 62%, lymphocytes 31%, monocytes 7%), reticulocytes 16%. Blood-film showed anisocytosis and polychromasia.

The first dialysis on the 11th day lasted 2 hours and reduced the blood-urea to 225 mg. per 100 ml. Irregular left-sided convulsions, which later became generalised, started after dialysis. These were partially controlled by 5% calcium gluconate, 10 ml. intravenously. The urine output remained low, and the blood-urea rose, so that a second dialysis was required 4 days later. Two more dialyses were performed, but there was no clinical improvement. Gradually he assumed a decerebrate posture, the urine output remaining negligible. He died on the 32nd day.

At necropsy immediately after death the brain was grossly œdematous, with widespread cortical softening. Macroscopically the kidneys showed confluent cortical necrosis.

Case 2

This girl of 14 months had mild diarrhœa and vomiting from which recovery was apparently complete. But 2 days later her general condition deteriorated. On admission to hospital frequent epileptiform convulsions were observed, and

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