Adhesive obstruction

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Key words: INTESTINAL OBSTRUCTION; CHANGING PATTERNS; MECHANISMS OF TREATMENT OF ADHESIONS; MORBIDITY; MORTALITY

Intestinal obstruction due to adhesions is a subject of importance to all general surgeons. Its changing incidence, aetiology and management have attracted increasing interest in recent years. The challenge presented is that of a benign condition causing severe morbidity and significant mortality in patients of all ages despite improvements in management in latter years.

Incidence

The changing incidence of the condition during the past 25 years is now illustrated by presenting 2 series of patients from Dudley Road Hospital, a district general hospital in a dense urban area in the West Midlands.

Series 1

The first is a personal series of 414 patients treated during the 16 years 1960–1975 (Table I). These fall into 3 main groups—252 cases with simple occlusion, 93 strangulations and 69 of inflammatory origin. In terms of the total workload of the surgical division, 3000 patients were admitted with intestinal obstruction during this period, forming just over 3% of the emergency surgical admissions to the hospital (Table II).

TABLE I *Intestinal obstruction, 1960–1975, personal series*

Occlusions	252
Strangulations	93
Inflammatory	69
	414

TABLE II *Dudley Road Hospital, 1960–1975*

Number of general surgical inpatients	160 000
Emergencies (60%)	96 000
Intestinal obstruction (3.1% of emergency surgical admissions)	3000

When the numbers admitted annually are analysed, a significant reduction is found in the later years (Fig. 1). During the first 8 years of the series (1960–1967), 261 (63.0%) presented compared with 151 (37.0%) during the second 8 years (1968–1975)—a reduction to nearly one half. During this time the catchment population, surgical staffing and total workload remained the same, and the only material change in the pattern of the work resulted from a deliberate policy of cultivating outpatient work in relation to emergency admissions; in 1960 90% of surgical admissions were emergencies and 10% elective, but by 1970 the proportion had become 50%, which has remained the position today.

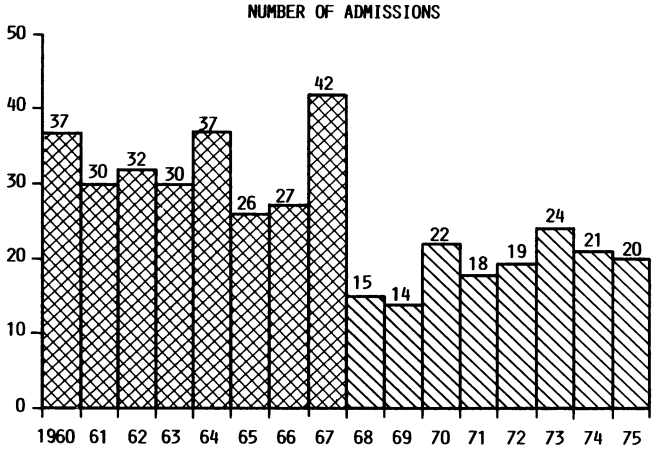

FIG. 1 Histogram to show annual incidence of cases of intestinal obstruction in Series I (1960–1975 inclusive).

When the 3 groups are considered individually, the commonest cause of the occlusion was carcinoma, mostly of the large bowel, greatly in excess of cases presenting with adhesions (Tables III and IV). Throughout this period the incidence of carcinoma as a cause of obstruction was found to have been falling, especially from 1967 to 1975 (Fig. 2), and that of adhesions rising, particularly during the past few years of this series (Fig. 3). As will be shown below, these tendencies have continued since then in a dramatic way, and have been confirmed by other authors *(1)*.

TABLE III *Cause of occlusions*

Carcinoma	122
Volvulus	19
Adhesions	58
Food bolus	10
Faecal	4
Neonatal	12
Intussusception	8
Others	19
	252

TABLE IV *Malignant obstructions*

Small bowel { 3 lymphosarcomas, 2 carcinomas }	5
Carcinoma of right side of colon	18
Carcinoma of left side of colon	66
Palliative procedures	28
Inoperable carcinomatosis	5
	122

Based on the Bradshaw Lecture delivered at The Royal College of Surgeons of England on 14th April, 1983.

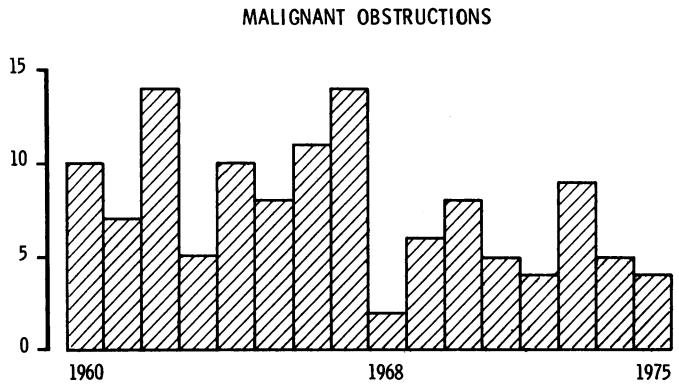

FIG. 2 Annual incidence of carcinoma as a cause of intestinal obstruction (Series I, 1960–1975).

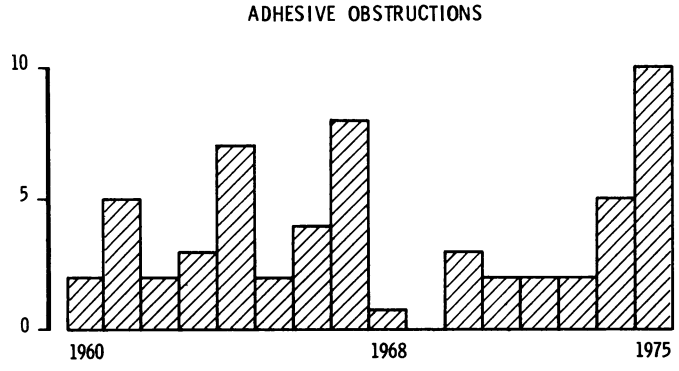

FIG. 3 Annual incidence of adhesions as a cause of intestinal obstruction (Series I, 1960–1975).

With regard to the strangulations, by far the commonest cause was hernia, especially in the groin (Table V), and again the annual incidence shows a marked fall (Fig. 4). The group of patients with functional obstruction resulting from superior mesenteric arterial thrombosis forms a special problem; all 9 had developed irreversibly ischaemic bowel, with resection feasible in 6 and 3 adjudged hopeless. In none was there a possibility of reconstructive surgery.

TABLE V *Strangulations*

Internal	7
SMA obstruction	9
Hernia	77
	93

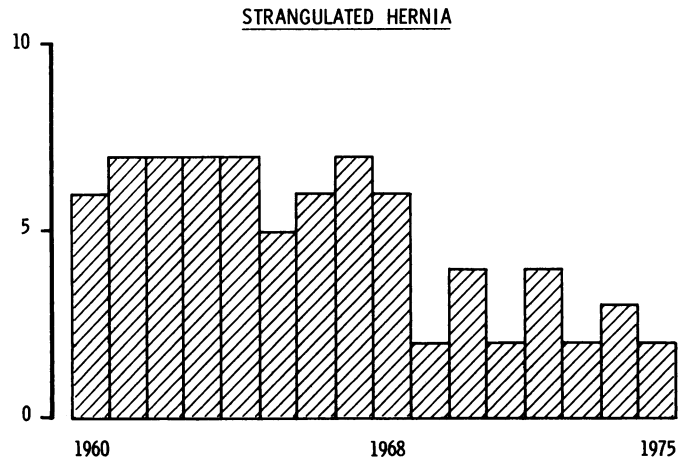

FIG. 4 Annual incidence of intestinal obstruction due to strangulation (Series I, 1960–1975).

The inflammatory group shows an interesting variety of causes of obstruction (Table VI). The incidence of diverticular disease of the colon fell markedly during these years with the advent of bran; cases of Crohn's disease maintained a steady level, and tuberculosis of the small intestine reappeared associated with the establishment of large ethnic minorities from India and the West Indies in the West Midlands.

TABLE VI *Inflammatory causes of obstruction*

Diverticulitis	21
Granulomatous ulcer	10
Abscess	4
Appendix	4
Omental necrosis	4
TB	5
Colitis	6
Crohn's disease	13
Others	2
	69

Series 2

The second series includes the total number of patients admitted to the same hospital during the 5 years subsequent to the first series. These cases were admitted under all 6 general surgeons from 1976 to 1980 inclusive, and I thank my colleagues for having allowed me to analyse their patients (Table VII). In all, there are records of 277 patients, spread fairly evenly over the 5 years. Adhesions now form the commonest cause of obstruction by far—104 cases out of 277 (37.9%). Carcinoma is relegated to second postion—47 cases (16.9%). The other causes form small but consistent contributions; the inflammatory group includes diverticular disease, Crohn's disease, tuberculosis and non-specific ulcers (possibly ischaemic) and the pseudo-obstructions of uraemia, acute diabetes, senile bowel distension and malignant infiltration of the mesentery.

TABLE VII *Total Admissions to a District General Hospital for Intestinal Obstruction, 1976–1980*

	1976	1977	1978	1979	1980	*Total*
Adhesions	29	18	11	20	26	104 (37.9%)
Carcinoma	5	7	5	10	20	47
Faecal impaction	10	2	3	2	2	19
Strangulations	13	7	10	4	3	37
Inflammatory	9	7	7	8	9	40
Others	3	5	3	3	3	17
Pseudo-obstruction	4	2	2	3	2	13
	73	48	41	50	65	277

Thus adhesions have become the most frequent cause of intestinal obstruction in recent years. The two commonest causes of 20 years ago, carcinoma of the large bowel and hernia, have fallen considerably in incidence recently. This is probably due to the great increase in elective, as distinct from emergency, surgery that has taken place with signal advantage to the patients who have their operations before obstruction can occur. This is one index of the efficiency of a hospital service.

Aetiology

Analysis of the 104 patients presenting with adhesive obstruction from 1976 to 1980 reveals that 91 (88.2%) had had previous abdominal surgery and of these 36 (34.6%) multiple operations (Table VIII). Seventy-six (73.1%) needed

TABLE VIII *Adhesive obstructions, 1976–1980*

Total	104
Operated	76
Died	7 (3 had no operation)
Previous operations	91
Previous multiple operations	36

operative treatment as a matter of urgency. Seven of these patients died (6.7%), all with a benign condition, 3 of these being moribund on admission due to perforation and peritonitis. We are, therefore, confronted with an iatrogenic condition; the alarming increase in the number of patients developing obstruction from adhesions is related to the vast and progressive increase in the number of laparotomies performed nowadays. The previous operations responsible for subsequent adhesive obstruction are listed in Table IX.

TABLE IX *Previous operations*

Appendicectomy	34
Operations for adhesions	23
Caesarian section, hysterectomy, and other gynaecological operations	21
Cholecystectomy	11
Colectomy	9

As far as could be ascertained, there were five main causes for intra-peritoneal adhesions in this series. They may be congenital or result from injury, irritants, inflammation or ischaemia. Congenital adhesions were uncommon accounting for obstruction in a few of the cases who had not had previous laparotomy. Some of the worst examples of adhesive obstruction were encountered in patients who had needed previous laparotomy for severe intra-abdominal injuries.

Irritant materials or particles accounted for a number of adhesions needing surgical resolution. Of particular importance is talc powder used on rubber gloves; although starch has replaced magnesium sulphate (notorious for causing adhesions), small starch granulomata and adhesions are still frequently reported *(2)*, but are found only if looked for by double refraction. Antibiotic granulomata are becoming less common, but are still the price paid by a few patients for prolonged antibiotic exhibition. Other irritants introduced at laparotomy include filaments of gauze and cotton wool, and non-absorbable sutures.

In regard to inflammation, peritonitis of whatever cause, localised or generalised, leaves most patients with adhesions, especially after spillage of gastric or intestinal contents. Infective conditions involving peritonealised organs—appendix, gall bladder, sigmoid colon or pelvic genitalia—usually lead to the same result, especially the chronic granulomata such as Crohn's disease and tuberculosis.

Tissues deprived of blood supply necrose and attract vascular adhesions. This is probably the commonest cause of post-laparotomy adhesions, and has been demonstrated by Ellis in a series of elegant experiments at the Westminster Hospital *(3)*.

Clinical manifestations

Adhesions may cause no trouble at all, or may be present for many years, or indefinitely, without becoming a source of symptoms. This is well illustrated by a recent personal case: a man of 62 needed laparotomy for adhesive obstruction in March 1983, having undergone partial gastrectomy for chronic duodenal ulcer in 1940 and appendicectomy in 1941; he had been free of symptoms until 2 days before the onset of intestinal obstruction.

The commonest clinical consequence of adhesions is pain, which may be sharp and sudden, or chronic and nagging. Its occurrence is often incalculable as it may be episodic, with episodes worsening or dying out, or take the form of a single isolated attack. Diagnosis is difficult and may be only exclusive after a battery of negative investigations. I have found three clinical features of help in diagnosis:

(i) Change of posture, such as turning over in bed at night, may either precipitate or relieve the pain.
(ii) The pain is usually situated in the region of an abdominal incision.
(iii) Tenderness is elicited by pressure on the scar of an old incision.

Adhesive obstruction is attended by two hallmarks—obstruction of the small intestine and the rapid development of strangulation. Once obstruction is confirmed by the presence of the four cardinal features of abdominal pain, progressive vomiting, distension and constipation, a serial assessment must be made *(4)* to select those who need operative treatment and to decide on its timing (Table X).

TABLE X *Serial diagnosis*

Cardinal features of obstruction
Level of obstruction
Degree of dehydration
Modality
Clinical pattern
Pathological cause

The small intestine is always involved, but the level is important as shown by straight radiographs in the erect position demonstrating the number and location of fluid levels, and is related to the degree of dehydration. Low level small intestinal obstruction is associated with increasing abdominal distension and slowly progressive dehydration. Blockage at mid small bowel level is usually accompanied by severe colic. Upper small intestinal obstruction is deceptive, causing rapid and severe dehydration without distension *(5)*.

The four modes are simple occlusion, strangulation, closed loop and functional obstruction. That due to adhesions is commonly closed loop, with the imminent danger of perforation or strangulation, both potentially lethal.

Analysis of the clinical pattern of development of symptoms showed five patterns in this series—acute or progressive usually due to carcinoma, remitting as with sigmoid diverticular disease, chronic in Crohn's disease and tuberculosis, and recurrent, typical of adhesions.

Strangulation is the most serious complication and yet difficult to diagnose in view of its inconsistent clinical features. Here the criteria established by Lefall and Syphax *(6)* are helpful (Table XI). Perforation with peritonitis and severe blood loss from the strangulating loop may be the final *coup de grace* for the patient afflicted by adhesive obstruction.

TABLE XI *Symptoms and signs of strangulation (6)*

Abdominal tenderness
Tachycardia
Leucocytosis
Constant pain
Fever

This serial diagnosis enables selection of those patients in need of urgent laparotomy—high level small intestinal obstruction, closed loop obstruction, the acute clinical pattern and evidence of strangulation.

Distribution of adhesions

At laparotomy adhesions may be interintestinal and mesenteric, viscero-parietal with loops of small bowel and omentum stuck to the parietes, or a combination of these two types.

On computing the operative findings regarding the distribution of adhesions in patients included in the two series presented above, 4 main groups were found—diffuse, single band, incisional and omental bowstring. It is important to distinguish these from the point of view of management, and erect radiographs are a valuable guide.

Multiple adhesions are difficult to deal with technically, needing lengthy dissection and running the risk of producing multiple intestinal fistulae. Where possible surgery should be avoided and every effort made with conservative measures. When the obstruction is irreversible, there is much to commend the method of complete small intestinal intubation using the Baker tube *(7)*. The alternative is gentle and meticulous freeing of all the adherent intestinal loops. The Noble plication procedure seems illogical and is potentially hazardous by promoting artificial adhesions. Multiple diffuse adhesions are suggested by the appearance of scattered fluid levels throughout the abdomen on the erect scout film.

The single band is the most dangerous type of adhesion, so often leading to rapid strangulation and perforation, and occasionally a sad finding at autopsy. It is a simple operation to divide or excise the offending band. The erect film shows a localised clump of gas shadows and fluid levels.

Of no less urgency is the loop of small bowel densely adherent to the back of a previous incision, causing a closed loop effect and suggested by a single distended loop with two main fluid levels on the erect radiograph.

The omental bowstring type (Fig. 5) results from adherence of the greater omentum to the pelvis or iliac fossae, and a loop of small bowel prolapsing through the gap thus produced. From the operative point of view, this is an indication for omentectomy.

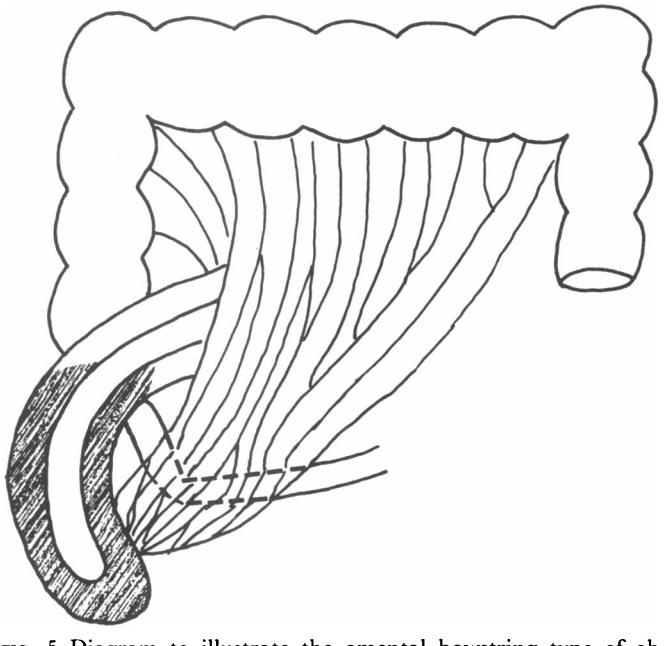

FIG. 5 Diagram to illustrate the omental bowstring type of obstruction causing strangulation of a loop of small bowel.

Other interesting but uncommon varieties of adhesions met in these series include Meckel's obstruction (Fig. 6), appendicular obstruction of the terminal ileum (Fig. 7) and circumferential soft tissue bands (Fig. 8) that may involve small or large bowel.

The mechanism and prevention of adhesions

All laparotomies are probably followed by adhesion formation; in the majority the adhesions disappear, but in some they organise, persist and cause pain or obstruction. The

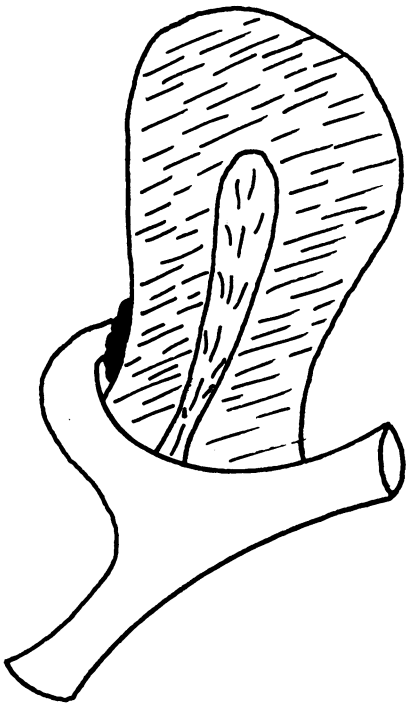

FIG. 6 Diagram showing the findings in a case of obstruction due to a strangulating loop of small bowel from an adherent Meckel's diverticulum.

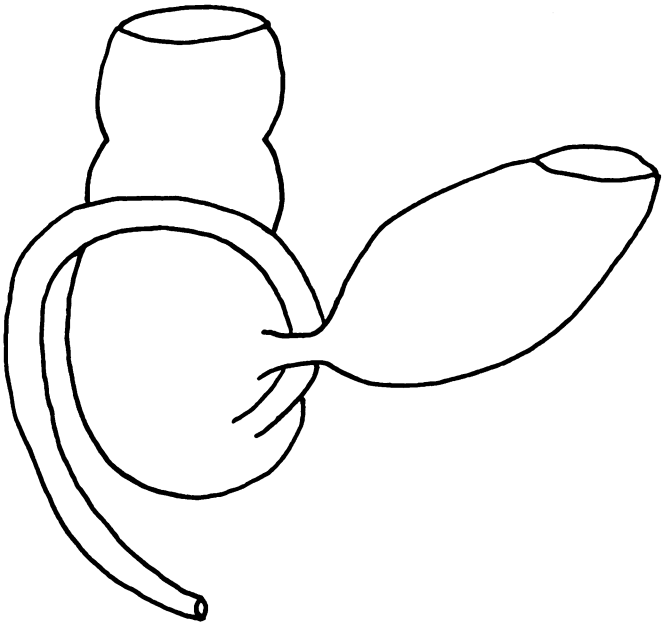

FIG. 7 Obstruction of the terminal ileum due to encirclement by the appendix.

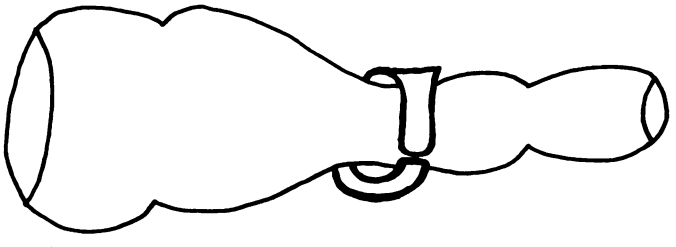

FIG. 8 Circumferential soft tissue bands as a cause of obstruction which may involve small or large bowel.

sequence of events is shown in Table XII. Fibrous organisation must result from the failure of lysis of fibrinous adhesions.

TABLE XII *Mechanism of adhesion formation*

stimulus
↓
increased blood vessel permeability
↓
exudate formation
↓
fibrinous adhesions
↓
fibrous organisation
↓
fibrinolysis overwhelmed

Tissue injury leads to the formation of a fibrin clot via the sequence of events in the coagulation cascade (Fig. 9, [8]), in which thromboplastins, prothrombin and thrombin are activated in turn to convert fibrinogen into fibrin. The platelet clot from platelet aggregation combines with the fibrin clot to form fibrous adhesions; this can be opposed or stimulated by different members of the prostaglandin group.

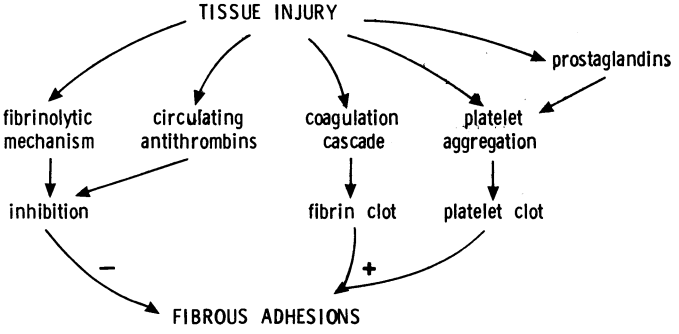

FIG. 9 The patho-physiological pathways through which adhesions form, and are opposed by fibrinolysis. Reproduced by permission of Blackwell Scientific Publications. (8)

This process is inhibited by the fibrinolytic mechanism activated by circulating antithrombins, another cascade phenomenon in which activated plasminogen produces plasmin and lyses the fibrin coagulum before fibrous organisation can occur (Fig. 10, [8]). Fibrin degradation products inhibit coagulation and add to the fibrinolytic effect. Ischaemic tissues show low plasminogen activity. New revascularising adhesions bring blood-borne antiplasmins to counteract fibrinolysis and perpetuate adhesions.

Logically, adhesions could be prevented by administering fibrinolytic agents such as streptokinase and urokinase systemically or by intraperitoneal instillation. These agents have been tried and have failed, and even worse have produced serious side effects such as infection and haemorrhage. Surgeons have inserted a large variety of agents into the peritoneal cavity in attempts to prevent adhesion formation (Table XIII), illustrating the ingenuity of the surgical mind, but achieving no positive results, except for the formation of more adhesions.

Further research on these humoral and cellular processes may produce an answer in the future but, for the present, we must continue to strive for surgical solutions to the problem. Surgical measures can be listed as preventive and therapeutic and are summarised in Tables XIV and XV. Surgical technique becomes all important. There is ever an important

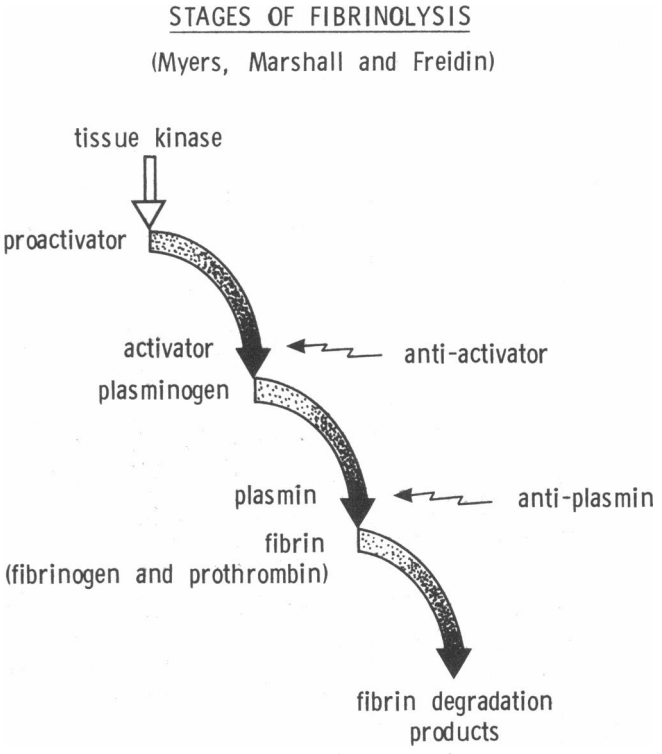

FIG. 10 The stages of fibrinolysis. Reproduced by permission of Blackwell Scientific Publications. (8)

TABLE XIII *Intra-peritoneal agents used to prevent adhesions*

Pneumoperitoneum	Saline, dextran, dextrose
Anticoagulants	Liquid paraffin, olive oil
Fibrinolytic agents	Gelatin
Steroids	Antihistamines
Hyaluronidase	Povidone-iodine
Oxygen	Hydrophilic polymer coatings
Noxyflex	

TABLE XIV *Operative measures—preventive*

1. Exclude irritants, especially talc
2. Avoid spillage and cleanse peritoneal cavity
3. Minimise operative trauma; haemostasis
4. Excise necrotic tissue
5. Technique of wound closure

TABLE XV *Operative measures—therapeutic*

1. Produce controlled adhesions, eg Noble's procedure
2. Thorough division of adhesions and debridement
3. Small intestinal intubation
4. Omentectomy where indicated
5. Positional replacement of bowel

place for gentleness in abdominal surgery and restricting unnecessary dissection at the primary operation. Our methods of wound closure must be reconsidered; in this regard mass closure of the abdominal wall (rectus muscle with anterior and posterior rectus sheaths) is increasingly favoured, leaving the peritoneum unsutured. It will be interesting to discover if this reduces the incidence of adhesive obstruction.

Complications

Certain it is that, despite our best efforts, the rate of severe complications and mortality of operations for adhesive obstruction remains unduly high. Review of the causes of

death in these series reveals the prevalence of five serious complications—sepsis, dehydration, thrombo-embolism, renal failure and broncho-pneumonia.

Severe septic complications take the form of septicaemia due to gram negative organisms entering the portal venous system especially from intestinal strangulation, or peritonitis following perforation or intraperitoneal spillage which may lead to subphrenic, intraperitoneal or pelvic abscesses. Our practice is to give a cephalosporin and metronidazole for 5 days postoperatively starting with a preoperative dose, or intravenous gentamycin peroperatively when an unexpected situation is met.

Fluid and electrolyte balance becomes badly deranged and where surgery is urgent full correction has to be made postoperatively. Regarding preoperative intravenous therapy, experience has shown that where fluid loss has been so rapid and severe as to induce hypotension, rapid restoration of blood volume with large amounts of fluid, salt and potassium is needed to restore normotension before starting surgery; where the patient is in need of urgent operation but with no circulatory collapse, overtransfusion is to be avoided and time not to be wasted in attempting to replace the fluid deficit fully before surgery. Postoperatively there is less awareness of the loss of fluid and electrolytes that continues for some days and needs replacement in excess of the charted fluid loss. This results from sequestration of fluid into the bowel wall and soft tissues of the abdomen. Fig. 11 shows the fluid balance of a patient in this situation, with a positive balance of 11 l necessary for replacement during the first 2 postoperative days, and a second phase of positive postoperative balance of 4.7 l on days 4–9 to overcome the sequestration.

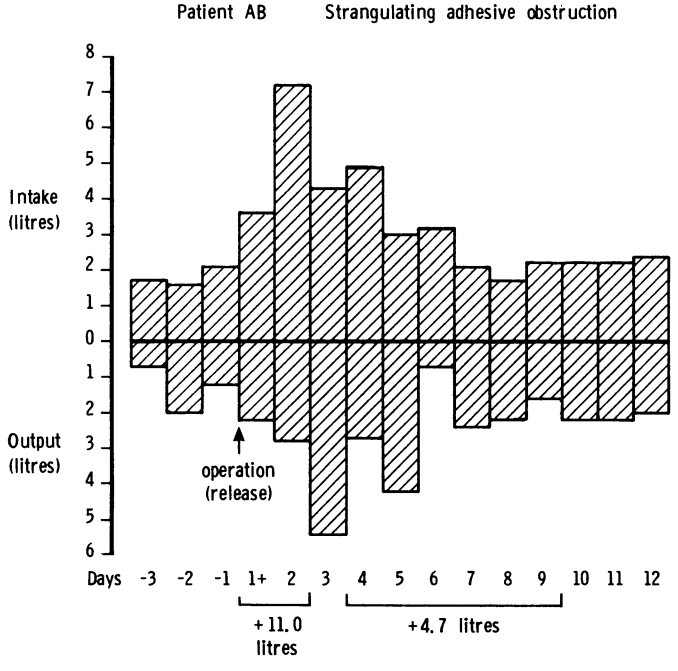

FIG. 11 The fluid balance of a patient with adhesive obstruction treated surgically. Each column represents 1 day. The values below the baseline represent fluid losses, and the amounts needed to keep the patient in balance are shown above the baseline.

Deep vein thrombosis and pulmonary embolism are particularly liable after surgery in view of dehydration, abdominal distension and other factors. In our practice these patients all receive subcutaneous heparin for at least 7 days.

Tubular necrosis of the kidney and renal failure can result from fluid deficit, hypotension and infection, demanding adequate fluid and electrolyte provision and prophylactic mannitol into the intravenous infusion.

The final threat to life is 'bronchopneumonia', chronic ventilatory failure resulting from a combination of factors summarised in Table XVI, and culminating in the shock lung syndrome.

TABLE XVI *Factors in Bronchopneumonia*

Sepsis
Pre-existing lung disease
Hypotension
Pulmonary oedema
Inhibition of diaphragmatic movement
Aspiration

Acute intestinal obstruction still presents one of the greatest challenges to surgical diagnosis, assessment and management, and calls for expert judgement, technique and monitoring.

My grateful thanks are due to the Department of Medical Photography and Illustration, Dudley Road Hospital, for producing the Figures, and to Mrs Valerie Evans for typing the manuscript.

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