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REVIEW

# Postoperative adhesions: their treatment and relevance in clinical practice

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The clinical picture of intestinal obstruction is well known to all general surgeons. When combined with a history of previous abdominal surgery the most likely cause is adhesions. In 1932 Vick reported that adhesions accounted for only 7% of all cases of intestinal obstruction *(1)*. At that time the most common cause of intestinal obstruction was strangulated external hernia. Later in this century the incidence of intestinal obstruction caused by adhesions had increased. Table I shows four commonly quoted series of large and small bowel obstruction from 1952 to 1982. Adhesions caused obstruction in approximately 30% of cases. In the series in Table II small bowel obstruction alone is reported. Adhesions caused more than two-thirds of the cases of small intestinal obstruction. This illustrates the dramatic change from the first half of this century. Adhesions are now the most common cause of intestinal obstruction. This increase in the proportion of intestinal obstructions that are produced by adhesions may still be continuing. In Table III, the last two series of intestinal obstructions indicate that adhesions now account for more than 40% of all cases of intestinal obstruction.

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Adhesions may be classified as either acquired or congenital. The acquired type of adhesion is subdivided into inflammatory or postsurgical. The true incidence of each of these types of adhesions is unknown. Congenital and inflammatory adhesions have been reported to cause intestinal obstruction, although they do so only rarely *(12)*. The majority of cases are postsurgical.

## Incidence of adhesions

An assessment of how many people develop adhesions after surgery was performed in a post-mortem series by Weibel and Majno in 1973 *(13)*. In cadavers with no preceding abdominal surgery, adhesions were found in 28%, and in those that had had abdominal surgery 67% had adhesions. Where minor abdominal surgery had been performed, adhesions were present in about 50%. If major surgery had been undertaken adhesions were present in 76%, and in cases of multiple abdominal surgery 93% had adhesions present.

The incidence of adhesions in a live population has been examined *(10)*. Inflammatory adhesions in patients who had not undergone any preceding abdominal surgery were found to be present in 10%. In patients who had had previous abdominal surgery, postoperative adhesions were found in 93% and inflammatory adhesions in

*Table I.* Spectrum of intestinal obstruction of large and small bowel in the UK and USA in the latter half of this century

| | *Cause of obstruction (%)* | | | | *Total cases* |
|---|---|---|---|---|---|
| | *Adhesions* | *Hernia* | *Malignancy* | *Other* | |
| Nemir *(2)* | 30 | 21 | 19 | 30 | 430 |
| Perry *et al. (3)* | 31 | 10 | Not indicated | | 1252 |
| Bevan *(4)* | 38 | 13 | 17 | 32 | 277 |
| McEntee *et al. (5)* | 32 | 25 | 26 | 17 | 228 |

*Table II.* Spectrum of small bowel obstruction

| | *Cause of obstruction (%)* | | | *Total cases* |
|---|---|---|---|---|
| | *Adhesion* | *Hernia* | *Malignancy* | |
| Playforth *et al. (6)* | 54 | 23 | 9 | 111 |
| Laws and Aldrete *(7)* | 69 | 8 | 10 | 465 |
| Stewardson *et al. (8)* | 64 | 24 | 7 | 238 |
| Bizer *et al. (9)* | 74 | 8 | 9 | 405 |

*Table III.* Proportion of obstructions which require surgery produced by adhesions in the most recent series of intestinal obstructions of both large and small bowel

| | *Adhesions (%)* | *Total number* |
|---|---|---|
| Menzies and Ellis *(10)* | 148 (41) | 359 |
| Fuzun *et al. (11)* | 256 (44) | 582 |

20%. Congenital adhesions were identified in less than 1%. The difference in the incidence of adhesions between the two studies is due to a difference in the age groups between the post-mortem and live studies. In the post-mortem study inflammatory adhesions were rarely seen in those under the age of 60 years. The mean age of patients without earlier abdominal surgery in the live study was 63 years. In patients who had had surgery the mean age was 75 years and therefore they were far more likely to have had intra-abdominal inflammatory episodes that resulted in adhesions, such as cholecystitis and diverticulitis.

## Workload from adhesions

In 1914 Payer *(14)* reported that 3.5% of all laparotomies were for intestinal obstruction from any cause. Since then the spectrum of intestinal obstruction has changed. Bevan, in 1984, calculated that 1.9% of all admissions were for intestinal obstruction *(4)*. Irvin *(15)* noted that 3.5% of all emergency surgical admissions that resulted in laparotomy, were for adhesive intestinal obstruction. In a review over a 25-year period it was shown that adhesions accounted for 1% of all surgical admissions and 3% of all laparotomies in one surgical unit *(10)*. In this series the number of laparotomies performed each year for adhesive intestinal obstruction was found to increase with time (Fig. 1), but this matched the increase in the total number of laparotomies performed each year for any reason (Fig. 2). It is likely that although the incidence of adhesive obstruction is increasing it is doing so because more and more people are being submitted to a laparotomy each year and, as has already been noted, more than 90% of these will develop intra-abdominal adhesions after surgery.

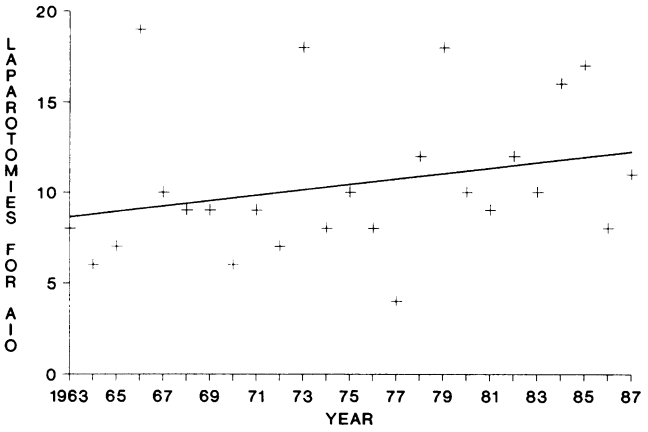


*Figure 1.* Laparotomies for adhesive intestinal obstruction each year (1963–1987). AIO = Adhesive intestinal obstruction.

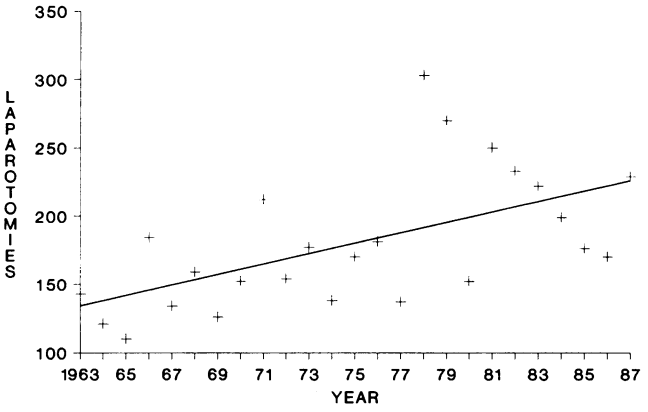


*Figure 2.* Total number of laparotomies performed each year (1963–1987).

## What proportion of laparotomies develop adhesive obstruction?

It is well known that adhesions may present as obstruction 30 or more years after abdominal surgery, and it would be impossible to follow up a group of patients for this period of time to find out how many ultimately obstruct from their adhesions. Stewart *et al. (16)* followed a series of over 8000 patients for 1 month after surgery and found that 0.6% developed obstruction which required surgery within 1 month of their initial operation. This figure was confirmed at Westminster Hospital where 2700 laparotomies were followed for 1 month and 0.5% obstructed within that time *(10)*. With continued follow-up, 1% were found to require operation for adhesive obstruction within 1 year.

Few series of adhesive obstructions report the time interval from surgery to obstruction in any detail but, in the two series that reliably report the interval *(10,17)*, it appears that more than one-third of patients with adhesive obstruction present within 1 year of surgery. Thus, if 1% of all laparotomies produce obstruction within 1 year and this represents one-third of all adhesional obstructions, then it might be expected that 3% of all

laparotomies will eventually obstruct at some time after their original surgery. This proportion, although impossible to confirm, does agree with the figure that 3% of all laparotomies are for adhesive obstruction.

## Which adhesions produce obstruction?

It has been reported that operations below the transverse mesocolon are more likely to produce adhesive intestinal obstruction *(16)*. The reason for this is almost certainly the exposure of the small bowel to trauma at the time of surgery.

In a study from Japan of a series of 88 adhesive intestinal obstructions, the authors simply classified the adhesions which produced the obstruction into those which involved the small bowel alone and those which formed between the small bowel and neighbouring viscera (which included the site of previous surgery) *(18)*. They found 29% of obstructing adhesions were between loops of the small bowel alone and 48% were between the small bowel and other viscera. The remaining cases had adhesions that were too dense to accurately identify the obstructing adhesion type.

At Westminster Hospital two series of patients have been examined to try to identify which adhesions form after surgery and which adhesions are at particular risk of producing intestinal obstruction. In one series the distribution of intraperitoneal adhesions that developed after abdominal surgery was recorded. In the other the adhesions that actually produced intestinal obstruction were identified.

The distribution of all adhesions after abdominal surgery has been studied in a series of 210 patients who underwent a laparotomy after previous abdominal surgery and is shown in Table IV. Here the most common sites for adhesions were to the undersurface of the abdominal wound which occurred in 84% or to the site of the previous surgery in 58%. The omentum was the organ most commonly involved in adhesions to the scar (72% of scars) and to the site of the previous surgery (22%). Adhesions from the small bowel to the wound occurred in only 18% of wounds and from the small bowel to the site of surgery in 16%. Adhesions which involved the small bowel alone occurred in only 8% of cases. Overall, the omentum was involved in 57% of the sites for adhesions and the small bowel was involved in 27% of sites.

*Table IV.* Sites of postsurgical adhesion formation in 210 patients with previous abdominal surgery

| *Site of adhesions* | *Number of occasions* |
|---|---|
| Omentum to scar | 170 |
| Small bowel to scar | 42 |
| Colon to scar | 13 |
| Liver to scar | 3 |
| Uterus to scar | 1 |
| Omentum to site of previous surgery | 47 |
| Small bowel to site of previous surgery | 33 |
| At site of previous surgery alone | 57 |
| Small bowel to small bowel | 17 |
| Small bowel to parieties | 4 |
| Omentum to small bowel | 10 |

*Table V.* Site of adhesions which produced obstruction in 80 cases of adhesive obstruction

| *Site of adhesions* | *Number of occasions* |
|---|---|
| Small bowel to site of previous surgery | 42 |
| Small bowel to small bowel | 19 |
| Small bowel to scar | 6 |
| Small bowel to small bowel and site of previous surgery | 1 |
| Small bowel to pelvis | 1 |
| Obliterated peritoneal compartment | 1 |
| Omentum to scar | 1 |
| Omentum to site of previous surgery | 1 |
| Unknown | 8 |
| Total | 80 |

The distribution of obstructing adhesions was identified over a 13-year period in a series of 80 cases of adhesive intestinal obstruction, by reviewing the operation notes. The distribution of these adhesions is shown in Table V. The adhesions involved the small bowel in 86%. Adhesions between the small bowel and the site of previous surgery caused obstruction in 52%. Adhesions which involved the small bowel alone caused obstruction in 24%. In only two cases was the obstruction caused by adhesions which involved the omentum.

If the distribution of these obstructing adhesions is compared with that of any adhesion that develops after abdominal surgery, it is clear that, although omental adhesions are the most common adhesion to be found, they are at low risk of producing intestinal obstruction. Adhesions between small bowel and other viscera or other loops of small intestine occur less frequently but are far more likely to cause adhesive obstruction.

The omentum plays a protective role in adhesion formation. Adhesive obstruction after total colectomy is well known. In the Japanese series *(18)*, the most common initial operation was gastrectomy, both these operations may involve omentectomy and thus will remove the organ that forms 'safe' adhesions. This would leave adhesiogenic areas exposed to the small bowel and will result in a higher incidence of the more dangerous small bowel adhesions.

A further consideration is that at the time of a subsequent laparotomy, performed for any reason, it is frequent practice to divide any adhesions that are encountered. This may not be sound surgery. The division of adhesions which involve the small bowel, that are of high risk of later obstruction, is acceptable. The division of adhesions between organs other than the small intestine may not be necessary as they are unlikely to precipitate intestinal obstruction. Indeed their division

will almost certainly result in recurrent adhesion formation which may involve the small intestine and only serve to increase the chance of ensuing small bowel obstruction.

## Adhesion control

Clinically, there is no means of completely preventing adhesion formation. Two commonly used solutions in clinical practice that have some anti-adhesion effect in the laboratory animal are povidone-iodine *(19)* and 32% dextran 70 *(20)*. Povidone-iodine is popular among some general surgeons as a peritoneal lavage, more for the antimicrobial effect than the anti-adhesion properties. Dextran is often used by gynaecological surgeons for adhesion prevention in infertility surgery and there is some clinical evidence that it has some effect in these cases *(21)*.

Independent of adjuvant therapy for the prevention of adhesion formation, there are several operative steps that can be taken to reduce the extent of adhesion formation and to minimise the chances of subsequent adhesive obstruction:

1 Handle the bowel carefully to reduce serosal trauma.
2 Avoid unnecessary dissection.
3 Exclude foreign material from the peritoneum, eg use absorbable ligatures and sutures where possible, preferably those that are hydrolysed rather than phagocytosed. If non-absorbable material is to be used then keep the cut ends as short as possible. Avoid excessive use of gauze swabs. Wear starch-free gloves.
4 Adequately excise ischaemic or infected debris within the peritoneum.
5 Preserve the omentum, if possible. Place the omentum around the site of surgery and run the omentum under the wound to encourage low-risk adhesions to form.
6 Avoid the division of adhesions that do not involve the small bowel.

## Surgical treatment of adhesive intestinal obstruction

Once adhesions have produced intestinal obstruction, between 30% and 60% require surgical relief of the obstruction *(10,22–25)*. The incidence of recurrent obstruction after successful conservative therapy is poorly documented. The only study to have examined it in any detail reported a 12.5% recurrence rate in 40 patients with follow-up of between 3 and 13 years *(26)*.

Simple adhesion division is usually employed in those patients who require surgery for adhesive obstruction. Recurrent obstruction after simple adhesion division occurs in 11% to 21% (Table VI).

In those patients with recurrent obstruction, adhesion division may be combined with a sutured small bowel plication procedure or insertion of a long intestinal tube. The plication procedures of Noble *(28)* or Childs and Phillips *(29)* rely on sutures to hold the small bowel in a specific position so that further adhesive obstruction cannot occur. The long intestinal tube *(30)* is designed to hold the small bowel in a series of open loops until subsequent adhesions form to maintain the bowel in position and then the tube can be removed. The Noble plication has now been largely abandoned due to high complication rates *(31)*.

Despite the use of these additional procedures, recurrence rates are still significant, between 4% and 32% (Table VII). Although encouraging results have been reported for the long intestinal tube, its use should be confined to patients after division of extensive intra-abdominal adhesions. If used after division of only a few adhesions, when the adhesions re-form they may not be extensive enough to hold all the small bowel in an open looped position and therefore will permit movement and twisting of the bowel and allow subsequent adhesive intestinal obstruction to develop *(35)*.

## Fibrinolysis and adhesion formation

At present there is no pharmacological means available to prevent the formation or re-formation of adhesions that has been shown to be clinically effective. In the last few years exciting advances have been made in the laboratory which help to understand the cause of adhesions and identify a possible solution.

Peritoneal trauma is well known as a cause for adhesion formation. Ellis identified ischaemia as a potent stimulus for adhesion formation *(36)*. In 1963 Von Benzer demonstrated the fibrinolytic property of the peritoneum *(37)*. The majority of this fibrinolytic activity was thought to be contained within the mesothelial cell layer *(38,39)*. This was confirmed later *(40)*. The fibrinolytic activity was identified as plasminogen activation *(38,41)*. A reduction in plasminogen activation activity was linked to adhesion formation. Changes in plasminogen activator activity levels were shown to be due to stimuli well known to cause adhesions and were particularly marked in the presence of ischaemia *(42–46)*. This

*Table VI.* Incidence of recurrent intestinal obstruction after simple adhesion division

| | *Recurrences (%)* | *Follow-up years* | *Total cases* |
|---|---|---|---|
| Krook *(17)** | 14 | 4–24 | 135 |
| Brightwell *et al. (22)*† | 13 | 4–7 | 30 |
| Close and Christensen *(27)*† | 11 | ½–12 | 107 |
| Bizer *et al. (26)*† | 21 | 3–13 | 103 |

* Obstructive episodes treated by surgery
† Obstructive episodes treated both conservatively and by surgery

*Table VII.* Recurrent intestinal obstruction after plication procedures

| *Procedure/ recurrences* | *Sutured plication* | *Long intestinal tube* | *Follow-up (years)* |
|---|---|---|---|
| Brightwell *et al. (22)** | – | 9/28 (32%) | 4–7 |
| Close and Christensen *(27)*† | 4/28 (14%) | 3/37 (8%) | ½–12 |
| Weigelt *et al. (32)** | – | 12/140 (8.6%) | 0–5 |
| Hollender *et al. (33)** | 2/51 (4%) | – | 1–17 |
| Jones and Thomson *(34)** | – | 4/123 (3.3%) | ¼–11 |

* Obstructive episodes treated by surgery
† Obstructive episodes treated both conservatively and by surgery

reduction was not only due to the removal of plasminogen activators but also to the release of plasminogen activator inhibitors *(44,47)*, especially in the presence of inflammation and ischaemia.

This pathway for adhesion formation supports the use of fibrinolytics as anti-adhesion agents. Years before the plasminogen activator activity of the peritoneum was isolated, various fibrinolytic agents had been tested as adhesion inhibitors. Streptokinase had been used in the dog *(48)*, rat and rabbit *(49)*. Urokinase had been tested in the dog with some success *(42)*, but this was not seen earlier in the rat *(50)*. Various forms of activated plasmin had been examined in the rabbit *(51)*, rat *(52,53)* and dog *(54)*, again with varying degrees of success. The reason for the conflicting results may be due to an inter-species variation in the ability of streptokinase and urokinase to activate plasminogen. An additional and probably more important confounding variable is the type of stimulus that was used to create the adhesions in the laboratory animal. This varied from simple serosal stripping to extensive bowel abrasion or the creation of ischaemic tissue. The presence of ischaemia stimulates the release of plasminogen activator inhibitors *(44,47)* which will interfere with any anti-adhesion effect of a tested compound and make comparisons between stimuli difficult.

The commercial production of tissue plasminogen activator (rt-PA) by recombinant DNA techniques has allowed the study of the use of this agent in adhesion prevention. It has been used to replace the reduced plasminogen activator activity of traumatised peritoneum *(55,56)*.

In a rabbit adhesion model, rt-PA or a placebo was applied to an area of peritoneal trauma and to divided adhesions (produced by peritoneal trauma) and found to reduce the primary and recurrent adhesion rates from 80% with a placebo to 7% with rt-PA application. In doses effective in adhesion prevention, rt-PA has been shown to be safe in the presence of colonic anastomoses and not to alter abdominal wound strength or increase postoperative haemorrhage. The effectiveness of rt-PA as an adhesion deterrent has since been confirmed by others *(57–59)*, even when different adhesion models have been used; although it was noted that adhesions were not prevented around an anastomosis when the safety of rt-PA was being assessed in the presence of a colonic anastomosis *(56)*. This may have been due to the release of plasminogen activator inhibitors at the site of the anastomosis. The use of rt-PA has not yet been tried in the human as an adhesion deterrent, but it is the most promising agent yet described.

## Future advances

In the absence of any clinically proven means of preventing adhesions from forming, the onus lies with the surgeon to try and minimalise their occurrence by improved and assiduous surgical techniques. The advent of laparoscopic surgery may alter the incidence of adhesions and adhesive obstruction after abdominal surgery. The reduced bowel trauma from handling, the absence of large abdominal wounds and the exclusion of foreign material such as lint, gauze and starch from the abdominal cavity must reduce adhesion formation after laparoscopic surgery. One study has already demonstrated that when a stimulus is applied at open laparotomy in an animal it produces more adhesions than when the same stimulus is applied through the laparoscope *(60)*. No additional adhesions were found at remote areas when the laparoscope had been used.

Despite the promise of laparoscopic surgery, adhesions will continue to be a major source of concern for surgeons, not only because of the technical difficulties they present but also because of the volume of work they generate. It is possible that in the future these problems may be reduced by some form of rt-PA peritoneal lavage after surgery or adhesion division that will deter adhesion formation or re-formation and this will go some way in lightening the burden of a pathological process produced by surgery itself.

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