A group of patients with severe mitral stenosis but with multiple features has been analysed separately. Inevitably the operative risk was greater and the prospects of obtaining a good result were poorer, but the majority maintained worth-while improvement for some years. A subgroup with considerable cardiac enlargement, congestive failure, and atrial fibrillation were thought to have important myocardial damage and fared less well.

Some degree of traumatic (operative) mitral incompetence, as judged by auscultation, was produced in 97 (20%) of cases. In 72 of these there is nothing to suggest that the systolic murmur reflects mitral incompetence of dynamic significance. In 14 patients (2.8%) of the whole series) it is thought that the incompetence produced was responsible for death or for subsequent deterioration, and in another 11 (2.2%) it is a possible contributory cause; but other adverse factors are also present which could be responsible. Since the introduction of the transventricular dilator there has been a higher incidence of systolic murmurs produced but not of traumatic incompetence of dynamic significance.

Clot was present in 107 of the 500 patients and systemic embolism occurred in 20% of those with clot. Clot is most likely to be present in those with atrial fibrillation, large hearts, calcified valves, and relatively advanced years. Embolism with sinus rhythm is rare but occurred in 10% of all those with atrial fibrillation. This incidence has fallen during the past five years. Subsequent embolism has occurred in 16 patients, but it is not yet possible to assess if this would have been greater without valvotomy.

It is concluded from this analysis that, although most patients with severe mitral stenosis are improved by valvotomy, surgical treatment is but an incident in the relentless progress of rheumatic heart disease, whether from activity of the rheumatic process or from the progressive fibrosis which follows activity.

The great majority of these patients were operated upon by Mr. Andrew Logan, to whom we are also indebted for the relevant data. We are also grateful to his colleagues, Mr. D. Wade, Mr. R. J. M. McCormack, Mr. P. Walbaum, and Mr. B. T. Le Roux, and to Dr. Arthur Kitchin, who has taken part in many of the assessments and contributed much to the haemodynamic studies. Our patients have been referred from many parts of Scotland and England, and we are therefore grateful to too many physicians for individual acknowledgment. Miss Sheila Crawford and Miss Ann Malcolm have recorded most of the electrocardiograms. Mr. Raymond Allford and Mr. J. M. Ramsay have been in charge of the cardiac laboratory. Sisters Catherine MacGregor, Janet Milligan, and Elizabeth Scott have been responsible for nursing all these patients. Finally, and by no means least, we are deeply grateful to Miss Joyce Cochrane for unbelievably hard work and patience in dealing with so many notes.

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HIGH INTESTINAL OBSTRUCTION PRESENTING AS ACUTE RENAL **FAILURE**

RY

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Acute obstruction high in the small intestine produces early nausea and vomiting, severe fluid loss, marked imbalance of the body electrolytes, and minimal abdominal distension. Two such cases were referred to the Liverpool Renal Insufficiency Centre with the diagnosis of "uraemic vomiting" for consideration with a view to extracorporeal haemodialysis with the artificial kidney. Both patients were six to seven months pregnant when the vomiting started and miscarriage occurred spontaneously in each case, two weeks after the onset of the vomiting. Initially the diagnosis of intestinal obstruction had been thought unlikely as there was no abdominal distension, but as copious biliary vomiting persisted after the miscarriages, and oliguria was associated with a progressively increasing blood urea, transfer to the renal insufficiency centre was advised. Ultimately the symptoms in both cases proved to be due to a high intestinal obstruction situated in the jejunum, 3 to 4 in (7.5 to 10 cm.) below the duodenojejunal flexure.

On admission to the renal insufficiency centre both patients showed evidence of severe dehydration associated with a profound disturbance of the blood electrolytes. As a first measure of therapy they were placed on a standard basic regime (Ross et al., 1961) and the fluid and electrolyte deficiency corrected by intravenous infusion of electrolyte solutions.

Case 1

A woman aged 34 began to vomit all she ate during the seventh month of her eighth pregnancy. Prior to this she had maintained perfect health and the previous seven pregnancies had been completely normal. Two weeks after the vomiting began she was delivered of a stillborn infant. However, the copious biliary vomiting persisted and two weeks later she became confused and was transferred to Sefton General Hospital. Intravenous therapy was initiated for a state of gross dehydration which was almost immediately followed by a diuresis. The blood-urea level fell rapidly (Fig. 1) and it was possible to improve the condition of the patient sufficiently to carry out a laparotomy three days later. Prior to the laparotomy it had been possible to demonstrate the site of the obstruction radiologically by injecting 15 ml. of 60% urographin down the Ryle tube, thus outlining the stomach, duodenum, and jejunum up to the site of the obstruction (Fig. 2).

The operation was performed through a left paramedian incision and the cause of the obstruction was found to be due to an intussusception in the jejunum about 4 in. (10 cm.) from the duodeno-jejunal flexure. At the apex of the intussusception was situated a primary carcinoma encircling the small intestine and producing a ring stricture. The growth had not metastasized and it was possible to resect the intussusception with the neoplasm, restoring the continuity of the small intestine by end-to-end anastomosis. Microscopical section of this 2-in. (5-cm.) diameter tumour showed a fairly well differentiated columnar-celled adenocarcinoma.

The patient has been followed up for six months and remains perfectly well with no evidence of any recurrence of the growth.

Case 2

A woman aged 26 had previously been delivered of two normal children. Two years prior to the present event a transperitoneal division of the isthmus connecting "sigma kidneys" had been performed. The upper pole of the right kidney had been attached to the lower pole of the left kidney. Copious biliary vomiting began during the sixth month of her third pregnancy and a diuresis did not follow on the institution of intravenous therapy. Ten days after admission to hospital she was delivered of a living infant which survived only a few days.

A large volume of bile-stained fluid continued to be aspirated from a Ryle tube and a diuresis did not follow the continuation of intravenous therapy. Fluid administration

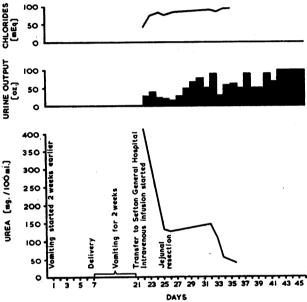


Fig. 1.—Blood urea, serum chlorides, and urinary output in Case 1.

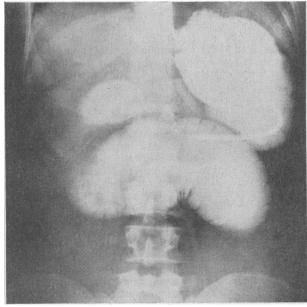


Fig. 2.—Case 1. Urographin outline showing jejunal obstruction.

by this route could be administered only to the extent of restoring existing water depletion; further correction of electrolyte imbalance, without the danger of overhydration, could then be effected only by extracorporeal haemodialysis with the artificial kidney (Fig. 3). During the third hour of this first dialysis the patient became hypotensive owing to a state of hypovolaemia, the blood-pressure being restored by the transfusion of a pint (570 ml.) of packed red cells. It was thought that the patient had been slightly hypovolaemic at the start of the dialysis and that the circulating fluid volume had been further depleted by ultrafiltration with the Skeggs and Leonards (1948) artificial kidney.

A noticeable improvement in the general condition of the patient followed this six-hour dialysis with the artificial kidney but three days later she complained of sudden severe abdominal pain with generalized guarding of the abdominal musculature. An immediate laparotomy demonstrated a band obstruction of the jejunum 4 in. (10 cm.) below the duodeno-jejunal flexure. Above the obstruction was a small perforation of the distended jejunum producing a generalized peritonitis. The peritoneal cavity was cleaned and the necrosed area of bowel with the perforation resected, the continuity of the small intestine being restored by end-to-end anastomosis. After this operation oliguria persisted, and eight days later the electrolyte imbalance and condition of the patient had to be improved once again by extracorporeal haemodialysis with the artificial kidney. Five days after the second and uneventful dialysis diuresis ensued, with ultimate complete recovery.

Discussion

In spite of the rarity with which obstruction occurs in the jejunum, it arises with sufficient frequency for the differential diagnosis and overall management to be considered. Initially the diagnosis in these two cases rested between vomiting due to toxaemia of pregnancy, vomiting secondary to renal failure, and high intestinal obstruction. Toxaemia of pregnancy was excluded when the vomiting persisted after the miscarriages, and in Case 1 renal failure was eliminated when a diuresis followed replacement of the fluid and electrolyte deficiency. A diagnosis of high intestinal obstruction was possible in Case 2 when a perforation occurred as

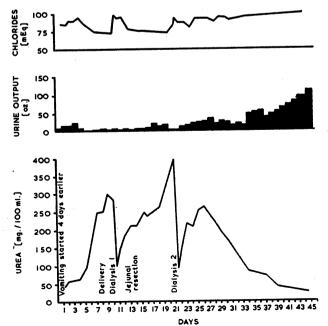


Fig. 3.—Blood urea, serum chlorides, and urinary output in Case 2.

a complication of the obstruction. If there is any question of a high intestinal obstruction the diagnosis may be confirmed by inserting 15 ml. of 60% urographin down a Ryle tube (Case 1, Fig. 2).

The sudden onset of symptoms occurring with obstruction of the jejunum is usually due to an associated perforation, to the development of an intussusception, or if there is a sudden occlusion of the lumen of the bowel. The two cases described above demonstrate the first two methods of onset of symptoms.

The copious vomiting produced by obstruction high in the intestinal tract causes a rapid alteration in the normal fluid and electrolyte balance, and the subsequent salt-and-water depletion may result in renal failure. This must be distinguished from the sodium, chloride, and water deficiency due to renal failure (de Wardener, 1958). When salt deficiency is secondary to renal failure, the urinary specific gravity is around 1010, whereas if the kidneys were previously normal tubular function will remain normal and the specific gravity will at first be greater than 1020. If, however, the vasoconstriction persists and acute renal failure develops, the specific gravity will fall towards 1010. In chronic pyelonephritis or glomerulonephritis the syndrome is sometimes known as "salt-losing nephritis."

In Case 1 spontaneous recovery of renal function returned with the institution of an intravenous infusion replacing the fluid and electrolyte deficiency. In Case 2 oliguria persisted after the fluid deficiency had been replaced by an intravenous infusion and continued for a total of 28 days. This indicated that the renal failure was not simply secondary to the intestinal obstruction, but that there was also primary intrinsic renal parenchymal damage. In this case it was possible by the aid of two dialyses with the artificial kidney, each of six hours' duration, to restore the electrolyte imbalance to approximately normal levels until renal function returned spontaneously. Haemodialysis also made it possible to deal with the perforation and high intestinal obstruction safely by operation during the period of uraemia.

Laparotomy with intestinal resection was carried out in both these cases only after the fluid and electrolyte imbalance had been corrected as well as possible. This may be done by simple intravenous therapy, but if there is any associated renal damage extracorporeal haemodialysis should be considered. A review of 27 years' experience with jejunal neoplasm dealt with by operation has put the mortality for such cases at 16% (Pridgen et al., 1950), and it would seem that by earlier diagnosis and the modern methods available to correct fluid and electrolyte imbalance the mortality figure should be greatly reduced.

Summary

Two case of jejunal obstruction are described. The first presented as an intussusception caused by a primary carcinoma and the other as a perforation of the distended jejunum above a band adhesion.

Both patients showed severe fluid and electrolyte imbalance, which had to be corrected before it was possible to carry out successful resection of the obstructions. Intravenous therapy alone was sufficient in one case, but as the other was associated with intrinsic renal damage it was necessary to correct the electrolyte imbalance on two occasions by dialysis with the artificial kidney.

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A WEIGHING-BED: ITS CONSTRUCTION AND USE IN HAEMODIALYSIS

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The connexion of the vascular system of the organism to an extracorporeal circuit, with the inclusion of a dialysing membrane for the purpose of haemodialysis, invokes a particular chain of events which affect the distribution and volume of the body fluids.

There may be an imbalance in the outflow and inflow rates of blood, resulting in hypervolaemic or hypovolaemic states. The patient may be bleeding either as a result of uraemia or as a consequence of systemic heparinization. The added volume of the extracorporeal circuit is countered by prior priming with bank blood. There are, however, two specific factors which have to be taken into account in the use of the disposal twin-coil Kolff kidney. The first is the capacity of the coil itself, and the second that of ultrafiltration.

When the machine is primed the coil is filled with a volume of blood which is the static volume. In connecting the patient to the extracorporeal circuit the coil distends. This increase in capacity (dynamic volume) results in a depletion of the patient's blood volume, and is manifested by a drop in blood-pressure. This initial hypotension is a common occurrence, and it is important to correct and anticipate its development. Blood can be transfused into the extracorporeal circuit. The twin-coil Kolff kidney utilizes the principle of ultrafiltration whereby considerable quantities of water can be removed.

Thus, in summary, there can be an imbalance in outflow-inflow rates, the patient can bleed, the dynamic-static volume difference can cause hypotension, blood can be transfused, and fluid can be removed by ultrafiltration.

The importance of having a simple means of assessing and correlating these changes with a view to effective management during haemodialysis is apparent. It was felt that weighing the patient should be able to afford the necessary help in this respect.

Although Wangensteen (1947) and Merril (1955), among others, have stressed the importance of weighing in the management of fluid balance, its routine use has not received wide application. This can be explained to a large extent by the cumbersome and complex weighing structures commonly in use, many of which involve a most complicated procedure in taking a reading (Wangensteen, 1947; Alwall, 1952; Hamburger and Mathé, 1952).

The construction of a weighing apparatus for use during haemodialysis must be determined by, and should comply with, the following criteria: (1) accuracy,