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This section provides a medium through which Canadian surgeons can declare themselves, briefly and informally, on the day-to-day affairs of surgery.

Surgery for Morbid Obesity — Where Are We Going?

Surgery for morbid obesity is predicated on the fact that obesity is a life-threatening condition for which there is no successful nonoperative treatment. Over the past two decades, we have gone from jejunocolic to several modifications of jejunoileal bypass, to gastric bypass and finally, to at least a dozen variations of gastric partitioning (gastroplasty) procedures.

In this issue of the Journal (page 283), Deitel and colleagues review their experience with intestinal bypass and gastric partitioning. Over a 9-year period, 174 patients were operated on. Initially, jejunoileal bypass was used exclusively (64 patients) and thus the authors have longer follow-up data for this group. An average weight loss of 35% is certainly acceptable, particularly because weight regaining did not occur. However, we do not know how many of the 64 individuals had this acceptable weight loss.

The authors are apparently fastidious in their follow-up because they were not bothered by the multitude of well-known metabolic complications associated with this procedure. Apparently, regular clinic visits kept the problems to a minimum. One bypass had to be reversed and two revised. The implication is that the other 61 patients are well and being followed. However, this is not specifically stated and one wonders if the authors really have perfect (100%) follow-up. If this is not the case, the paucity of metabolic complications and low incidence of reversal cannot be properly assessed. Similarly, the authors use expressions such as "some" and "generally" in discussing serial liver biopsies. Not one patient had serious liver disease. While it is known that fatty infiltration improves with time after jejunoileal bypass, particularly if protein is administered, more precise data would have been appropriate. The authors were apparently able to control diarrhea — the greatest problem after jejunoileal bypass. They stated that diarrhea after 1 year was not a problem and that it was easily controlled with medical treatment. This conflicts sharply with the world literature, which shows progressive, intractable diarrhea in up to 45% of the patients in the early postoperative period, persisting after 1 year in nearly 20% of patients and being the main cause for reversal of the bypass.

Gastric partitioning was performed in 110 patients. The authors used the simple technique of Pace and colleagues to solve a real surgical dilemma. Gastric bypass was a time-consuming, dangerous technique whereas removal of three or four staples from a TA90 stapler produced a simple gastroplasty that could be performed by anyone. Many surgeons jumped onto the bandwagon and used this operation, only to obtain similarly dismal results when patients were followed up for 1 year or longer. Deitel and colleagues operated on 56 patients before realizing that weight loss with this technique was inadequate. One wonders if they could have operated on a smaller number of patients and followed them up before undertaking such a large number.

Subsequently, 54 patients had reinforcement of the 12-mm stoma with a "nonexpanding" imbricating suture. Our experience and that of others clearly indicates that a running, seromuscular suture around the gastrogastrostomy does not guarantee perfect results. Although there was a mean 31% weight loss at 1 year, only five patients were followed up for more than 12 months. We predict that with a longer follow-up Deitel and his colleagues will see more patients with dilated stomas and inadequate weight loss or weight gain.

There are four causes for inadequate weight loss: three are technical — stomal dilatation, pouch dilatation and staple-line disruption; the other is patient compliance — one cannot assume that the mere placement of two rows of staples across the stomach will cure a lifetime addiction to food! Each of the technical problems can occur at the time of operation or subsequently, when they are generally related to poor patient compliance. Admittedly, stomal dilatation is the single greatest problem because most surgeons doing this operation are careful to make a small pouch and to apply the stapling instrument twice rather than once. However, reinforcing sutures are not the final solution. Despite initial problems with Marlex wrapped around the stoma, more surgeons are returning to this technique because of the persistent and troublesome problem of stomal dilatation. Incidentally, most deaths have been related to aggressive attempts to narrow the stoma because such attempts, with or without Marlex, devascularize the stomach and lead to leaks. In this regard, the paucity of serious complications and absence of deaths in Deitel's series is commendable.

Deitel and colleagues reoperated on five patients, again without serious mishap, although note should be made, in passing, that the mortality for gastroplasty revisions is nearly 4%. As stated, dilatation of the stoma is the commonest cause of inadequate weight loss or weight regain. If a properly constructed, nonobstructing stoma can enlarge, is it not reasonable to presume that one which is too small will also distend with time? A feeding jejunostomy, routinely inserted during gastric partitioning, permits one to wait for 6 months or even longer in patients with stomal obstruction and avoids the necessity for early reoperation. Such patients go home

feeding themselves through the jejunostomy. When they remain in hospital, the temptation to tackle the problem surgically is too great even for the most conservative surgeon.

Bariatric surgical data should always include absolute failure rates rather than average weight lost. For example, if two patients, each weighing 140 kg, had gastroplasties and one lost 50% (70 kg) while the other lost only 10% (14 kg), the *average* percentage lost would be 30% with a failure rate of 50%! Similarly, data should be presented as a percentage of weight lost (or, alternatively, as a percentage of excess weight lost) and not as kilograms lost. Using the preceding example, the *average* weight loss was 70 + 14 ÷ 2 = 42 kg but with a 50% failure rate. Kilograms lost are even more misleading when patients weighing over 140 kg (who tend to have, but do not necessarily sustain, huge weight losses in the early postoperative months) are compared with less obese patients weighing between 90 and 140 kg. Each report should list the number of patients with inadequate results, with failures defined as those patients who have lost less than 15% of their initial weight, at a minimum follow-up of 24 months, with patients lost to follow-up being included as failures. Adequate follow-up is essential. Many patients who lose much weight initially, fail to correct the poor eating habits that led to the operation and hence will regain lost weight if not seen monthly for dietary and exercise counselling.

Taking Deitel's gastroplasty group as a whole, one can illustrate the importance of looking critically at data. They did 110 gastroplasties over an unstated period of time. The first 56 patients were all failures because weight loss was inadequate and the procedure was abandoned. In the second group of 54 patients, only 5 were followed up for more than 1 year and the percentage of patients lost to follow-up is not given. Our data show a minimum technical failure rate of 20% at 24 months — it is higher if one includes patients who refuse to return for follow-up or those who require revision for inadequate weight loss. So, *at least* 11 patients (20% of 54) from Deitel's second group of patients will likely be failures in addition to all 56 patients from the first group. This adds up to 67 failures out of 110 for a *minimum* failure rate of 61% at 1 year.

On a more positive note, one must appreciate that certain modifications are necessary during the temporal development of surgical procedures. With time, we may come closer to perfection with gastric partitioning. Also, the success of nonoperative treatment for morbid obesity is less than 5%. Hence, even a failure rate of 60% in a surgical series still leaves 40% of patients operated on with adequate weight loss — an eightfold improvement over conservative therapy.

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Editors' note.—The response of Deitel and colleagues to this editorial appears in the Correspondence section of this issue (page 348).

Alkaline Reflux Gastritis

In this issue (page 337) Tassé and colleagues describe the collective experience of the Hôpital St-Luc, Montreal, in the operative management of patients putatively afflicted with the elusive (perhaps illusory) syndrome of alkaline reflux gastritis.

In one respect, this report is unusual because more than one third of the patients subjected to remedial operation had not undergone an earlier procedure involving the creation of a gastroenteric stoma. Indeed, in three, no previous intra-abdominal surgery of any sort had been performed. Under these circumstances, the diagnosis appears to have been established by the histologic demonstration of a "primary (as opposed to a secondary) reflux gastritis" — a distinction of which I was previously unaware — associated with the presence of typical symptoms, typical endoscopic findings and, presumably, the absence of other demonstrable abnormalities of the upper gastrointestinal tract or biliary tree.

In most respects, however, this report gives one a sense of *déjà vu* because it echoes the findings of so many of the earlier surgical series addressing the syndrome: for selecting patients for operation, a heavy reliance is placed on the presence or absence of typical symptoms and of those endoscopic and histologic indices supposedly specific for reflux; there is no quantitative assessment of reflux volume or composition per se; near universal failure of a variety of nonoperative treatment regimens is followed by near-universal success on short-term follow-up when upper intestinal content is shunted away, at least in theory, from the gastric mucus membrane by creating a Roux-en-Y limb. Indeed, the superlative results described in the present study, when added to those of earlier surgical reports, make it extremely tempting to declare an unequivocal Q.E.D. for the syndrome under consideration.

This would be a mistake. In the first instance, for reasons that are not entirely clear, these results contrast sharply with those reported in two recent series, equally carefully analysed. In one, patient selection was just as rigorous, yet the outcome was very different: only two thirds of patients subjected to remedial operation had complete or even partial relief of symptoms. Furthermore, when symptomatic, endoscopic or histologic predictors of success or failure were sought, few could be reliably identified on a statistical basis. Outcome seemed almost random in this regard. In the second study,[2] even less salutory results were reported: only 47% of patients achieved a satisfactory result from diversion. Patients with delayed gastric emptying preoperatively fared especially poorly, undoubtedly because a Roux-en-Y limb itself can cause marked and troublesome delays in gastric emptying.[3] It is of interest that 5 (22%) of the 23 patients in Tassé's series demonstrated gastric stasis postoperatively, although none required further operation. Most importantly, the objective indices that Tassé and associates accept as being specific to the syndrome may

not be as specific as many have claimed.[4] The typical endoscopic findings of mucosal edema and erythema almost invariably disappear following diversion, but, as noted, patients do not invariably improve. In those who do, the histologic features of the residual gastric pouch have been reported to revert towards normal,[5] to remain the same[6] or to worsen,[7] depending upon the histologic criteria used. The capacity of the residual gastric pouch to secrete hydrochloric acid, bile staining of the mucosa, even the demonstration of enterogastric reflux during endoscopy, all have been shown to be inaccurate predictors of outcome. Given these ambiguities and discrepancies, it is not surprising that some have questioned the very existence of the syndrome.[8]

In my opinion, the clinical diagnosis of alkaline reflux gastritis will be rendered scientifically acceptable and, therefore, clinically useful only after four general conditions have been satisfied. First, a wider appreciation must be gained of which symptoms can reasonably be ascribed to excessive enterogastric reflux. Tassé and colleagues may have hit upon them: the simultaneous complaints of mild burning epigastric pain associated with bilious vomiting. These are the only symptoms that are regularly induced when endogenous upper intestinal content from typically symptomatic patients is instilled into their residual stomachs[9] or are predictably relieved when reflux has been quantitatively eliminated by operation.[5,7]

Second, the existence of a specific clinical syndrome based on the relation between symptoms and excessive reflux must be rigorously established, by demonstrating that the syndrome always occurs in the presence of excessive reflux, never occurs in the absence of excessive reflux, is eliminated by eliminating excessive reflux and is induced by inducing excessive reflux. None of the major clinical series purporting to assess the syndrome even begin to address these conditions because not one has studied a simultaneous cohort of asymptomatic patients.

Third, the precise etiologic factors in upper intestinal content that are responsible for inducing symptoms (if, indeed, symptoms and reflux are related) must be clearly identified. As Tassé and colleagues indicate, several reasonable candidates are available although none can be implicated with certainty at present.

Finally, and most importantly, objective and discriminative diagnostic maneuvers must be developed to distinguish reliably those patients who have the syndrome from those who do not. Promising approaches are the use of provocative testing with either concentrated sodium hydroxide[10] or autologous intestinal content,[9] the measurement of intragastric bile-acid concentration[5] and the direct assessment of reflux volume by gamma scintigraphy following the injection of hydroxyiminodiacetic acid (HIDA) labelled with technetium-99m.[11] Because methodologies for these last two tests are well described and they can be done at most major hospital centres, I believe no patient should be subjected to remedial operation until an objective assessment of reflux volume is obtained by one or both of these modalities.

Until these conditions are satisfied, the syndrome of alkaline reflux gastritis cannot be considered a proven clinical entity and the prudent surgeon should approach remedial therapy, particularly in the absence of previous gastric surgery, with considerable circumspection.

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BOOKS RECEIVED

This list is an acknowledgement of books received. It does not preclude review at a later date.

A B C de chirurgie plastique et esthétique. Roger Amar. 81 pp. Illust. Masson, Paris, 1981. Prix non mentionné, broché. ISBN 2-225-74426-2.

Amputation Surgery and Rehabilitation: The Toronto Experience. Edited by John P. Kostuik and Robert Gillespie. 448 pp. Illust. Churchill Livingstone, Edinburgh; Academic Press Canada, Don Mills, Ont., 1981. $62.40. ISBN 0-443-08024-0.

Aneurysms: Diagnosis and Treatment. Edited by John J. Bergan and James S.T. Yao. 670 pp. Illust. Grune & Stratton, Inc., New York; Academic Press Canada, Don Mills, Ont., 1982. $70.85. ISBN 0-8089-1440-5.

Angiography of Spinal Column and Spinal Cord Tumors. Rene Djindjian. 258 pp. Illust. Thieme-Stratton Inc., New York; J.B. Lippincott Company of Canada, Toronto, 1981. Price not stated. ISBN 0-86577-025-5.

Cancer Medicine. James F. Holland and Emil Frei III. 2465 pp. Illust. Lea & Febiger, Philadelphia, 1982. $168. ISBN 0-8121-0603-2.

Chirurgie 81. Résumés des Tables Rondes des Forums et Communications du 83e Congrès Français de Chirurgie. Paris, 21 au 24 Septembre 1981. Textes publiés sous la direction de J.-Cl. Patel. 184 pp. Illust. Masson, Paris, 1981. Prix non mentionné, broché. ISBN 2-225-75010-6.

Endodontic Surgery. Edited by Donald E. Arens, William Ray Adams and Rolando A. DeCastro. 238 pp. Illust. Harper & Row, Philadelphia; J.B. Lippincott Company of Canada, Toronto, 1981. Price not stated. ISBN 0-06-140267-2.

Frontiers in General Surgery. Edited by D.W. Jirsch. 390 pp. Illust. F.A. Davis Company, Philadelphia, 1982. $35. ISBN 0-8036-5028.

General Surgery Therapy. Edited by Oliver H. Beahrs and Robert W. Beart, Jr. 509 pp. Illust. John Wiley & Sons, New York, 1981. $65. ISBN 0-471-09548-6.

Genitourinary Reconstruction with Prostheses. L.V. Wagenknecht, W.L. Furlow and J. Auvert. 213 pp. Illust. Thieme-Stratton Inc., New York; J.B. Lippincott Company of Canada, Toronto, 1981. Price not stated. ISBN 0-86577-028-X.

Management of Medical Problems in Surgical Patients. Mark E. Molitch. 795 pp. F.A. Davis Company, Philadelphia, 1982. $40. ISBN 0-8036-6286-6.

Manual of Cardiac Surgery II. Bradley J. Harlan, Albert Starr and Fredric M. Harwin. 347 pp. Illust. Springer-Verlag New York, Inc., 1981. $125. ISBN 0-387-90563-4.

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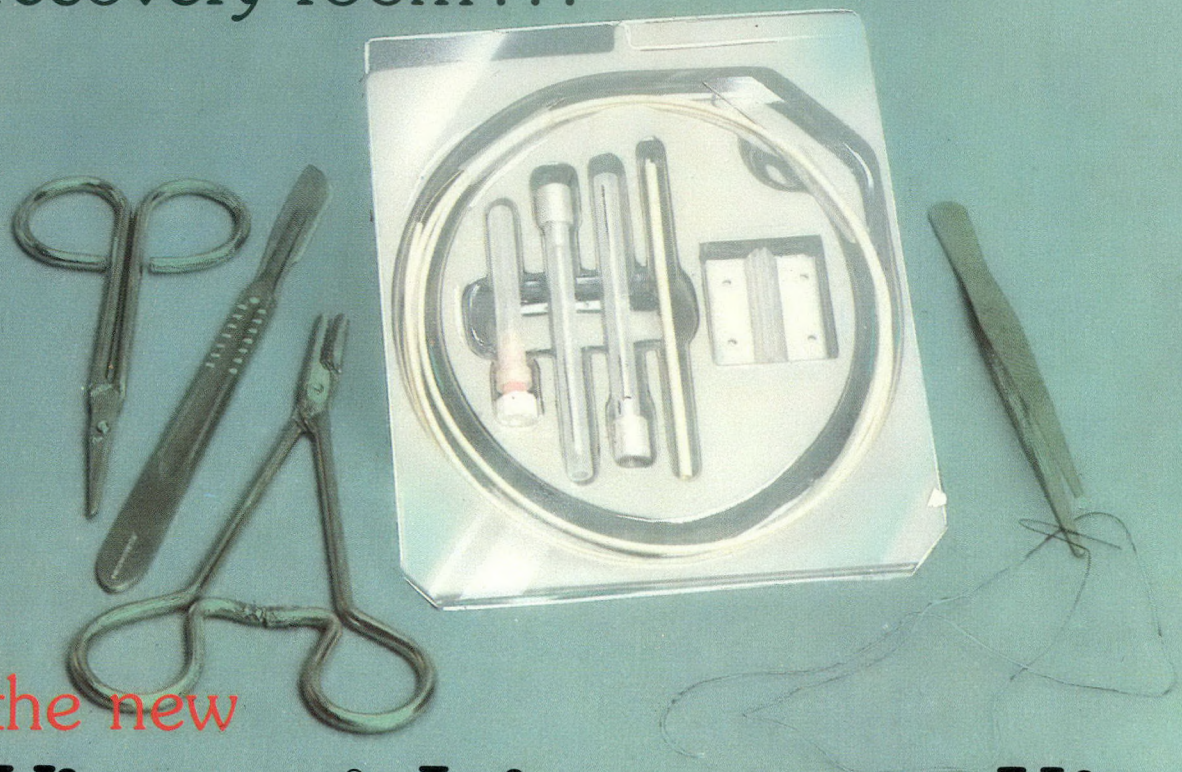

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Carcinoma of the Sigmoid: a Complication of Ureterosigmoidostomy

JULES CHARRON, MD, FRCS[C]* AND RÉJEAN DELISLE, MD†

The case of a 58-year-old man who had carcinomatous change in the sigmoid colon many years after ureterosigmoidostomy prompted the authors to review the French and English literature on the subject. They found 47 cases in addition to their own, and noted a dramatic increase in the last 10 years, even though ureterosigmoidostomy is now seldom used. The latency of this complication makes it pertinent today.

In the hope of determining the etiology of the complication, the authors studied the relation between the initial lesion for which the diversion was carried out and the nature of the colonic tumour that ensued, and have attempted to draw some conclusions. None of the current theories of pathogenesis seem satisfactory.

By underlining the early clinical manifestations, the authors attempt to promote earlier diagnosis and better chances of survival for patients with carcinoma of the colon after ureterosigmoidostomy.

L'apparition chez un homme de 58 ans de changements carcinomateux dans le côlon sigmoïde, plusieurs années après une urétéro-sigmoïdostomie a incité les auteurs à fouiller la documentation médication française et anglaise sur le sujet. Ils y ont découvert 47 cas additionnels et ont noté une augmentation spectaculaire au cours des 10 dernières années, en dépit du fait que l'urétéro-sigmoïdostomie soit rarement utilisée. La période de latence de cette complication en fait actuellement un sujet fort pertinent.

Dans l'espoir de déterminer l'étiologie de cette complication, les auteurs ont étudié le rapport pouvant exister entre la lésion initiale pour laquelle la dérivation fut pratiquée et la nature de la tumeur colique qui a suivi, et ils ont tenté d'en tirer des conclusions. Aucune des théories actuelles sur la pathogénèse de cette atteinte ne semble satisfaisante.

Soulignant les manifestations cliniques précoces, les auteurs essaient de favoriser un diagnostic plus hâtif et d'améliorer les chances de survie des patients atteints d'un cancer du côlon après une urétéro-sigmoïdostomie.

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Because the bowel wall adjusts readily to the constant presence of urine, bowel has been used extensively as a means of urinary diversion.[1,2] It has been used as a bladder and ureter substitute for almost a century and is the only tissue capable of transporting and storing urine with a minimum of complications.

Transplanting the ureters into the sigmoid colon is perhaps the oldest technique of closed urinary diversion. Its complications are well known and their prevention and management are well defined. However, the sigmoid mucosa, after a long period, becomes vulnerable to carcinomatous change. We recently treated a patient with carcinoma of the sigmoid colon after ureterosigmoidostomy and this prompted us to review the literature on the subject. We found an apparent increase in the incidence of carcinomatous change. Although the earliest case reported was by Hammer[3] in 1929, half of the 48 cases reported to date were found in the last 10 years (Table I). The incidence of this complication is impossible to determine and is obviously low, but we have been struck by the apparent delay in making the diagnosis, betraying a lack of awareness of the complication.

The mechanism by which ureterosigmoidostomy may cause carcinoma of the colon is presumptive and cannot be proven, even on a statistical basis, because of the paucity of cases. But the tendency of these lesions to develop near the anastomotic site and the occasional transitional cell carcinoma is strongly suggestive of a carcinogenic role of ureterosigmoidostomy.

Although the ileal conduit is now preferred as a means of urinary diversion, the latency of carcinoma resulting from ureterosigmoidostomy makes this complication a matter of current interest, since patients operated on more than 20 years ago may only now be vulnerable.

In an attempt to define the various facets of this complication, we report a recent case and analyse those

Table I—Incidence of Carcinomatous Change in the Sigmoid after Ureterosigmoidostomy

Period	No. of cases reported
1910–1930	1
1930–1940	0
1940–1950	2
1950–1960	3
1960–1970	18
1970–1980	23
Total	47

reported in the French and English literature.

Case Report

A 58-year-old white man had a history of chronic pain in the left flank and renal insufficiency from progressive hydronephrosis. He had refused operation for his bladder exstrophy until, at 38 years of age, he accepted an "internal" urinary diversion. A bilateral ureterosigmoidostomy was carried out using a Coffey I technique. Pyelonephritic changes were already present bilaterally and a small caliceal stone was identified on the left side. The blood urea nitrogen value was 49 mg/dl (17.5 urea, mmol/l).

Renal stones eventually developed bilaterally; a right pyelolithotomy and a left nephrolithotomy had to be done.

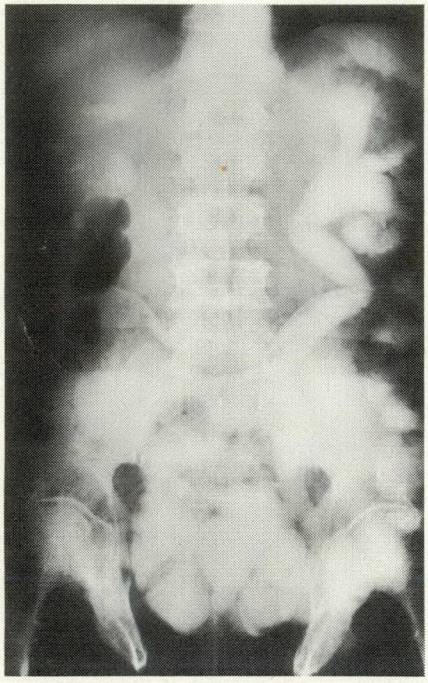

FIG. 1—Intravenous pyelogram showing dilatation of left collecting system and distribution of contrast medium in left colon and sigmoid.

At the age of 45 years he was again admitted to hospital because of colicky abdominal pain and vomiting, compatible with partial occlusion of the intestine. The blood urea nitrogen level was 42 mg/dl (15.0 urea, mmol/l) and there was mild acidosis.

Roentgenography after a barium enema revealed a mass, 4 cm in greatest dimension, in the lumen of the sigmoid, possibly at the level of the ureterosigmoid anastomosis. However, there were no signs of obstruction on the intravenous pyelogram, which showed good excretion of dye on both sides. Pyelonephritic changes had progressed slightly. The acute episode of occlusion subsided and the patient was discharged without further investigation. He was seen at irregular intervals for the following 12 years, when he complained again of abdominal pain. An intravenous pyelogram showed severe obstruction at the left ureterosigmoid anastomosis, demonstrated only by a drip infusion of dye (Fig. 1). The blood urea nitrogen level was 47 mg/dl (16.8 urea, mmol/l), creatinine 1.8 mg/dl (159 μmol/l) and hemoglobin 10.2 g/dl. The patient again refused surgical treatment. He was discharged and was not seen again until the present admission 1 year later.

He complained of persistent pain in the left flank, anorexia and weakness. Laboratory findings were as follows: serum creatinine 2.5 mg/dl (221 μmol/l), chloride 110 mmol/l and carbon dioxide 13.2 mmol/l, blood urea nitrogen 48 mg/dl (17.1 urea, mmol/l) and hemoglobin 10.1 g/dl. Intravenous pyelography by drip infusion showed progression of the pyelonephritic changes of the right kidney and an almost inactive left kidney.

The patient finally accepted operation in the hope that his pain would be alleviated. A double-barrelled cutaneous ureterostomy was chosen because the difficult personality of the patient made it imperative to do a diversion that was most likely to reduce the obstruction and necessitate the least postoperative care. Because the ureters were wide and tortuous from chronic distension, we thought that a bowel conduit might not relieve the obstruction adequately, and the patient would not accept a second procedure.

At operation the ureterosigmoid anastomoses were identified with surprising ease. The left anastomosis was very firm on palpation and suggested the presence of an impacted stone. The right anastomosis appeared soft and supple. The left anastomosis was widely resected, including a 3-cm segment of ureter and a large segment of sigmoid. A similar procedure was carried out on the right side.

The left ureter was brought out through a tunnel under the parietal peritoneum and the right ureter was brought out alongside the sigmoid mesentery; the possibility of volvulus was diminished by tenting a long segment of posterior peritoneum to the abdominal wall around the ureter.

A frozen section of the excised specimen from the left side showed a fibrotic and chronically inflamed lesion; there was no neoplasm. However, the permanent microscopic section revealed a moderately differentiated adenocarcinoma of the sigmoid colon, with superficial muscular invasion. This confirmed our earlier suspicion of a neoplasm (Figs. 2 and 3).

The patient refused more extensive bowel surgery. The findings on roentgenography after a barium enema and proctoscopy were normal. Kidney function was improved and the patient adapted remarkably well to the cutaneous ureterostomy.

Discussion

In the English and French literature we found, in addition to our own case, 47 cases of carcinoma of the colon in patients who had undergone a bilateral ureterosigmoidostomy for urinary diversion. Although the anastomotic technique may have varied, in no instance was an isolated segment of bowel used initially, thus permitting

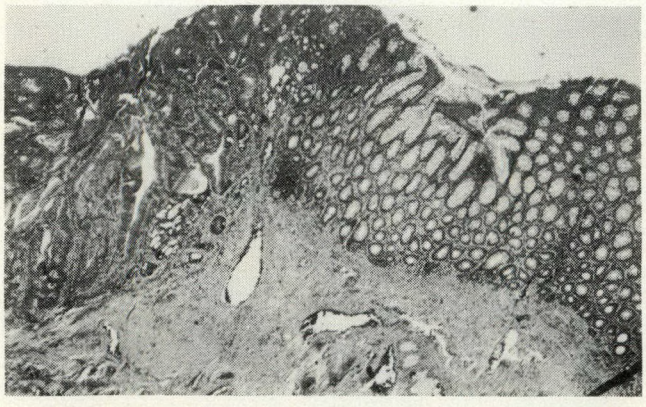

FIG. 2—Moderately differentiated adenocarcinoma originating from colonic mucosa. Area of submucosal scarring suggests site of ureteric anastomosis (hematoxylin-phloxine-saffron stain, reduced by 49% from ×25).

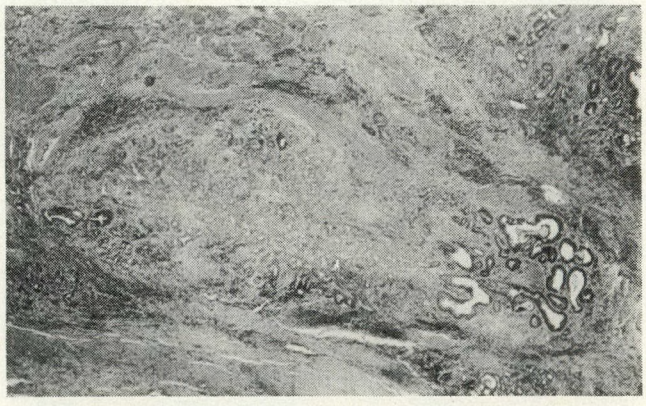

FIG. 3—Invasion of muscle layers of colon by adenocarcinoma (hematoxylin-phloxine-saffron stain, reduced by 49% from ×250).

free contact of urine with normal intestinal contents.

The cases could be divided into four groups:

- Thirty patients who underwent diversion for a benign condition (e.g., exstrophy of the bladder, urinary incontinence, trauma) and subsequently had carcinoma (Table II[3-25]).
- Ten patients who underwent diversion for a benign condition and subsequently had an adenoma (Table III[4,10,14,17,24,26-28]).
- Six patients who had diversion for a primary malignant condition and then had carcinoma (Table IV[9,29-32]).
- Two patients who had diversion for a primary malignant condition and who later had an adenoma (Table V[33,34]).

A male predominance of 2 to 1 in the benign group is in keeping with a much higher incidence of exstrophy of the bladder in men, as is the somewhat similar sex ratio in the group with malignant disease, where carcinoma of the bladder is much more common in men.

The age of the patients at the time of the initial operation is irrelevant and did not seem to influence the length of the latent period. However, it is important to note that this period varied from 10 to 46 years if the initial lesion was benign, but was much shorter, a few months to 9 years, if the initial lesion was malignant. This could be explained by the fact that patients with cancer are

Table II—Carcinoma of the Sigmoid Arising at or near Site of Bilateral Ureterosigmoidostomy for a Benign Condition

Author	Clinical indication	Sex	Age at operation, yr	Latency period, yr	Side of ureter involved by cancer
Hammer, 1929[3]	Bladder exstrophy	M	50	10	Trigone
Dixon and Weismann, 1948[4]	Bladder exstrophy	M	3	30	Left
Wilson, 1957[5]	Urinary incontinence	F	16	31	Left
Aldis, 1961[6]	Bladder exstrophy	F	18	15	Left
Sugg, 1962[7]	Bladder exstrophy	F	5	15	Left
Kozak and associates, 1966[8]	Bladder exstrophy	M	2	15	Left
Urdaneta and associates, 1966[9]	Bladder exstrophy	F	4	29	—
	Interstitial cystitis	F	21	17	Right
Kille and Glick, 1967[10]	Urinary incontinence secondary to epispadias	M	10	16	Left
Richter and Ginsberg, 1967[11]	Bladder exstrophy	F	2	23	Left
Oetjen and associates, 1970[12]	Bladder exstrophy	M	3	23	Left
Whitaker and associates, 1971[13]	Rupture of posterior urethra	F	7	15	Left
	Bladder exstrophy	M	31	27	Right
	Bladder exstrophy	M	19	20	Bilateral
Brekkan and associates, 1972[14]	Bladder exstrophy	F	3	20	Right
	Bladder exstrophy	M	6	38	Right
	Bladder exstrophy	M	6	41	Trigone
Tank and associates, 1973[15]	Bladder exstrophy	M	9	46	Bilateral
Mueller and Thornbury, 1973[16]	Bladder exstrophy	M	9	46	Bilateral
Lien, 1973[17]	Traumatic rupture of urethra	M	7	26	—
Preissig and associates, 1974[18]	Gunshot wound	M	8	26	Left
Shapiro and associates, 1974[19]	Bladder exstrophy	F	4½	14	—
Harguindey and associates, 1975[20]	Bladder exstrophy	M	2	24	—
Rivard and associates, 1975[21]	Bladder exstrophy	M	15	23	—
Carswell and associates, 1976[22]	Bladder exstrophy	F	2½	23½	Right
Besancenez and associates, 1976[23]	Bladder exstrophy	M	4	25	Right
Potet and associates, 1977[24]	Bladder exstrophy	M	7	25	Right
Parsons and associates, 1977[25]	Bladder exstrophy	M	3	26	—
	Bladder exstrophy	M	1½	33½	Right
Present case	Bladder exstrophy	M	38	20	Left

Table III—Benign Adenoma of Sigmoid Arising at or near Site of Bilateral Ureterosigmoidostomy for a Benign Condition

Author	Clinical indication	Sex	Age at operation, yr	Latency period, yr	Side of ureter involved
Dixon and Weismann, 1948[4]	Bladder exstrophy	M	3	30	Right
Markowitz and Koontz, 1966[26]	Bladder exstrophy	M	1	14	Left
	Bladder exstrophy	F	2	15	Right
	Bladder exstrophy	M	1	18	Right
Kille and Glick, 1967[10]	Urinary incontinence with epispadias	M	4	15	—
Haney and McGarity, 1971[27]	Bladder exstrophy	F	6	38	Left
Brekkan and associates, 1972[14]	Bladder exstrophy	F	3	20	Left
Lien, 1973[17]	Bladder exstrophy	F	1½	23½	—
Lasser and Acosta, 1975[28]	Bladder exstrophy	M	10	27	Bilateral
Potet and associates, 1977[24]	Bladder exstrophy	F	9	25	Bilateral

more susceptible to carcinogens.[35]

The histopathologic types of tumour varied little. There were only four varieties: 33 adenocarcinomas, 12 adenomas, 2 transitional cell carcinomas and 1 scirrous carcinoma. The nature of the initial lesion requiring urinary diversion bore no relation to the kind of neoplasm developing at the anastomotic site. Adenocarcinomas occurred with bladder exstrophy as well as with transitional cell carcinoma of the bladder and adenomas developed with either of these lesions, even with urinary incontinence.

Statistical evidence of a cause–effect relation between long-term ureterosigmoidostomy and carcinoma of the colon has been suggested by the work of Urdaneta and associates.[9] According to United States statistics, the incidence of carcinoma of the colon in persons 45 years of age is 24 per 100 000.[9] However, the 83 patients with ureterosigmoidostomy operated on by Urdaneta's group showed an incidence of 13 300 per 100 000.

The length of time after ureterosigmoidostomy is more important to the development of carcinoma of the sigmoid than the age of the patient. Probably the better understanding and control of complications of ureterosigmoidostomy, such as infection and acidosis, leading to longer survival, have permitted this relatively rare complication to occur.

Speculation concerning the etiology of neoplasia at the site of a ureterosigmoid anastomosis revolves around three factors: (a) the role of urine alone, (b) the role of urine mixed with fecal material and (c) the role of bowel-wall changes from ureteric prolapse into the lumen.

The possibility of a urinary carcinogen can be excluded, because carcinomas have developed near a "dry" anastomosis, following a nephrectomy, leaving ureteral stumps behind. This happened in two of the cases after operations performed 16 years[25] and 8 years[12,22] earlier. Scott and Boyd[36] could not induce carcinoma in the bowel of dogs using β-naphthylamine during a 20- to 30-month period of irrigation. Although this time period may not be comparable, the use of a known carcinogen as an added stimulant makes the results of this experiment more important. In spite of the widespread diffusion of urine in the colon, the proximity of the tumour to the anastomosis is notable.

The association of urine and sigmoid fecal material may be the common denominator, as we are not aware of the development of bowel carcinoma in ileal or isolated sigmoid conduits.

As a third possibility, the ureteral nipple may be irritative like a polyp in the lumen of the bowel. Repeated microtrauma from feces could induce malignant transformation.[15] However, we could not find any scientific basis for this hypothesis; the true etiology of this cancer remains a mystery.

We believe that the paucity of reports of bowel tumour after ureterosigmoidostomy does not reflect its true incidence. Better long-term follow-up of these patients with an awareness of this complication is required.

Although there are no pathognomonic symptoms and signs, there are some clinical indications that may suggest the diagnosis. Most patients have had one or all of the following at an early stage: vague abdominal pain suggestive of a ureteral stone, proctorrhagia sometimes leading to anemia, and recent obstructive changes at one or both ureterosigmoid anastomoses, demonstrated by intravenous pyelography, rapidly interfering with kidney function. These symptoms suggest neoplasia of the sigmoid and must be so considered until proven otherwise.

Intestinal obstruction is a late stage that betrays irreversible damage and is associated with poor survival.

Increased awareness of carcinomatous change after ureterosigmoidostomy will result in earlier diagnosis if abdominal pain, proctorrhagia and obstructive changes are rapidly inves-

Table IV—Carcinoma of the Sigmoid Arising at or near Site of Bilateral Ureterosigmoidostomy for Malignant Condition

Author	Clinical indication	Sex	Age at operation, yr	Latency period, yr	Side of ureter involved by cancer
Riches and Page, 1956[29]	Transitional cell carcinoma of bladder	F	61	1	Left
Massachusetts General Hospital, 1958[30]	Transitional cell carcinoma of bladder	M	55	8	Right
Scheinman, 1961[31]	Transitional cell carcinoma of bladder	M	42	9	Left
Amar, 1961[32]	Transitional cell carcinoma of bladder	M	46	4	Left
	Transitional cell carcinoma of bladder	M	58	7 mo	Left
Urdaneta and associates, 1966[9]	Epidermoid carcinoma	M	55	9	Right

Table V—Benign Adenoma of the Sigmoid Arising at or near Site of Ureterosigmoidostomy for a Malignant Lesion

Author	Clinical indication	Sex	Age at operation, yr	Latency period, yr	Side of ureter involved by cancer
Gillman, 1964[33]	Transitional cell carcinoma of bladder	—	75	7	Left
MacGregor, 1968[34]	Transitional cell carcinoma of bladder	M	57	5	Left

tigated. The condition of patients who have undergone ureterosigmoidostomy should be monitored by proctoscopy, roentgenography after barium enema and intravenous pyelography, periodically for life. The longer the time interval after surgery, the more critical the assessment becomes. After 10 years, in asymptomatic patients, a double contrast barium enema, done every other year, alternating with intravenous pyelography, should detect early neoplastic changes.

Simple nephrectomy for a nonfunctioning kidney is inadequate, as many examples of carcinomatous change around the ureteral stump have been reported.[4,12-17,21,22,24,25] A "collarette" of colon should be included in the excision and more radical colonic resection is necessary if malignant change has occurred.

It has been suggested by Carswell and associates[22] that in view of this complication a reappraisal of the indications for ureterosigmoidostomy should be made and that it should be restricted to patients over 50 years of age. We believe that ureterosigmoidostomy in younger patients carries the risk of neoplasia but may be done provided that follow-up is regular and thorough.

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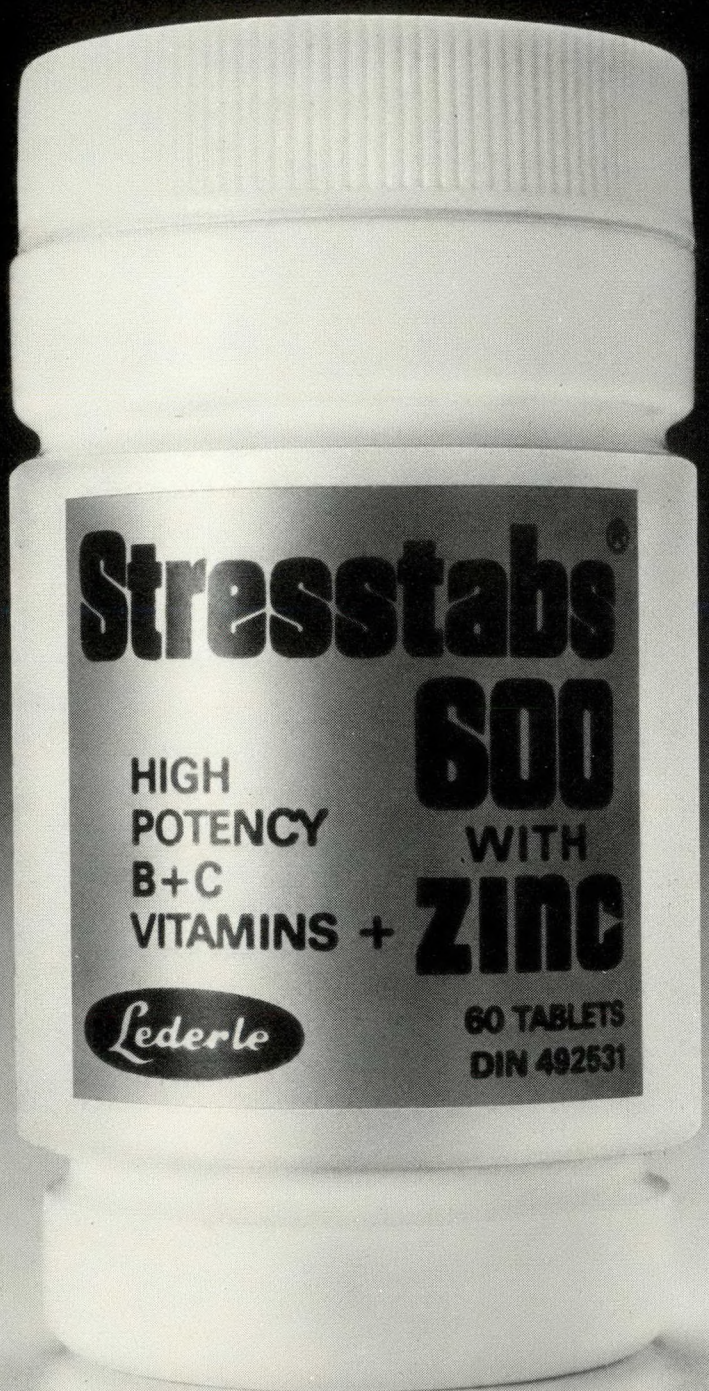

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Dysfunction of the Detrusor and Urethra in Multiple Sclerosis: the Role of Drug Therapy

SAID A. AWAD, MB, B CH, FRCS[C],* JAMES W.L. WILSON, M SC, MD,† JANET FENEMORE, RN† AND H. GEORGE KIRULUTA, MB, B CH, FRCS[C]‡

Urinary tract disorders secondary to multiple sclerosis are common. In this series of 24 patients with multiple sclerosis, 5 had normal function of the detrusor, 3 had detrusor hypotonicity and 16 had detrusor hyperreflexia. The proximal urethra was evaluated using radiologic and electromyographic techniques. These studies showed that 5 patients had a normal urethra, 15 had some degree of somatic dyssynergia and 3 had sympathetic dyssynergia. Detrusor hyperreflexia with somatic dyssynergia was found in 11 patients and was the most common pattern. The therapeutic response to standard pharmacologic preparations was also evaluated. The regimen was based on the clinical and urodynamic findings for each patient. Dicyclomine hydrochloride was the drug of choice for detrusor hyperreflexia, bethanechol chloride for hypotonicity, dantrolene sodium for somatic dyssynergia and phenoxybenzamine hydrochloride for sympathetic dyssynergia. The authors found that most of their patients were amenable to drug therapy, the exception being those with advanced neurologic lesions.

Les problèmes des voies urinaires secondaires à une sclérose en plaques sont fréquents. Parmi un groupe de 24 patients souffrant de sclérose en plaques, 5 avaient un muscle vésical fonctionnant normalement, 3 avaient une hypotonicité de ce muscle et 16 avaient une hyperréflexie. L'urètre proximale a été examinée par des moyens radiologiques et électromyographiques. Ces études ont révélé que 5 malades avaient une urètre normale, 15 souffraient à divers degrés de dyssynergie somatique et 3 avaient une dyssynergie sympathique. Une hyperréflexie du muscle vésical et une dyssynergie sympathique ont été retrouvées chez 11 patients et ceci représente le tableau le plus fréquent. On a également évalué l'activité thérapeutique des préparations pharmacologiques de référence. Le régime thérapeutique a été adapté aux résultats des examens cliniques et urodynamiques chez chaque patient. Le chlorhydrate de dicyclomine a été utilisé comme médicament de choix pour l'hyperréflexie du muscle vésical, le chlorure de béthanécol pour l'hypotonicité, le dantrolène sodique pour la dyssynergie somatique et le chlorhydrate de phenoxybenzamine pour la dyssynergie sympathique. Les auteurs ont trouvé que, à l'exception de ceux qui avaient des lésions neurologiques avancées, la plupart de leurs malades ont répondu à la médication.

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The incidence of urinary tract dysfunction in patients with multiple sclerosis has been documented in previous reports.[1,2] Other investigators[3-8] have also reported on the abnormalities of detrusor and sphincter function in patients with this disease, with particular emphasis on the results of urodynamic testing. However, there have been fewer reports on the results of pharmacologic treatment in patients with multiple sclerosis who have urinary dysfunction.[9] In this paper we evaluate the treatment of 24 patients with urinary dysfunction as a result of multiple sclerosis.

Patients and Methods

Twenty-four patients (19 women, 5 men) with multiple sclerosis have been referred to the urodynamic unit of the Kingston General Hospital over the past 5 years for evaluation and management of urinary tract disorders. The mean age was 44.8 years (range from 23 to 62 years) and the mean duration of symptoms of multiple sclerosis was 8.6 years (range from 3 to 20 years). The symptoms of urinary dysfunction present in these patients are listed in Table I. Infection, either chronic or recurrent, was noted in 11 patients.

Patients were assessed on the basis of their history, the findings on general physical and neurologic examinations and intravenous pyelography, the results of frequent urine cultures and determination of blood urea nitrogen and creatinine levels. Cystoscopy was done only when indicated, for example in those with hematuria or urinary lithiasis.

Usually, the urodynamic evaluation was carried out when urine was sterile. Cystometrograms were obtained with water as the filling medium at a rate of 25 ml/min using a pump with the patient supine. Because of the lack of standardization in interpreting the cystometrogram, the criteria used in this study will be listed. The cystometrogram was con-

Table I—Symptoms in Patients with Multiple Sclerosis

Symptom	No. of patients (%)
Incontinence	13 (57)
Hesitancy	8 (35)
Retention	8 (35)
Frequency	8 (35)
Urgency	7 (30)
Terminal dribbling	1 (4)

sidered normal when bladder capacity was 300 to 700 ml with a minimal rise in intravesical pressure during filling until full capacity was reached.[10] In addition, patients had normal perception of bladder fullness and when a detrusor contraction occurred, the patient was aware of its presence and was able to inhibit it rapidly. Detrusor hyperreflexia was diagnosed if a detrusor contraction occurred when the bladder contained less than 300 ml of water. On the other hand, the bladder was considered hypotonic if its capacity was greater than 700 ml, the patient was unaware of bladder fullness and there was no change in intravesical pressure even at full capacity or with a detrusor contraction.

The proximal urethra was evaluated simultaneously using electromyography of the external sphincter. Needle electrodes were placed transperineally a few millimetres lateral to the external urinary meatus in women and into the bulbospongiosus muscle in men, and the electromyographic activity was monitored by an audio-amplifier and an oscilloscope. Muscle activity was recorded using a "leaky integrator" (i.e., with an exponential decay) of the electromyographic signals. Normally, electromyographic activity should disappear during detrusor contraction.[11] Persistent muscle activity indicated somatic dyssynergia. No reduction in activity was an indication of severe dyssynergia, reduced or intermittent activity signified partial dyssynergia. When there was obstruction of the lower urinary tract and somatic dyssynergia was either absent or very mild (seven patients), a voiding cystourethrogram was obtained before and after the intravenous administration of 5 mg of phentolamine, an α-adrenergic blocking agent, to rule out the presence of "sympathetic dyssynergia".[12] Relief of the obstruction after giving phentolamine, as shown radiologically by an increase in the diameter of the proximal urethra during voiding or a decrease in postvoiding residual urine, indicated that the patient had sympathetic dyssynergia.

The results of urodynamic testing served as a guideline for pharmacologic treatment as described by Awad and colleagues.[13] Briefly, the antispasmodic dicyclomine hydrochloride (60 to 100 mg/d) was used for the hyperreflexic bladder. One patient was unable to tolerate dicyclomine so propantheline bromide was used. Bethanechol chloride (100 to 200 mg/d) was used for the hypotonic bladder. Dantrolene sodium was given in divided doses increasing from 50 mg/d to 100 mg/d (while liver function was monitored), often with diazepam (5 to 20 mg/d), in patients with somatic dyssynergia. Phenoxybenzamine hydrochloride (Dibenzyline) (10 to 20 mg/d) was given when sympathetic dyssynergia was considered to be an etiologic factor in urethral obstruction.

Table II—Urodynamic Findings and Treatment in 24 Patients with Multiple Sclerosis

Patient no.	Age, yr	Sex	Detrusor urinae	Sphincter	Treatment	Response to drugs
1	55	M	Normal	Somatic dyssynergia (severe)	Dantrolene sodium	Fair
2	35	F	Normal	Somatic dyssynergia (severe)	Dantrolene	Good
3	32	M	Normal	Somatic dyssynergia (partial)	None	—
4	53	F	Normal	Somatic dyssynergia (partial)	Dantrolene + diazepam	Poor
5	58	F	Normal	Normal	None	—
6	49	M	Hyperreflexic	Normal	Dicyclomine hydrochloride	Good
7	62	F	Hyperreflexic	Normal	Dicyclomine	Good
8	43	F	Hyperreflexic	Normal	Dicyclomine	Poor
9	40	F	Hyperreflexic	Normal	Propantheline bromide	Good
10	57	F	Hyperreflexic	Somatic dyssynergia (partial)	Dicyclomine	Good
11	39	F	Hyperreflexic	Somatic dyssynergia (partial)	Dicyclomine	Good
12	60	F	Hyperreflexic	Somatic dyssynergia (partial)	Dicyclomine	Poor
13	43	F	Hyperreflexic	Somatic dyssynergia (partial)	None	—
14	45	M	Hyperreflexic	Somatic dyssynergia (partial)	Dantrolene	Poor
15	46	F	Hyperreflexic	Somatic dyssynergia (partial)	Indwelling catheter	—
16	44	F	Hyperreflexic	Somatic dyssynergia (severe)	Dicyclomine + dantrolene	Good
17	56	F	Hyperreflexic	Somatic dyssynergia (severe)	Dantrolene + diazepam	Fair
18	27	F	Hyperreflexic	Somatic dyssynergia (severe)	Dicyclomine	Poor
19	23	F	Hyperreflexic	Somatic dyssynergia (severe)	Dicyclomine	Lost to follow-up
20	52	F	Hyperreflexic	Sympathetic dyssynergia	Dibenzyline	Good
21	49	M	Hyperreflexic	Sympathetic + somatic dyssynergia (severe)	Indwelling catheter	—
22	55	F	Hypotonic	Sphincter function indeterminable	Bethanechol chloride	Poor
23	28	F	Hypotonic	Sphincter function indeterminable	Bethanechol + dantrolene	Good
24	51	F	Hypotonic	Sphincter function indeterminable	Intermittent catheterization	—

Results

The urodynamic characteristics of each patient and the response to pharmacologic treatment are set forth in Table II.

The cystometrogram revealed three patterns of detrusor function: normal (5 patients), hyperreflexic (16 patients) and hypotonic (3 patients). The commonest abnormality was detrusor hyperreflexia in 16 of the 24 patients. Bladder capacities ranged between 50 and 300 ml (mean 130 ml).

In three patients with detrusor hypotonicity, the presence of somatic dyssynergia could not be determined because detrusor contraction was absent. Eleven of the 15 patients with somatic dyssynergia had associated detrusor hyperreflexia, and of the 16 patients who had detrusor hyperreflexia, 5 did not have somatic dyssynergia (Table II). Thus, the two commonest abnormalities were frequently, but not always, associated. Three patients showed evidence of sympathetic dyssynergia in radiologic studies, one as an isolated abnormality, one combined with somatic dyssynergia and one with detrusor hypotonicity. No patients had pyelographic evidence of hydroureter or hydronephrosis secondary to outlet obstruction.

The therapeutic regimen for each patient (Table II) was based on the urodynamic findings. The antispasmodic dicyclomine was used to treat 10 of 16 patients with detrusor hyperreflexia. Five had good results, which meant that urinary incontinence, if present, disappeared, and both urgency and frequency appreciably decreased although none of the patients became asymptomatic. In three patients with minimal or no improvement, treatment was considered to have failed. One patient had a good response to propantheline after she failed to tolerate dicyclomine. One patient was lost to follow-up. In general, the best results were obtained when the bladder capacity was greater than 200 ml.

Of three patients with a hypotonic bladder, two were treated with bethanechol and one responded well. The responding patient was assumed to have a spastic external sphincter in association with limb spasticity so dantrolene was added to the regimen, resulting in a decrease of postvoiding residual urine from more than 200 ml to less than 100 ml. The third patient, whose urinary retention was associated with acute exacerbation of multiple sclerosis, was treated by intermittent catheterization rather than by bethanechol; her bladder function gradually improved. The number of patients treated with bethanechol in this series was too small to warrant any conclusion as to its value.

Of the patients with somatic dyssynergia, 1 refused treatment and 6 of the remaining 15 were treated with dantrolene sodium; 2 of the 6 also received diazepam. Somatic dyssynergia was not treated unless the patient had symptoms of obstruction and large volumes of residual urine. Two of the six treated patients had good results, symptoms lessened and residual urine decreased by approximately 40%. Two patients had fair results with subjective improvement in symptoms but without a decrease in residual urine volume and two had poor results with no improvement.

Of the three patients with evidence of sympathetic dyssynergia, one had the isolated lesion and was treated with Dibenzyline with a good response. Residual urine volume decreased from 250 ml to 50 ml and there was also symptomatic improvement. The patient with associated detrusor hypotonicity was unable to tolerate the drug because of postural hypotension and the patient with combined sympathetic and somatic dyssynergia refused treatment and required placement of an indwelling catheter. The response to the various drugs is summarized in Table III. Overall, of the 17 patients treated with drugs and available for follow-up, the response was good in 9, fair in 2 and poor in 6.

Discussion

The spectrum of symptoms found in our patients correlates with that presented by Bradley and colleagues.[3] However, in our series there were slightly more obstructive symptoms (hesitancy and large postvoiding residual urine volume) and fewer irritative symptoms (frequency and urgency) than in their series. Our results of urodynamic testing also agree closely with Bradley's, particularly the cystometric findings; both showed the predominance of detrusor spasticity. This contrasts with the findings in Schoenberg's series,[8] in which slightly more than half the patients showed either hyporeflexic or areflexic detrusor muscles. Also, the combined lesion of spastic detrusor and somatic dyssynergia was encountered most frequently in our series (11 of 24 patients) in contrast to Schoenberg and colleagues[8] who reported only 13 of 58 patients in this category. Although detrusor hyperreflexia and somatic dyssynergia were commonly associated, several patients had one abnormality without the other, suggesting that the pathogenesis of these dysfunctions differs.

When detrusor hyperreflexia was associated with somatic dyssynergia, the abnormality that appeared to be the predominant cause of the patient's symptoms was treated first. This was often the detrusor hyperreflexia. Of the 11 patients with the combined abnormality, 5 were treated with dicyclomine, 2 with dantrolene and 1 with both drugs. We preferred dicyclomine for detrusor hyperreflexia because it is better tolerated than propantheline. However, one patient in this series tolerated propantheline better than dicyclomine. Generally, the results with dicyclomine were better if bladder capacity was greater than 200 ml. There was no apparent increase in the volume of postvoiding residual urine following dicyclomine therapy even in the presence of somatic dyssynergia.

Two patients who had severe detrusor hyperreflexia (capacity less than 100 ml) and severe somatic dyssynergia required an indwelling Foley catheter. Both patients needed long-term nursing care because of the severity of their neurologic lesion.

Table III—Response to Drug Therapy

Drug	No. of patients	Result		
		Good	Fair	Poor
Dicyclomine hydrochloride	9	5*	—	4†
Dantrolene sodium	7	3*	2	2
Dibenzyline	2	1	—	1
Bethanechol chloride	2	1*	—	1

*2 patients had combination drugs, 1 dicyclomine + dantrolene and 1 bethanechol + dantrolene.
†1 patient failed to tolerate dicyclomine but responded well to propantheline bromide.

Obviously, treatment goals had to be adjusted to take into account the nature of the handicap. The minimally handicapped person could achieve near-normal bladder function but the severely handicapped, because of poor mobility, limb spasticity and deformities, may have great difficulty achieving urinary continence and normal voiding habits. In the rapidly deteriorating patient or one in relapse, re-evaluation and subsequent adjustment of the therapeutic regimen was necessary; this occurred in two patients as symptoms changed. Recovery of bladder function following a relapse was also seen.

In summary, bladder and urethral dysfunction in patients with multiple sclerosis is amenable to pharmacologic therapy. The regimen should be drawn up on the basis of clinical symptoms and the urodynamic findings. The majority of patients did well with this approach but there is a small group of patients with advanced neurologic lesions whose lower urinary tract dysfunction fails to respond to pharmacologic therapy.

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Lymphadenectomy for Testicular Carcinoma

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Since 50% of patients with testicular tumour have retroperitoneal metastases at the time of presentation, there has been little argument that the area of primary drainage requires further treatment following orchiectomy. Patients with nonseminomatous germ cell tumours in stages A and B are candidates for retroperitoneal lymphadenectomy because this procedure is of therapeutic value and allows accurate staging of the disease. The development of extremely effective modern, cyclic, multidrug chemotherapy for extensive nonseminomatous testicular tumour has led to a rethinking of the role of lymphadenectomy in treating testicular cancer. Clinical staging is still not 100% accurate even with modern techniques. Lymphadenectomy, therefore, continues to be an important step in managing patients with nonseminomatous germ cell tumours. These facts are endorsed by the authors' review of 72 cases of nonseminomatous germ cell tumours managed at McGill University teaching hospitals over a 10-year period. The authors also discuss the indications for retroperitoneal dissection.

Comme 50% des patients atteints de tumeurs testiculaires présentent des métastases rétropéritonéales, on a rarement mis en doute la nécessité d'un traitement d'appoint de la région de drainage primaire à la suite d'une orchiectomie. Les patients porteurs de tumeurs à cellules germinales non séminomateuses de stade A et B sont candidats pour une lymphadénectomie rétropéritonéale car cette intervention possède une valeur thérapeutique en plus de permettre d'établir de façon précise le stade de la maladie. Le développement de régimes de polychimiothérapie cyclique extrêmement efficaces dans les cas de tumeurs testiculaires non séminomateuses étendues, a provoqué une remise en question du rôle de la lymphadénectomie dans le traitement des cancers testiculaires. Le stade clinique ne peut encore être établi avec 100% de précision, même avec les techniques modernes. En conséquence, la lymphadénectomie demeure une étape importante dans le traitement des patients souffrant de tumeurs à cellules germinales non séminomateuses. Ces faits trouvent appui dans l'étude faite par les auteurs de 72 cas de tumeurs à cellules germinales non séminomateuses traités dans les hôpitaux affiliés à l'Université McGill au cours d'une période de 10 ans. Les auteurs commentent également les indications de la dissection rétropéritonéale.

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The role of the urologic surgeon in the initial recognition and removal of a testicular tumour is not disputed. But subsequent surgical management is more controversial, particularly because the development, in recent years, of extremely effective chemotherapeutic regimens for advanced testicular cancer[1,2] has led to a reconsideration by oncologists of the indications for, and extent of, retroperitoneal lymphadenectomy. A review of the history, rationale and results of this surgical procedure is timely.

At the turn of this century, Most[3] and Cuneo[4] emphasized that the primary lymphatic drainage of the testis

was to the region of its embryonic origin. The first successful retroperitoneal lymphadenectomy was performed by Cuneo in 1906.[5] In 1948, Lewis[6] reported on 169 retroperitoneal lymphadenectomies performed without a single death and in the late 1950s, Patton and Mallis[7] advocated bilateral lymphadenectomy for testicular cancer.

Ray and colleagues[8] in 1974 reported on the predictable primary lymphatic drainage from the testes. Drainage from the right testis is first to the interaortocaval nodes, then to the precaval, preaortic, paracaval and right common iliac nodes in that order. Subsequently, the left para-aortic and left iliac nodes may be involved. Drainage from the left testis is, in sequence, to the upper left paraaortic nodes (between the left renal artery and the superior mesenteric artery), the preaortic nodes and the left common and external iliac nodes. Subsequent drainage may be to the interaortocaval, precaval, paracaval and right common and external iliac nodes. Ray and associates also observed that most, but not all, lymph nodes draining the testes are filled when bipedal lymphangiography is done and that lymphangiographic crossover from right to left is common and may be immediate, whereas crossover from left to right is rare and probably occurs after the left primary nodes are filled with tumour.

Approximately 50% of patients with nonseminomatous germ cell tumours have retroperitoneal lymph-node metastases at the time of surgical exploration (Table I[9-13]). Our experience is similar. In spite of modern, sophisticated diagnostic techniques, clinical staging is still far from precise. Serum tumour markers (α-fetoprotein and β-human chorionic gonadotropin) give false-negative results in at least 15% of cases.[14,15] Bipedal lymphangiography is unreliable in demonstrating retroperitoneal lymph-node metastases, and computerized axial tomography and ultrasonography consistently fail to demonstrate small metastatic deposits in the retroperitoneum.[16] Since untreated retroperitoneal metastases result in the patient's death, more accurate clinical staging is necessary to identify patients who require additional therapy following orchiectomy. Retroperitoneal lymphadenectomy, to date, is the most accurate staging procedure available.

Currently in North America retroperitoneal lymphadenectomy is favoured over radiotherapy for treating the primary lymphatic drainage area of nonseminomatous germ cell tumours because of its value in staging and because of its therapeutic efficacy. Although the results of numerous uncontrolled studies cannot be used as a reliable comparison, the survival rates in patients subjected to retroperitoneal lymphadenectomy, with or without adjuvant chemotherapy, in stages A (tumour confined to scrotum) and B (metastatic disease in retroperitoneum only) are impressive (Table II[17]); these results are at least as good as those in which radiotherapy has been used.[18-20] The strong preference in North America for retroperitoneal lymphadenectomy following orchiectomy for nonseminomatous germ cell tumours is based on the demonstrated effectiveness with which regional control can be achieved without adjuvant chemotherapy. Local recurrence following meticulous lymphadenectomy is rare (Table III[17]). The effectiveness of combined chemotherapy in stage C (metastatic disease beyond the retroperitoneum) tumours has been well documented.[1,2] It is also well recognized that because the maximum tolerable doses of the drugs must be given for optimal effect, their use may be compromised by permanent bone-marrow damage

Table II—Nonseminomatous Germ Cell Tumours: Tumour Free Survival at 24 Months* of Patients Treated Consecutively by Retroperitoneal Lymphadenectomy since 1974

Series[17]	Stage, no./total no. (%)		
	A	B	A + B
Staubitz	21/22 (95)	15/16 (94)	36/38 (95)
Donohue	57/57 (100)	54/55 (98)	111/112 (99)
Skinner	42/42 (100)	32/35 (91)	74/77 (96)

*Relapse after 24 mo is extremely rare.

Table III—Nonseminomatous Germ Cell Tumours: Recurrence after Meticulous Lymphadenectomy without Radiotherapy

Series[17]	Stage, no. of patients			Retroperitoneal recurrence within 24 mo,* no. of patients
	A	B	A + B	
Donohue	57	55	112	1
Staubitz	22	16	38	0
Skinner	42	35	77	0
Total	121	106	227	1 (0.44%)

*Relapse after 24 mo is extremely rare.

Table I—Incidence of Retroperitoneal Lymph-Node Metastases in Patients with Nonseminomatous Germ Cell Tumours of Testis Subjected to Abdominal Exploration

Series	Primary tumour type, no./total no. (%)			
	Embryonal carcinoma	Teratoma	Teratocarcinoma	Choriocarcinoma
Thompson and colleagues, 1961[9]	10/13	←	22/29	→
Ekman and colleagues, 1964[10]	←		29/61	→
Robson and colleagues, 1965[11]	←		12/38	→
Whitmore, 1968[12]	24/45	1/3	←	16/51 →
Maier and colleagues, 1969[13]	←		97/206	→
Total	←		211/446 (47)	→

secondary to previous radiotherapy. For this reason and because excellent results can be obtained by lymphadenectomy with or without adjuvant chemotherapy and without radiotherapy, the majority of medical and surgical oncologists treating testicular cancer in North America omit radiotherapy as a primary treatment in nonseminomatous germ cell tumours. The admittedly high rate of infertility due to loss of ejaculation following the procedure is a relatively small price to pay for such an excellent chance of cure. It should be emphasized that erectile potency is not affected by the operation nor is the ability to experience orgasm.

Retroperitoneal Lymphadenectomy

Indications

Not all patients with testicular carcinoma are necessarily candidates for this procedure. Since localized seminoma (stages A and B) can be cured by radiotherapy, most patients with pure seminoma do not undergo retroperitoneal lymphadenectomy. However, all patients with nonseminomatous germ cell tumours in stages A, B_1 (metastases to retroperitoneal nodes confined to four or fewer nodes, none of which is larger than 2 cm) and B_2 (metastases to retroperitoneal nodes involving more than four nodes or any node larger than 2 cm) should undergo retroperitoneal lymphadenectomy. Patients with stage B_3 disease (bulky, probably nonresectable retroperitoneal disease without pulmonary metastases) should be treated initially with intensive multidrug cyclic chemotherapy. If there is incomplete resolution of retroperitoneal tumour, these patients should be explored and the mass or masses resected.[21] About one third of these patients will have fibrosis only, one third will have mature teratoma and the remainder will have viable residual tumour.

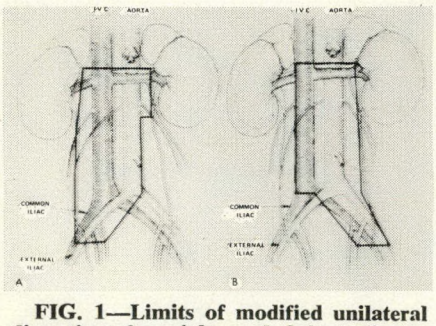

FIG. 1—Limits of modified unilateral dissection for right and left testicular tumours.

Only a small number of patients with seminoma are candidates for retroperitoneal lymphadenectomy. Patients with apparently pure seminoma in the primary specimen but who have an elevated serum α-fetoprotein level are considered to have nonseminomatous germ cell tumours and should be treated as such.[22]

Patients with clinical stage B seminoma who have incomplete resolution or progression of metastases following standard curative doses of radiotherapy should probably undergo retroperitoneal exploration if there is no evidence of pulmonary metastasis. This may be a relative indication and the role of chemotherapy in such cases is becoming more prominent as the next step in management. Many centres[21,23-25] now treat bulky stage B seminoma initially with multidrug cyclic chemotherapy, because the prognosis when such patients are treated initially with radiotherapy appears to be poor, there being a high rate of pulmonary metastases subsequently. Patients with anaplastic seminoma frequently present with more advanced disease than those with "classic" seminoma. While some[26,27] believe that, stage for stage, patients with anaplastic seminoma fare as well as those with the classic type, and respond similarly to radiotherapy, others are of the opinion that anaplastic seminomas behave more like nonseminomatous germ cell tumours and should be managed differently from patients with classic seminoma.[28-31] The significance of elevated β-human chorionic gonadotropin levels in patients with pure seminoma is uncertain. Any patient with seminoma, of classic or anaplastic types, who persistently has an elevated β-human chorionic gonadotropin level after standard radiotherapy of the retroperitoneum is a reasonable candidate for retroperitoneal lymphadenectomy. Finally, in the rare case of sequential bilateral seminomas where removal of the first tumour has been followed by radiotherapy to the retroperitoneum, retroperitoneal lymphadenectomy may be indicated after removal of the second tumour, if it is thought that the patient may not tolerate further curative doses of radiotherapy.

Technical Aspects

In view of the pattern of primary lymph-node drainage from the left and right testes, and because the error in intraoperative clinical impression of the status of the nodes is less than 10%, we believe that a modified unilateral lymphadenectomy should be performed (Fig. 1) if the lymph nodes are judged to be free of metastatic disease at the time of exploration. If, however, metastatic nodes are discovered, we recommend a more extensive bilateral dissection. Extended suprahilar dissection requiring thoracoabdominal exposure is not necessary or desirable, owing to the associated increase in morbidity. No improvement in survival has been demonstrated for patients who have undergone such a dissection. Using a high midline transabdominal approach, we accept as the upper limit of the dissection the inferior margins of the adrenals (i.e., about 2 cm above the renal arteries). Exposure for this part of the dissection is not especially difficult. The inferior mesenteric vein is divided with the ligament of Treitz and the posterior peritoneum is incised along the root of the small bowel mesentery, carried around the cecum and up the right paracolic gutter to the porta hepatis, thus allowing complete evisceration of the small bowel to expose the full field of dissection. The dissection entails complete removal of all lymph-node-bearing, fatty areolar tissue surrounding the great vessels within the boundaries of the dissection (Fig. 1). In a right-sided dissection, because of the known possibility of right to left lymphatic crossover, a left hilar dissection is also routinely performed down to the level of the inferior mesenteric artery. An attempt is made to preserve one or two lumbar arteries on each side as well as the sympathetic trunks bilaterally, although the sympathetic fibres are unavoidably removed with the lymphatic tissue. The great vessels are stripped to the subadventitial plane (Fig. 2), and the psoas muscles and anterior longitudinal spinal ligament are bared. This meticulous dissection is time-consuming, but the liberal use of hemostatic clips is most useful in reducing the operating time. All lymphatic channels must be either clipped or ligated to prevent formation of postoperative lymphocele. An interiliac dissection, baring the promontory of the sacrum below the confluence of the common iliac veins, is not routinely performed if the modified unilateral dissection is chosen. Much of the presacral autonomic plexus will thereby be preserved and perhaps also ejaculation.

This procedure is well tolerated by young patients and the complication rate is low. The most common post-

operative problem is prolonged paralytic ileus, but this almost always resolves within 7 to 9 days. Careful attention must be paid to postoperative fluid balance, as there is considerable third space loss. Vigorous postoperative physiotherapy and early mobilization probably reduce the incidence of pulmonary and thromboembolic complications.

Experience with Testicular Tumours at McGill University

In the 10-year period 1970 to 1980 the McGill testis tumour registry recorded 131 testicular tumours (47 seminomas and 84 nonseminomas). Although not all seminomas seen in the McGill-associated hospitals have been registered, all nonseminomatous germ cell tumours have been, and the follow-up of the patients has been complete. Seventy-two patients have undergone retroperitoneal lymphadenectomy; 2 had anaplastic seminoma (1 with an elevated β-human chorionic gonadotropin level) and 70 had primary nonseminomatous germ cell tumours. Another 10 patients have been registered with extragonadal germ cell tumours. Table IV summarizes the status, 2 years after operation, of the 72 patients who underwent retroperitoneal lymphadenectomy. Until late 1976, all patients who underwent retroperitoneal lymphadenectomy received adjuvant (or prophylactic) cyclic chemotherapy either with actinomycin D alone or according to the Mendelson regimen[32] (5-fluorouracil, cyclophosphamide, methotrexate, and vincristine). Since then, patients in stages A and B_1 have not received adjuvant chemotherapy but have been followed by monthly estimation of the α-fetoprotein and β-human chorionic gonadotropin levels and chest roentgenography for the first year, then these studies every 2 months for the subsequent year.

FIG. 2—Completed modified left-sided dissection. Anomalous left renal vein passes behind aorta. Right renal artery marks upper limit of interaortocaval dissection.

Pulmonary metastases developed in two stage A patients not receiving adjuvant chemotherapy. One is in complete remission following 18 months of VAB-IV* chemotherapy[2] and the other died following an incomplete response to VAB-IV.

Of the stage B_1 patients, all but the three most recent patients received adjuvant chemotherapy. Of those who did not, pulmonary metastases developed within 4 months in one. This patient is in complete remission on the VAB-IV regimen. One patient with stage B_1 tumour died. He had microscopic involvement of two hilar lymph nodes. Pulmonary metastases developed within 4 months of the lymphadenectomy even though he was receiving the adjuvant Mendelson regimen. He had a complete remission with the VAB-IV regimen but then relapsed and died 19 months after resection.

In the stage B_2 group, the results were less impressive. Several of the nine patients who died did not comply with their chemotherapy schedules and several were treated before the VAB protocols were introduced. Two others simply did not respond even to the most recent chemotherapeutic regimens. In at least three of these cases, there was gross spillage of necrotic liquified tumour at the time of lymphadenectomy.

Five of 10 patients with stage C tumour are long-term survivors following retroperitoneal lymphadenectomy and cyclic chemotherapy. Two had thoracotomy for residual pulmonary lesions following chemotherapy. Twelve other patients with stage C nonseminomatous germ cell tumours were not subjected to retroperitoneal lymphadenectomy. Most had an accelerated downhill course despite chemotherapy, but one is in complete remission. We anticipate a higher complete response rate with the chemotherapy currently in use.[1,2] Our results compare favourably with those obtained at other centres.

*The VAB-IV regimen was the fourth in an evolving series of multidrug cyclic chemotherapeutic protocols, consisting of bleomycin, vinblastine and high-dose cis-platinum, and interval maintenance therapy, also with multiple agents, given for a total of 24 months. A further modification (VAB-VI) using the same drugs in a more condensed schedule, is our current protocol.

Table IV—Status 2 Years after Operation of 72 Patients Who Underwent Retroperitoneal Lymphadenectomy

Tumour stage	Alive, no. (NED,* %)	Dead, no.
A	30 (97)	1
B_1	11 (91)	1
B_2	10 (52)	9
C	5 (50)	5

*No evidence of disease.

Discussion

There can be no doubt that current, combined, cyclic chemotherapy using high doses of vinblastine, bleomycin and cis-platinum has greatly improved the cure rates in nonseminomatous germ cell testicular tumours. Mainly because modern chemotherapy is so effective in advanced disease, and because full doses are required for best results, most North American oncologists are now convinced that radiotherapy should be avoided in the primary treatment of these tumours. Permanent bone-marrow suppression frequently precludes the use of full dose schedules of vinblastine in patients who subsequently require chemotherapy. Radiation to the mediastinum with scatter to the lungs may limit the total dose of bleomycin that can be tolerated in view of the known risk of pulmonary fibrosis when this drug is used. Certainly it has been the experience of medical oncologists that the use of intensive chemotherapy is considerably more difficult in patients who have previously received radiotherapy.[1,23]

Will the surgeon's role change in the future? Certainly the high incidence of infertility due to aspermia following retroperitoneal lymphadenectomy is distressing, particularly in young men who have not yet started their families. It would be desirable to avoid retroperitoneal lymphadenectomy in these patients

provided their survival was not compromised. Nevertheless, subsequent chemotherapy, should it be indicated, will probably have permanent effects on spermatogenesis. Also, there is a fairly high rate of impaired spermatogenesis in patients who harbour testicular tumours.[33] Thus, the importance of loss of ejaculation may have been overemphasized in the arguments against the use of retroperitoneal lymphadenectomy for nonseminomatous germ cell tumours.

Projected improvements in the sensitivity of biochemical markers and other nonsurgical techniques may improve the accuracy of clinical staging to the point where extensive retroperitoneal lymphadenectomy will no longer be necessary for reliable staging; a more limited sampling of the primary lymph nodes may become accepted as adequate and be associated with a lower rate of aspermia. Even though clinical staging is imperfect, it may be that a very closely monitored "wait and see" policy following orchiectomy will spare an appreciable number of these patients extensive (and therapeutically unnecessary) operations. Provided early detection of metastatic disease is assured, these patients may do as well with delayed chemotherapy as those whose management is based upon surgical staging. Nevertheless, we believe that, at present, complete, meticulous lymphadenectomy is the single most important factor in providing definitive therapy and a guideline for the adjuvant management of patients with nonseminomatous germ cell testicular tumours.

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Control of Postoperative Renal Hemorrhage by Embolization with a Gianturco Coil

G. JOHN ANKENMAN, MD, FRCS[C]* AND JOHN B. MURRAY, MB, FRCR, FRCP[C]†

A 35-year-old woman underwent a left pyelonephrolithotomy for removal of a staghorn calculus. Blood loss was minimal for 5 days. Then there was profuse bleeding from the left kidney that did not respond to conservative management. Angiographic examination demonstrated extravasation of contrast medium from a minor intrarenal artery in the lower pole of the kidney directly into the pelvicalyceal system. An attempt was made to embolize the bleeding vessel with Gelfoam particles but this failed because the Gelfoam passed directly into the collecting system. A Gianturco coil, however, occluded the vessel and immediately the hemorrhage ceased; the patient had no further complications.

The authors emphasize that the introduction of a steel coil to control bleeding is advocated only when the use of temporary occlusive agents is unsuccessful.

Une femme de 35 ans a subi une pyélonéphrolithotomie gauche pour l'ablation d'un calcul dendritique. On observa pendant 5 jours un épanchement sanguin minime. Puis, il y eut alors une hémorrhagie abondante provenant du rein gauche qui ne répondit pas à un traitement conservateur. L'examen angiographique révéla une extravasation de la substance de contraste à partir d'une petite artère intrarénale située au pôle inférieur du rein, directement dans le système pelvicaliciel. On tenta d'oblitérer le vaisseau hémorrhagique à l'aide de particules de Gelfoam, mais sans succès puisque le Gelfoam fuit directement dans le système collecteur. Une spirale Gianturco permit toutefois d'oblitérer le vaisseau et d'arrêter immédiatement l'hémorrhagie; la patiente n'eut pas d'autre complication.

Les auteurs insistent sur le fait que l'emploi d'une spirale d'acier pour maîtriser un saignement n'est recommandé que lorsque les agents d'oblitération temporaire ont échoué.

Pyelolithotomy for removal of a staghorn calculus was performed successfully on a patient who presented with recurrent urinary tract infections. But 5 days later persistent renal bleeding commenced. The patient did not respond to conservative management and transcatheter segmental embolization with Gelfoam was also unsuccessful. However, when a steel (Gianturco) coil (Cook Bloomington Inc., Markham, Ont.) was introduced, the bleeding ceased immediately.

Case Report

A 35-year-old female nurse underwent investigation for recurrent urinary tract infection. The right upper tract was radiologically normal. On the left there was a large staghorn calculus (Fig. 1). A pyelonephrolithotomy was performed. The kidney was cooled by the slush technique and the renal artery clamped during operation. The calculus was removed by an extended pyelolithotomy and smaller nephrotomy incision. Subsequent roentgenograms confirmed complete removal of the calculus. The 1000 ml of blood lost during the procedure was replaced. At closure, bleeding from the kidney was minimal, so a fenestrated no. 10 feeding tube was inserted into the ureter and brought out through the renal parenchyma. A nephrostomy was not done.

The patient had no appreciable blood loss for 5 days. Then the urine from an indwelling Foley catheter in the bladder became sanguineous. Blood replacement was not required until 10 days after operation. Then, during the next few days, she received 8 units of whole blood, 2 units of packed cells and 6 units of fresh frozen plasma because of progressive, profuse bleeding through the Foley catheter.

When it was apparent that she would not respond to conservative management, angiographic examination was carried out to identify the precise site of bleeding in the hope that selective transcatheter embolization would stop the bleeding.

Renografin-76 (12 ml) was injected through a no. 6.5 F renal Gold catheter into the main renal artery; the bleeding site was demonstrated by focal accumulation of extravasated contrast medium

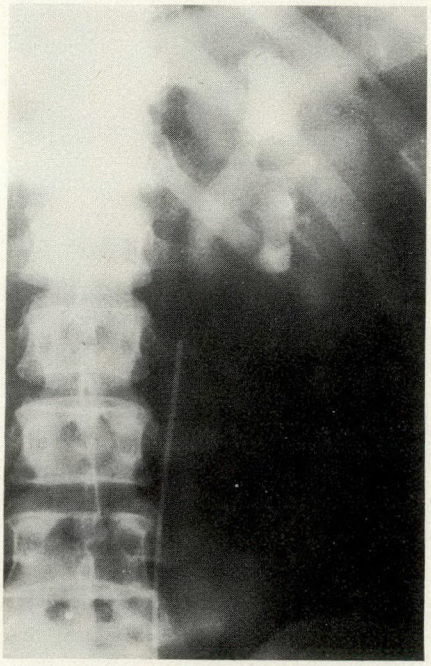

FIG. 1—Plain film obtained at time of retrograde pyelography showing opaque staghorn calculus.

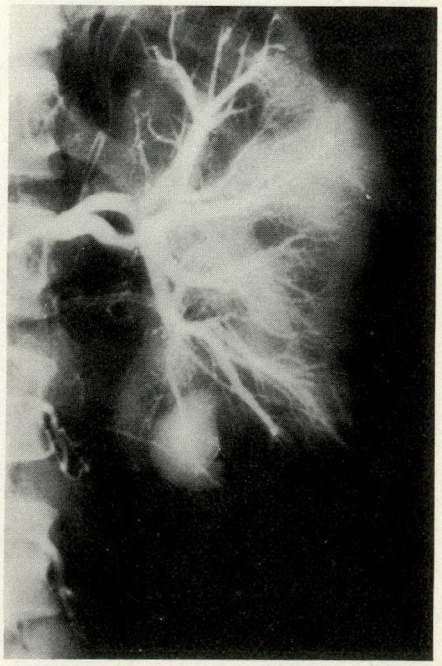

FIG. 2—Selective renal arteriogram. There is extravasated contrast medium in lower pole.

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in the lower pole (Fig. 2). The catheter was exchanged for a no. 6.5 F cobra catheter which was easily passed into the ventral branch of the main renal artery close to the site of bleeding; 4 ml of the contrast medium was injected and the pool of extravasated contrast medium was shown to communicate directly with the pelvicalyceal system, which contained blood clot (Fig. 3). With the catheter tip unaltered in its position, multiple fragments of gelatin sponge were injected in 2-ml increments of Gelfoam-saline and Gelfoam-contrast medium mixtures. Hemostasis was not obtained because the fragments passed through the vessel into the collecting system.

A 5-mm wide steel (Gianturco) coil was then introduced (Fig. 4), the catheter tip withdrawn to the origin of the ventral branch and 8 ml of contrast medium injected; complete occlusion of the interlobar artery was achieved (Fig. 5).

There was no further bleeding. The patient's postoperative course thereafter was smooth. Excretion urography done 3 months after operation showed contraction of the lower renal pole consistent with infarction but with satisfactory function of the upper pole.

Discussion

The feasibility of transcatheter renal infarction was initially evaluated experimentally in animals by Lalli and colleagues in 1969.[1] Since then, embolization of renal arteries has been employed in a variety of clinical situations,[2-5] most notably as an adjunct to nephrectomy for renal cell carcinoma, but also to control hypertension caused by segmental renal artery stenosis,[6] to occlude intrarenal aneurysms[7] and spontaneous fistulas associated with renal arterial disease, and in congenital malformations.[8] This method of control of post-traumatic renal hemorrhage is gaining acceptance with the increasing successes of "interventional" radiology.[9]

A variety of embolic materials have been used in humans. Autologous blood clot and gelatin sponge (Gelfoam) have proven the most popular temporary occlusive agents and with vascular hypernephromas are frequently used in combination with Gianturco steel coils for total obliteration of the main renal arterial supply.

In our case the catheter was advanced as far as possible into the inferior pole vessel of the ventral branch of the main renal artery in an attempt subselectively to embolize the bleeding vessel. Gelfoam particles of increasing size were used initially, to provide temporary occlusion, but proved unsuccessful; the particles passed freely into the adjacent calyx, which was in direct communication with the bleeding vessel. The Gianturco coil was then placed as close as possible to the bleeding site. The major objective of avoiding a nephrectomy overshadowed the risk of segmental infarction and hypertension. Immediate hemostasis was obtained. As shown on the subsequent excretion urogram, infarction of the lower pole occurred, but satisfactory excretion of contrast medium occurred from the upper pole and the patient is now normotensive 18 months later.

The best embolic agent would have been Gelfoam, which produces a clot that will last more than 24 hours, whereas autologous clot lyses more rapidly. A search of the literature did not reveal a similar case in which the use of Gelfoam particles was unsuccessful because they passed directly into the renal collecting system. The introduction of a 5-mm wide steel coil was our alternative at the time because the 3-mm coil was not commercially available. Our success in this case may be unprecedented, but we do not advocate the use of such a coil unless temporary occlusive agents are unsuccessful. Reoperation and increased morbidity were avoided, the iatrogenic infarction being the only apparent sequela.

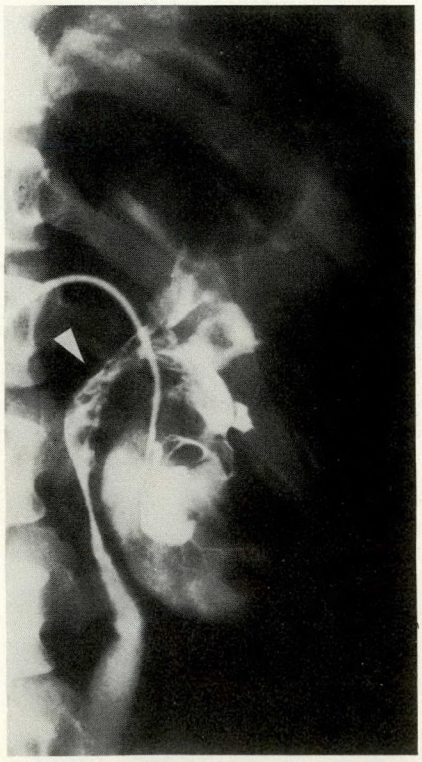

FIG. 3—Subselective arteriogram showing collecting system and proximal ureter opacified by extravasated contrast medium containing blood clot (arrow).

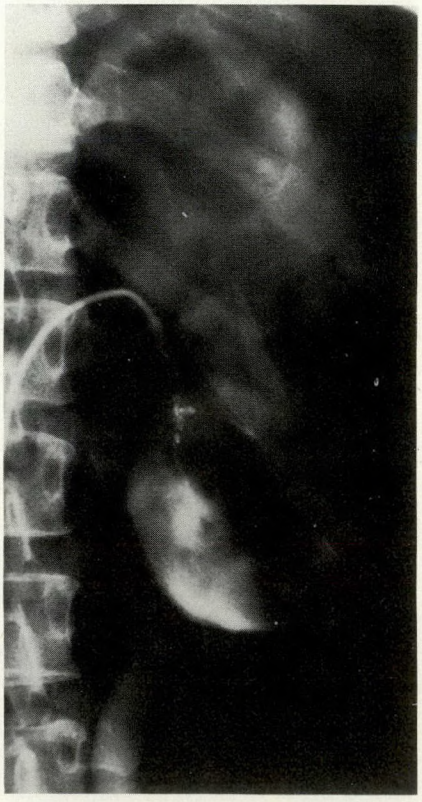

FIG. 4—Gianturco coil in place. Catheter withdrawn to more proximal position in ventral branch.

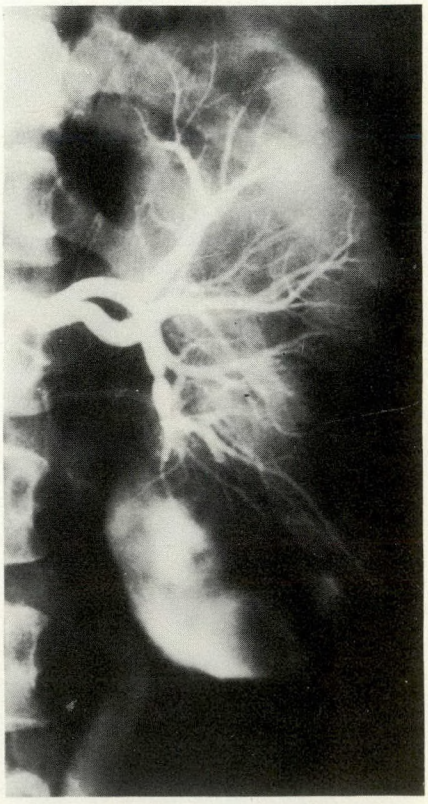

FIG. 5—Postcoil embolization; selective arteriogram shows occlusion of lower pole branch of ventral artery.

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Renal Angiomyolipoma Not Associated with Tuberous Sclerosis

J.L. CHIN, MD AND J.K. WYATT, MD, FRCS[C], FACS

Six cases of renal angiomyolipoma not associated with the tuberous sclerosis complex illustrate the common presenting clinical picture of this relatively rare hamartoma. The authors discuss the traditional and newer diagnostic studies used, particularly ultrasonography and computerized axial tomography and their applications in the diagnosis of angiomyolipoma. Special emphasis is placed on differentiation of renal angiomyolipoma from renal cell carcinoma. Although the newer imaging techniques are valuable diagnostic aids, treatment of these renal mass lesions should not be based entirely on results of such studies.

Six cas d'angiomyolipome rénal non reliés à une sclérose tubéreuse complexe servent à illustrer le tableau clinique habituel de cet hamartome relativement rare. Les auteurs commentent les techniques diagnostiques traditionnelles et plus récentes utilisées, particulièrement l'échographie ultrasonique et la tomographie transverse axiale avec ordinateur, et leurs applications au diagnostic de l'angiomyolipome. On insiste spécialement sur la différenciation entre l'angiomyolipome rénal et le carcinome à cellules rénales. Bien que les nouvelles techniques de visualisation soient des moyens diagnostiques utiles, le traitement de ces masses rénales ne doit pas reposer entièrement sur les résultats de telles études.

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Renal angiomyolipoma is a benign hamartoma of mixed mesenchymal origin. About 250 cases have been reported. Although its association with the tuberous sclerosis complex is well known, about half the reported cases are not associated with tuberous sclerosis. Patients without signs of tuberous sclerosis present a diagnostic challenge in the differentiation of renal angiomyolipoma from renal cell carcinoma and a therapeutic challenge in preserving renal tissue.

Clinically, tumours not associated with tuberous sclerosis are often unilateral, solitary and large. There is a female predominance[1] and the peak age range is the fifth to seventh decade.[2] The tumour involved the right kidney in 80% of reported cases.[3] In contrast, patients with the tuberous sclerosis type of tumour are usually juveniles and there is no sex predominance. The disease is usually bilateral with multiple small tumours.

Patients

Over the past 10 years, six patients (four women, two men) with no signs of tuberous sclerosis presented at our centre with unilateral renal angiomyolipoma. Their ages ranged from 39 to 66 years (three were in their 50s). All had left-sided tumours. As in other reports, the commonest presenting symptoms were severe flank pain (five patients), hematuria (five) and a palpable mass (two patients). Two patients presented with the rarer pictures of retroperitoneal hemorrhage and hypertension, respectively. All six patients had a space-occupying lesion demonstrated on an excretory pyelogram. Renal angiography done in five patients showed abnormal tumour vessels suggestive of renal cell carcinoma. The sixth patient presented with hypovolemic shock from retroperitoneal hemorrhage and was operated upon without undergoing angiography. Renal ultrasonography showed a characteristic solid lesion in two patients. Radical nephrectomy was performed in five patients because of the preoperative diagnosis of renal cell carcinoma. One patient with compromised renal function had a partial nephrectomy to preserve renal tissue.

Diagnosis

The diagnosis of renal angiomyolipoma is mainly radiologic. Renal mass lesions are usually discovered by excretory pyelography and, if suspected from clinical presentation, should be confirmed by infusion pyelography with tomography. Grey-scale ultrasonography differentiates true cystic lesions from solid and partly solid mass lesions. The simple cyst can be accurately diagnosed by ultrasonography and further investigation or treatment is rarely required. For all other renal masses renal angiography more accurately outlines the vascularity of the mass and aids in planning the surgical procedures that may be necessary. Angiographic examination does not reliably differentiate between the neovascularity of angiomyolipoma and the hypovascularity of renal cell carcinoma with areas of central necrosis and tumour infarction. The earlier attempts to differentiate angiomyolipoma from renal cell carcinoma were based on sup-

posedly characteristic angiographic features of angiomyolipoma, such as saccular aneurysmal dilatation of interlobar and interlobular arteries (Fig. 1), the "onion-skin" whorled pattern in the capillary phase and the absence of arteriovenous shunting. However, the current consensus is that renal angiography cannot be relied upon to differentiate angiomyolipoma from renal cell carcinoma, especially stage A carcinoma.

Renal Ultrasonography

Recent developments in ultrasonography help in establishing a more accurate preoperative diagnosis. Normal renal parenchyma is echo-free at both low and high gain settings. Renal cell carcinoma is usually echo-free at low gain, but will show some internal echoes at high settings, although there are some highly echogenic examples. Angiomyolipoma shows clusters of internal echoes at both low and high gain settings,[4] demonstrating the disorganization and the numerous interphases among blood vessels, muscles, fat and connective tissues. Fig. 2 shows the typical ultrasonographic picture of angiomyolipoma. We stress, however, that the diagnostic accuracy of ultrasonography is not such that the need for a radical cancer surgery approach can be dismissed, because we have encountered renal cell carcinomas that were highly echogenic at both low and high gain settings.

Computerized Axial Tomography

We have used computerized axial tomography in several cases of renal cell carcinoma mainly for staging, and there are recent reports[5,6] of computerized axial tomography used as an additional diagnostic tool for angiomyolipoma. Fat-dominant angiomyolipomas are clearly seen on the tomogram (Fig. 3). These tumours have very low attenuation coefficients (–30) compared with other tissues because of the low density of fat.[5,6] Renal cell carcinoma does not contain fat and therefore has a much higher attenuation coefficient (>0). Tumours with necrosis or hemorrhage have intermediate attenuation values (–10), usually quite easily distinguishable from angiomyolipoma.[5,6] However, not all angiomyolipomas contain fat in the quantities that would display the characteristic low density pattern on the computerized axial tomogram.

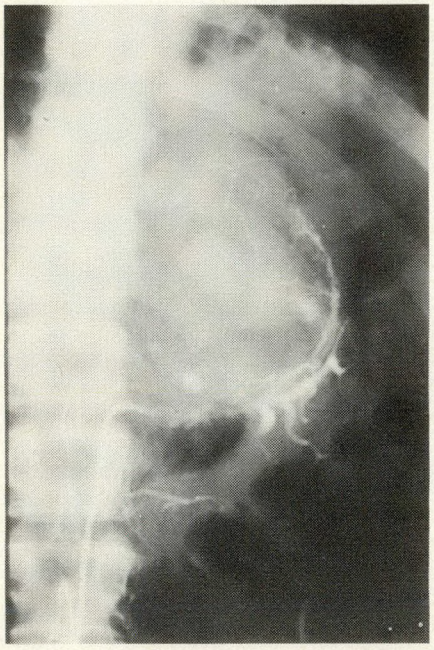

FIG. 1—Renal angiogram of angiomyolipoma showing typical interlobar and interlobular artery aneurysms.

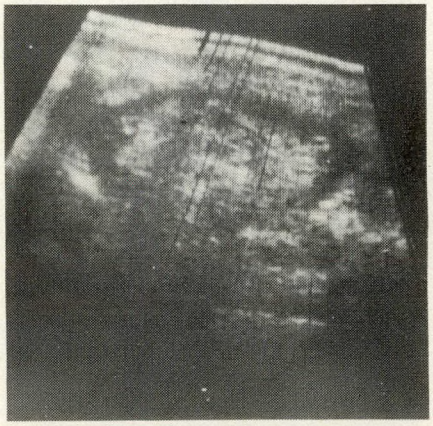

FIG. 2—Ultrasonogram showing kidney (as outlined by relatively sonolucent perirenal fat) with dense internal echoes reflecting disorganization and numerous interphases among blood vessels, muscles, fat and connective tissues that constitute tumour.

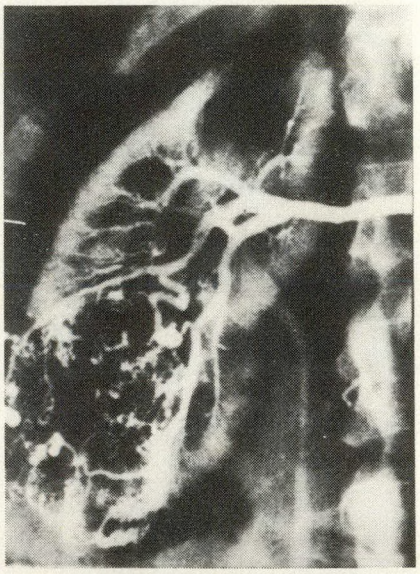

FIG. 3—Fat-dominant angiomyolipoma. Renal angiogram (top) and computerized tomogram (bottom) showing renal mass (arrow).

Discussion

The first aim of treatment is to distinguish angiomyolipoma from a malignant tumour preoperatively. The usual work-up for renal cell carcinoma should be performed. Ultrasonography and computerized axial tomography, if available, should be used as adjunctive tools. As emphasized earlier, these newer investigations cannot be depended upon completely to rule out a malignant lesion. We must therefore bear in mind when dealing with certain solid and vascular renal mass lesions that the tumour may be benign. The second aim in treatment is to preserve normal renal tissue. However, since we have not absolutely resolved the diagnostic dilemma in patients with angiomyolipoma but no signs of tuberous sclerosis, we cannot afford to break with the radical cancer surgery approach to justify preservation of renal tissue except in special circumstances.

Fig. 3 is reproduced by courtesy of John Wiley & Sons, Inc., New York, from Lee TG et al: Ultrasound findings of renal angiomyolipoma. *JCU* 1978; 6: 150–5

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Unilateral Renal Artery Embolism Managed Conservatively

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Renal artery embolism presents a diagnostic challenge because the condition is rare and it often mimics other more common abdominal problems. A conservative approach is favoured by most urologists but management is still controversial.

The authors present a case of unilateral renal artery embolism that illustrates the difficulties and delay in making a diagnosis. It also highlights the efficacy of anticoagulants in restoring adequate function in a kidney that had been totally nonfunctioning and partially ischemic for longer than 18 hours.

Le diagnostic de l'embolie d'une artère rénale présente un défi, du fait que cette affection est rare et qu'elle prend souvent les apparences d'autres problèmes abdominaux plus fréquents. La plupart des urologistes favorisent une approche conservatrice mais le traitement est toujours sujet à controverse.

Les auteurs décrivent un cas d'embolie unilatérale d'une artère rénale qui illustre les difficultés de diagnostic et les délais à y arriver. Il met également en lumière l'efficacité des anticoagulants à rétablir une fonction adéquate dans un rein qui avait été complètement non fonctionnel et partiellement ischémique pendant plus de 18 heures.

Renal artery embolism is uncommon. Patients with the lesion are usually seen in the emergency department and have flank and abdominal pain that often mimics other, more common, abdominal conditions.

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Whether the embolus should be removed surgically[1] or treated conservatively with systemic anticoagulants[2] is controversial. In a series of 24 cases of unilateral renal artery embolism, there were no deaths in patients managed nonsurgically but 18% of patients who were operated on died. The rate of kidney preservation in the two groups was 77% and 44% respectively.[3] Intra-arterial infusion of streptokinase to dissolve the embolus has also been recommended as an adjunct to systemic anticoagulant therapy.[4]

We recently treated a patient with left renal artery embolism who illustrates the problems associated with this uncommon condition.

Case Report

A 71-year-old woman was seen in the emergency department with a 6-hour history of left flank and lumbar pain of acute onset. The pain was constant and radiated to the umbilical region and left groin. Her medical history was important in that she was taking insulin for diabetes and medication for hypertension, and she had ischemic heart disease with mitral stenosis. Her blood pressure was 160/110 mm Hg, the pulse rate was 88 beats/min and was irregularly irregular, and the temperature was 37.5°C. The abdomen was not distended, but was tender throughout, particularly in the left costovertebral angle and left lumbar region. Peripheral pulses were present and no bruit was heard over the abdomen.

The hemoglobin level was 13.4 g/dl, leukocyte count 10×10^9/l; urinalysis showed 2 to 3 erythrocytes per high-power field, trace protein and 1 to 3 leukocytes per high-power field. The blood sugar level was 263 mg/dl (14.6 mmol/l), blood urea nitrogen 14 mg/dl (5 urea, mmol/l), serum creatinine 0.8 mg/dl (70.7 μmol/l), and serum electrolyte and amylase levels were normal. The electrocardiogram revealed atrial fibrillation with a controlled ventricular response.

Intravenous pyelography on admission revealed a normally functioning right and a nonfunctioning left kidney in the early and delayed films. Both kidneys were of normal size. Cystoscopy under local anesthesia revealed a normal bladder and a left retrograde pyelogram appeared normal. There was no urine output from the left kidney. A renal arteriogram showed that the renal vasculature was normal on the right; however, the left renal artery was completely occluded approximately 2 cm from its origin (Fig. 1). A selective left renal arteriogram revealed a few collateral vessels and a lucency at the site of obstruction (Fig. 2).

Heparin was given intravenously (1000 U/h), and digoxin (0.125 mg/*q6h*, intravenously, followed by 0.125 mg, once a day, orally) was administered to control her atrial fibrillation. Warfarin therapy (5 mg once a day, then 2.5 mg once a day) was started the next day and the heparin was discontinued after 5 days. The patient did well and at the time of

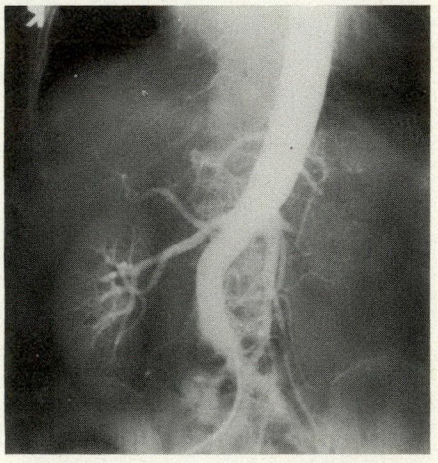

FIG. 1—Flush arteriogram shows normal right renal vasculature and complete occlusion of left renal artery.

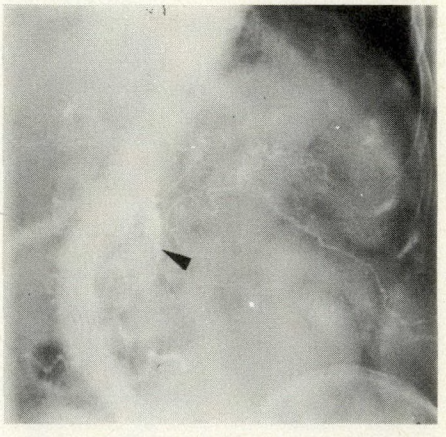

FIG. 2—Selective left renal arteriogram. There are several collateral vessels and lucency (embolus) (arrow) occluding left renal artery.

discharge, 3 weeks later, the left kidney was functioning well (Fig. 3).

Discussion

Since the first case of renal artery embolism was reported by Traube in 1856, a number of reports on this condition have appeared in the literature.[2] Some of the important etiologic factors in the development of renal artery embolism are: atrial fibrillation, myocardial infarction, subacute bacterial endocarditis, surgery on the abdominal aorta, dislodgement of plaques during angiographic procedures and blunt trauma.

The symptoms usually consist of acute unremitting pain localized to the lumbar region or spreading diffusely into the upper abdomen, sometimes associated with nausea or ileus. Invariably proteinuria and microscopic hematuria are present. The serum lactic dehydrogenase concentration is usually elevated and leukocytosis may be present. The intravenous pyelogram shows a kidney of normal size that is nonfunctional. Our patient had similar clinical findings and the characteristic pyelographic features, which made us highly suspicious of renal artery embolism. The lesion was confirmed by renal arteriography.

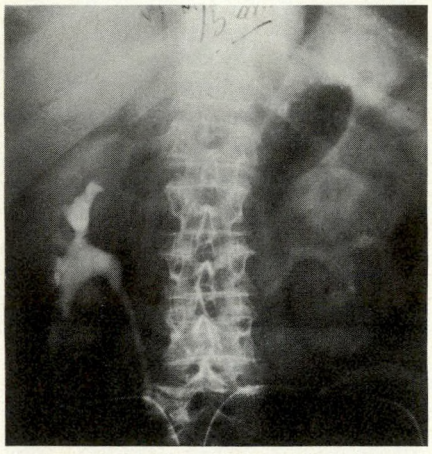

FIG. 3—Intravenous pyelogram after 3 weeks (15 minute film). There is good function in left kidney.

Recent reports suggest that the treatment of choice for unilateral renal artery embolism is the administration of systemic anticoagulants possibly with percutaneous transfemoral intra-arterial infusion of streptokinase for quick dissolution of the embolus.[5] Anticoagulant therapy prevents further propagation of clot and occlusion of small cortical collateral vessels by platelet microthrombi.[3] Other supportive measures are aimed at controlling the atrial fibrillation and correcting the dehydration, which are often present. Urinary infection is treated vigorously to prevent sepsis in the ischemic kidney. Occasionally hemodialysis is required for patients with acute renal failure due to bilateral renal artery embolization or to an embolus in a solitary kidney.

Although 18 hours had passed since the first attack of pain and the left kidney was completely nonfunctional in our patient, she responded well to systemically administered anticoagulants; function returned in that kidney within 3 weeks. Perfusion of the renal parenchyma via capsular and peripelvic collaterals has been noted to occur immediately after renal artery occlusion;[6] there is also minimal blood flow around the embolus. These two factors are important for preserving renal function in the presence of total renal artery occlusion as seen on angiography. This may also explain the success of conservative management using anticoagulants. Adjuvant intra-arterial streptokinase was not used in our patient because we were afraid of dislodging atheromatous plaques.

It is essential to be aware of this uncommon problem so that diagnosis is made quickly and correct treatment is instituted before irreparable damage occurs.

We thank Mrs. Cathy Breen for her secretarial assistance.

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BOOK REVIEWS

ATLAS DE RADIOLOGIE DU PIED. J. Montagne, A. Chevrot et J.-M. Galmiche. 277 pp. Illust. Masson Paris, 1980. Prix non mentionné. ISBN 2-225-48708-1.

This atlas on the foot, which has been written in French and translated into English, contains good-quality photographs and has an excellent bibliography. Each radiograph is succinctly described. The authors consider first the radiologic techniques and anatomy of the adult and pediatric foot. Normal variants and multiple radiologic measurements are detailed. The normal and abnormal biomechanics of the foot are also covered.

The chapters are divided according to radiologic abnormalities, so that the reader who is dealing with an unknown picture can find the answer rapidly. Each radiologic appearance is accompanied by a description of the different diseases that can produce this picture. Multiple topics are covered such as local or diffuse increased density, periostitis, localized or generalized osteopenia, expansile lesions, severe loss of bone, osteonecrosis, arthritides and soft-tissue abnormalities.

This is a well-organized and didactic reference book which will be useful for rheumatologists, orthopedic surgeons and radiologists.

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CONGENITAL DEFORMITIES OF THE HAND. An Atlas of Their Surgical Treatment. W. Blauth and F. Schneider-Sickert. 387 pp. Illust. Springer-Verlag New York Inc., New York, 1981. $230. ISBN 0-387-10084-9.

This impressive volume represents the authors' experience in over 1000 operations for congenital hand deformity. It is written for all practitioners of hand surgery, who will find advice and answers on surgical technique and timing. It has been translated from the original German but, thanks to the able translation of Ulrich Weil, one does not detect this during reading.

continued on page 327

Intestinal Bypass and Gastric Partitioning for Morbid Obesity: a Comparison

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M.A. BOJM, MD, FRCS[C], M.D. ATIN, B SC† AND G.S. ZAKHARY, MD‡

The authors have compared the results of jejunoileal bypass and gastric partitioning for the treatment of morbid obesity in a consecutive series of 174 patients. All patients were more than 45 kg overweight. There was a high incidence of hypertension, diabetes, respiratory distress, hyperlipemia, debilitating arthritis and stress incontinence, which where relieved by weight loss after surgery Thoracic epidural anesthesia was used.

In 64 patients who underwent jejunoileal bypass, weight loss was 27% at 1 year and reached a plateau of 33% at 2 years (range from 18% to 53%). Liver biopsy specimens obtained at operation showed fat in the centrilobular area; postoperative biopsy specimens showed worsening of this condition at 9 months, improvement at 18 months and a normal appearance of the lobules at 30 months. The in-continuity section of the bowel showed villous hypertrophy with cellular hyperplasia and the excluded bowel showed mucosal atrophy. Sixteen patients required replacement of electrolytes, 6 had polyarthritis, which responded to metronidazole therapy, and 5 had renal stones, which resolved after the patient was put on a low oxalate diet and oral calcium was prescribed. Diarrhea after 1 year was not a problem.

Gastric partitioning was carried out on 110 patients, forming a pouch of 50-ml capacity. In the first 56 of these patients the 1.2-cm diameter outlet enlarged, reducing the effectiveness of the operation. Weight loss at 1 year was 21%, but at 30 months it was only 16% (range from 0% to 36%). In the next 54 patients, the 1.2-cm outlet was reinforced about a calibrating bougie by a circumferential imbricating nonexpanding suture. In this group weight loss was 31% at 1 year.

In neither series have there been any deaths. Jejunoileal bypass gives acceptable weight loss, but patients require continued surveillance. Gastric partitioning provides effective weight loss if enlargement of the outlet can be prevented.

Les auteurs ont comparé parmi une série de 174 patients consécutifs, les résultats obtenus par dérivation jéjuno-iléale et par cloisonnement gastrique dans le traitement de l'obésité pathologique. Tous les patients avaient plus de 45 kg en trop. On notait une forte incidence d'hypertension, de diabète, d'insuffisance respiratoire, d'hyperlipémie, d'arthrite débilitante et d'incontinence à l'effort, que la perte de poids après chirurgie a améliorés. On eut recours à l'anesthésie thoracique épidurale.

Chez 64 patients qui avaient subi une dérivation jéjuno-iléale, la perte de poids était de 27% après 1 an et elle atteignit 33% après 2 ans (écart de 18% à 53%). Les biopsies hépatiques pratiquées au moment de l'opération montraient du tissu graisseux dans la région centro-lobulaire; les biopsies postopératoires révélèrent une détérioration après 9 mois, une amélioration après 18 mois et des lobules normaux après 30 mois. La section anastomosée de l'intestin montrait une hypertrophie villeuse avec hyperplasie cellulaire alors que la partie de l'intestin mise au repos présentait une atrophie de la muqueuse. Seize patients ont nécessité une recharge électrolytique, 6 ont souffert d'une polyarthrite qui répondit au métronidazole, et 5 ont eu des calculs rénaux qui disparurent lorsque les patients reçurent un régime alimentaire à faible teneur en oxalate et qu'on leur administra du calcium par voie orale. Après 1 an, la diarrhée n'était plus un problème.

Un cloisonnement gastrique par formation d'une poche de 50 ml fut pratiqué chez 110 patients. Chez 56 de ces patients, l'orifice de 1.2 cm de diamètre s'élargit réduisant l'efficacité de l'opération. La perte de poids après 1 an était de 21%, mais elle n'était plus que de 16% après 30 mois (écart de 0% à 36%). Chez les 54 patients suivants, l'ouverture de 1.2 cm fut renforcie autour d'une bougie d'étalonnage par une suture circonférentielle, imbriquée et non expansible. Dans ce groupe, la perte de poids était de 31% après 1 an.

Aucun décès n'a été enregistré dans l'une ou l'autre des séries. La dérivation jéjuno-iléale assure une perte de poids acceptable mais les patients nécessitent une surveillance continue. Le cloisonnement gastrique entraîne efficacement une perte de poids en autant qu'on puisse prévenir l'élargissement de l'ouverture.

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Diets, drugs, psychotherapy, behavioral and exercise programs, injections, hypnotherapy, acupuncture and tooth-wiring have all been espoused for weight loss in morbidly obese individuals, but none has proven reliable over the long term. Surgical approaches have therefore evolved as a last resort in an attempt to provide effective, lasting weight loss (Fig. 1). We review the development of these procedures and compare our experience with jejunoileal bypass and gastric partitioning.

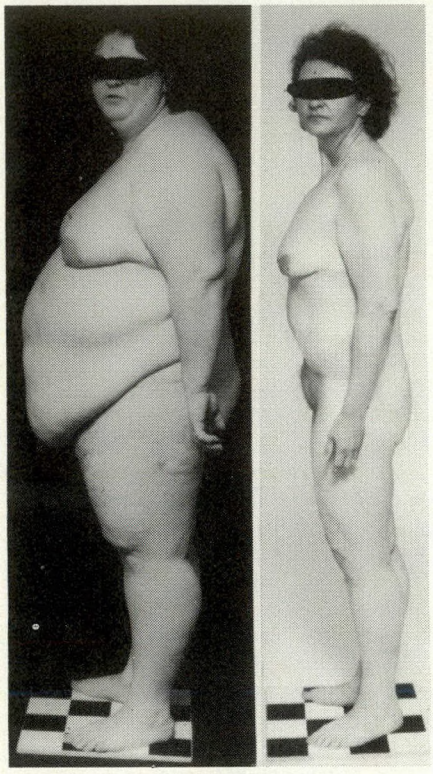

FIG. 1—Patient before and after surgery.

Historical Background

To allow food to bypass the major absorptive areas of the small bowel, Payne and colleagues[1] and Lewis and colleagues[2] experimented with the end-to-side jejunotransverse colostomy (Fig. 2), but the sequelae of electrolyte imbalance, occasional excess loss of weight and liver failure were too hazardous. Payne then popularized the end-to-side jejunoileal bypass[3-5] (Fig. 2); his initial measurements of 37.5 to 12.5 cm were associated with late weight gain in some patients, so were changed to 35 to 10 cm. These precise measurements from the ligament of Treitz along the mesenteric border of the bowel were found to allow the weight loss to be maintained after adaptation of the functioning small bowel. The jejunal stump was tacked to the root of the transverse mesocolon to prevent intussusception. Because some patients did not lose enough weight because of reflux occurring a short distance into the bypassed bowel,[6] other procedures were devised.

Scott and colleagues,[6-8] Salmon[9] and Buchwald and associates[10] developed the end-to-end jejunoileal bypass, draining the bypassed and isolated bowel into the transverse colon, sigmoid or cecum (Fig. 3), to discourage reflux into and absorption by the bypassed bowel. Palmer and Marliss[11] modified the Payne bypass by using a Y-shaped anastomosis (Fig. 3) to direct food to the cecum without reflux; furthermore, they simply tacked the jejunal stump to the adjacent ileum.

To avoid the undesirable sequelae of intestinal bypass, Mason and associates[12,13] pioneered the gastric bypass (Fig. 4), dividing the proximal 10% of stomach with a Billroth II-type retrocolic, short-loop, gastrojejunostomy, using a stoma 12 mm in diameter. The limited gastric functional capacity and narrow outlet necessitated decreased food intake. Alden[14] simplified this difficult procedure by a staple-line partitioning the stomach (which was not transected) and by an antecolic gastrojejunostomy to the proximal segment of 60-ml capacity. Griffen and colleagues[15,16] used a retrocolic Roux-en-Y jejunal loop anastomosed to a proximal 50-ml segment of divided stomach in order to avoid bile reflux, lessen the hazard from anastomotic leakage and facilitate moving jejunum in a retrocolic fashion to a high position (Fig. 5). Pace and coworkers[17,18] described the simpler technique of gastric partitioning (Fig. 6), removing three central staples before a single application of the TA90 stapler (United States Surgical Corp., Stamford, Conn.) leaving a 9-mm opening, but Cohn and associates,[19] using this method, had a mean weight loss of only 20% at 10 and 16 months.

Patients and Methods

Between Sept. 26, 1973 and Sept. 1, 1981 at the St. Joseph's Health

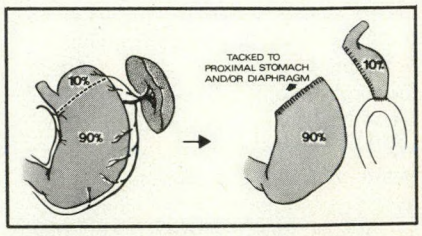

FIG. 4—Original gastric bypass of Mason.[12]

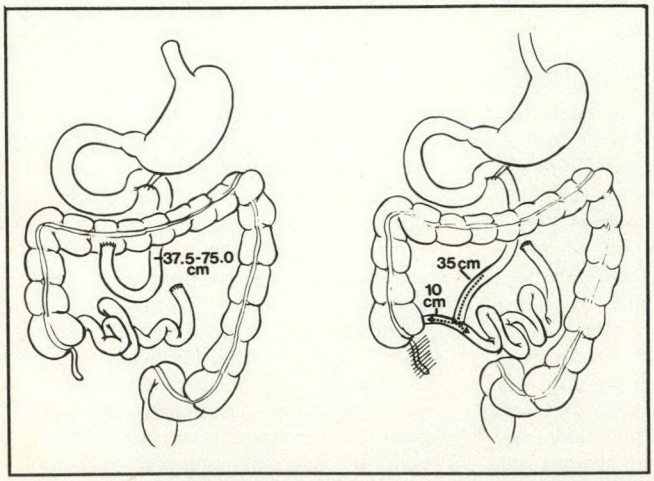

FIG. 2—Left: Jejunocolic bypass after Payne and colleagues[4] and Lewis and associates.[2] Right: End-to-side jejunoileostomy (T-shaped anastomosis) popularized by Payne.[3-5]

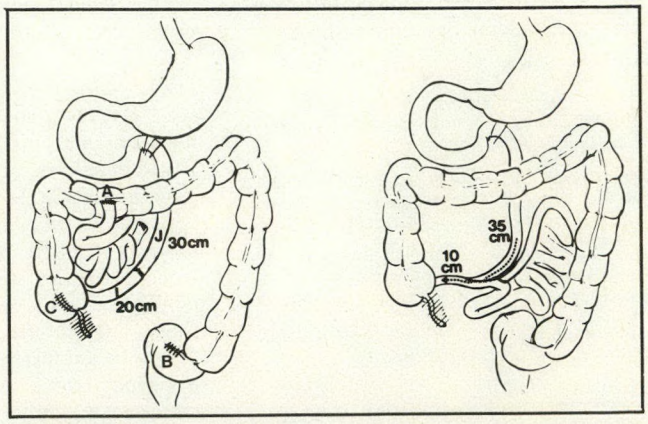

FIG. 3—Left: End-to-end jejunoileal bypass; Scott and associates[6-8] and Salmon[9] drained bypassed bowel into transverse (A) or sigmoid (B) colon; Buchwald and colleagues[10] chose cecum (C). Right: Modification of Payne bypass by Palmer and Marliss.[11] Appendix is removed in all jejunoileal bypasses.

Centre in Toronto, 174 consecutive patients underwent surgery for morbid obesity, according to established criteria (Table I). The first 64 patients underwent jejunoileal bypass. From 1973 to 1975 patients underwent the Scott jejunoileal procedure (Fig. 3, left). In the University of Toronto collaborative group, however, Palmer's patients had a similar weight loss, so from 1975 on we used his simpler method (Figs. 3 and 7). In patients who underwent jejunoileal bypass, the mean weight was 135 kg (range from 94 to 178 kg), the mean height was 156 cm (range from 125 to 186 cm) and the mean age was 36 years (range from 15 to 61 years). The female to male ratio was 15:1. Towards the end of the study period the Gomez-type[20] of gastric partitioning was becoming popular as a simpler and safer alternative to gastric bypass, so our next 110 patients underwent this procedure. In these patients the mean weight was 127 kg (range from 90 to 207 kg), the mean height was 157 cm (range from 126 to 188 cm) and the mean age was 33 years (range from 18 to 63 years). The female to male ratio was 14:1.

Table I—Criteria for Operation

Presence of morbid obesity, i.e., more than 45 kg overweight or twice the ideal weight for more than 5 years
Failure of conservative treatment for more than 5 years
Stable life pattern
Absence of endocrine disorder
Cooperative
Acceptable operative risk
Absence of alcoholism*

*Because of potential hepatic complications following operation.

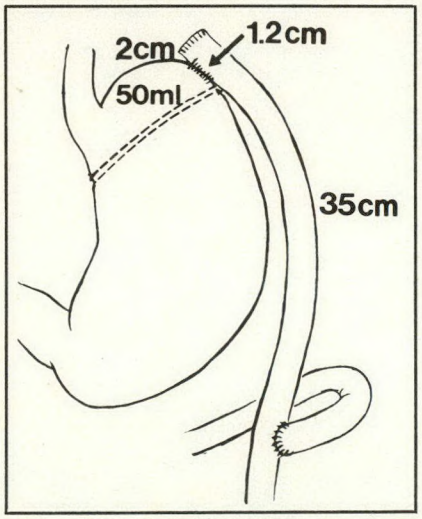

FIG. 5—Extension of Griffen Roux-en-Y gastric bypass.

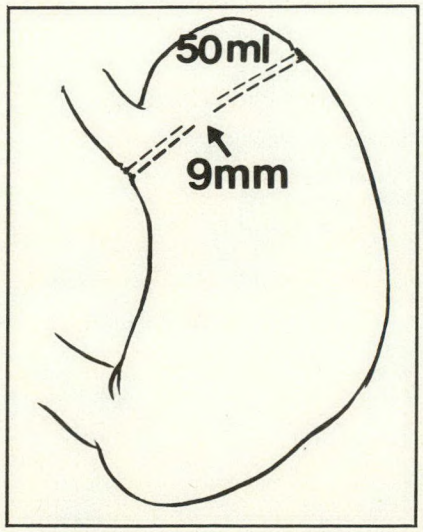

FIG. 6—Gastric partitioning with TA90 (4.8-mm staples) after Pace and associates.[17]

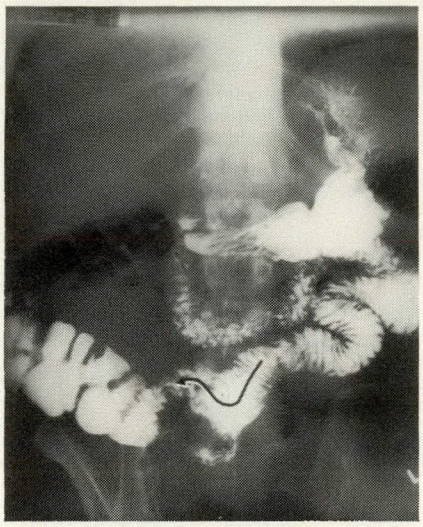

FIG. 7—Payne jejunoileal bypass modified by Palmer and Marliss.[11] Roentgenogram after barium meal at 2 years shows rapid transit to cecum. Reflux into bypassed ileum is negligible.

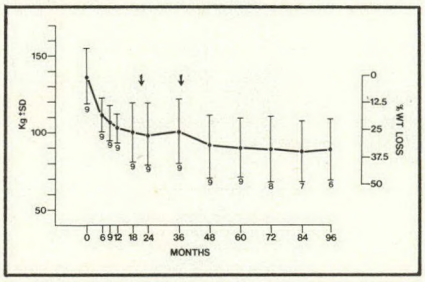

FIG. 8—Weight loss following Scott jejunoileal bypass. Number at each follow-up point is number of patients. Arrows indicate time at which two patients required further resection.

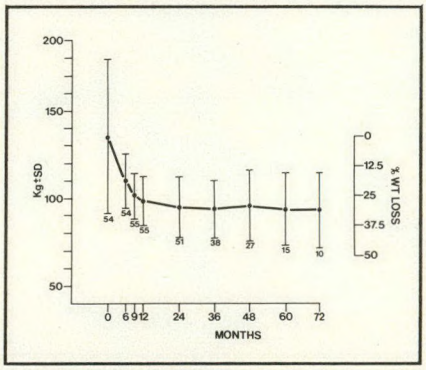

FIG. 9—Weight loss following Payne jejunoileal bypass with Y-shaped anastomosis.

Results

Jejunoileal Bypass

In the nine patients who underwent the Scott jejunoileal bypass, the mean weight loss was 35% (range from 22% to 52%) and had not risen at 96 months postoperatively (Fig. 8). However, two early patients who had slightly excessive measurements at operation required a further resection, at 22 and 37 months respectively, to achieve adequate weight loss.

In patients who had a modified Payne jejunoileal bypass with Y-shaped anastomosis, the weight loss after 72 months was 33% (range from 18% to 53%) and has not risen (Fig. 9). There has been no excessive weight loss.

The effects of the weight loss on the side-effects of morbid obesity are shown in Table II. Blood pressure fell from an average of 180/120 mm Hg with wide cuff to 140/90 mm Hg at 12 months (Fig. 10).

The early postoperative complications are shown in Table III and the late sequelae in Table IV. Hypokalemia was treated by Slow K or Kaon elixir,[10,21] hypomagnesemia by Magnesium-Rougier and hypocalcemia by Calcium-Sandoz.[22] Polyarthritis was eliminated by metronidazole.[10,23] Renal calcium oxalate stones were treated by a low oxalate diet (avoiding chocolates, cola drinks, tea, carrots, celery, spinach, gelatin, nuts, plums and strawberries)[22] and by giving calcium carbonate orally.[10,24] Excess "gas" resolved with metronidazole.

Only one jejunoileal bypass in our series has so far been reversed. The patient was a woman who weighd 123 kg and was 156 cm tall. She suffered from dyspnea, diabetes, osteoarthritis, urinary stress incontinence and recurrent varicose veins. A Payne jejunoileal bypass in March 1976 reduced her weight to 58 kg and the associated problems subsided. How-

ever, she had recurrent oxalate nephrolithiasis and would not cooperate with respect to diet and drug therapy. She underwent reversal of the bypass elsewhere November 1980 and is fighting weight gain.

The adaptation of the functioning small bowel consisted of dilatation and lengthening as well as prominence of the valvulae conniventes, shown by roentgenograms after barium ingestion, and marked elongation of villi associated with cellular hyperplasia (but not cellular hypertrophy) as in Fig. 11. This figure shows the histologic features in the bowel of the patient who required further resection at 37 months because of inadequate weight loss.[25] Bypassed bowel showed mucosal atrophy.

Of the 174 patients in the study, 168 had liver biopsy at the time of operation (Table V). Patients showed hepatic steatosis varying from severe to mild. The principle area of fatty distribution was centrilobular (37%) and diffuse (34%) (Fig. 12). The normal livers (10%) tended to be present in the younger patients.

After jejunoileal bypass, some serial liver biopsy specimens appeared worse at 9 and 12 months, but generally were improved by 30 months over the biopsy taken before operation (Fig. 13). There were transient elevations of serum glutamic oxalocetic transaminase or alkaline phosphatase, or both, which generally returned to normal after 24 months. These elevations in hepatic enzyme levels frequently did not correlate with the liver biopsy findings. All serum bilirubin levels remained normal. All patients were routinely given predigested protein capsules (3 capsules *tid*) containing the lipotropic factor L-methionine (3.9 mg/capsule), and larger doses were prescribed when the results of liver function tests or biopsy findings were abnormal. Patients were also given multiple vitamins and instructed regarding a high-protein, low-fat diet.[26,27] There have been no instances of liver failure.

Diarrhea after 1 year was not a problem. Indiscretions in dietary intake, such as of greasy or oily foods, still caused rapid transit[21] with steatorrhea and anal irritation in a few patients, and Lomotil was occasionally used.[20] Lomotil was also used when an abnormality was found in the electrolyte levels or results of liver function tests, to delay transit and prevent electrolyte and protein loss.

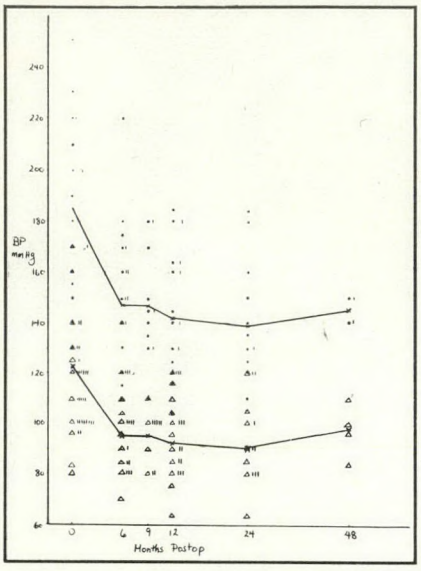

FIG. 10—Mean blood pressure accompanying weight loss. Circles represent systolic pressure, triangles represent diastolic pressure.

Gastric Partitioning

Fig. 14 shows the procedure we used. The left triangular ligament of these fatty livers was not divided. The upper portion of the Polytract retractor with the Gomez hand (Narco Pilling, Downsview, Ont.) placed over the padded liver facilitated exposure. A Penrose drain about the esophagus identified the cardia. The TA90 stapler was applied twice, high on the stomach, after four

Table II—Side-Effects of Morbid Obesity in 64 Patients Who Underwent Jejunoileal Bypass

Side-effect	Before	Effect of weight loss
Hypertension	45 (severe in 16)	Resolved in 24
Diabetes	36 (frank in 8, mild in 22, latent in 6)	No hyperglycemia
Respiratory distress	40 (severe in 17, 2 with Pickwickian syndrome)	Resolved
Hyperlipemia	15 (type II in 2, type IV in 13)	Resolved
Osteoarthritis (knees, ankles, low back)	46 (severe in 22, debilitating in 9)	Striking improvement in 43
Fatty liver	57 (marked in 17)	(See text)
Stasis ulcers and edema	4	Healed in 3
Varicose veins	11	—
Thrombophlebitis	4	—
Cholecystitis	27 (previous cholecystectomy in 19, present cholecystectomy in 8)	—
Urinary stress incontinence	35 of 60 women	Resolved in all but 1
Intertrigo	31	Resolved*
Psychosocial and economic problems	33	General improvement

*20 underwent subsequent lipectomy.

Table III—Early Complications after Jejunoileal Bypass

Complication	No.
Wound infection	1
Seroma	2
Deep vein thrombosis	2
Pulmonary embolism	0*

*2 patients had previous history of pulmonary emboli.

Table IV—Late Complications after Jejunoileal Bypass

Complication	No.
Persisting electrolyte abnormality	16*
Hypokalemia	9
Hypomagnesemia	4
Hypocalcemia	3
Polyarthritis	6
Renal (calcium oxalate) stones	5
Excess flatus and bloating	11
Incisional hernia	6
Mechanical obstruction of bypassed ileum	1†
Intussusception of jejunal stump	1†

*Requiring replacement.
†Same patient (Fig. 11).

Table V—Status of Liver at Operation

Principal area of fat distribution	% of patients (n = 168)
Centrilobular	37.4
Diffuse	34.4
Centrilobular-midzonal	9.8
Midzonal	3.7
Periportal	1.2
Other	3.1
Normal	10.4

staples had been removed from the greater curvature side of the instrument before each application, leaving a 50-ml pouch and a greater curvature channel. A no. 18 F nasogastric tube was placed through the outlet at the end of the operation to decompress both sides of the partition.

This procedure (Fig. 14, left) was performed on 56 patients. The staple lines were secured with through-and-through polypropylene sutures at the corners. The mean weight loss (Fig. 15) was 21% at 12 months but only 16% (range from 0% to 36%) at 30 months, due to stomal enlargement. Two patients weighed the same and two weighed more than preoperatively. After many months there was uninhibited emptying of the proximal pouch and enlargement of the stoma.

The next 54 patients underwent a similar procedure (Fig. 14, right), but a circumferential, seromuscular, imbricating, running, polypropylene suture was inserted against a no. 38 F Maloney bougie, to produce a nonexpandable outlet 1.2 cm in diameter. A large clip on the lesser curvature prevented disruption of the line of staples. We found use of a no. 32 F bougie (10.2 mm opening) unsatisfactory because we had to reoperate 4 to 6 weeks postoperatively to enlarge the outlet in five patients. The mean weight loss with the reinforced nondistensible channel (Fig. 15) at 12 months was 31%, and there has been no weight gain in the five patients now followed up for 15 months.

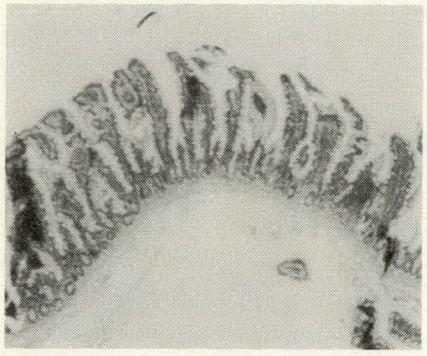

Fig. 11a

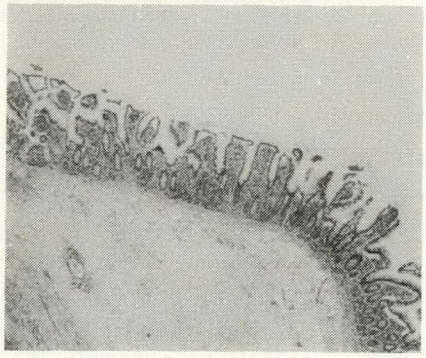

Fig. 11b

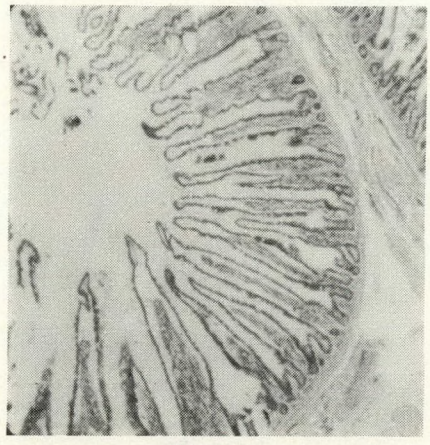

Fig. 11c

FIG. 11—(a) Normal jejunum during Scott jejunoileal bypass. (b) At 24 months after bypass, while pregnant, patient had obstruction of bypassed bowel, which had been drained into sigmoid colon; segment of ileum was resected. Mildly atrophic mucosa can be seen in bypassed bowel 25 cm from sigmoid. Patient subsequently had full-term normal delivery.[25] (c) At 37 months, she had resection of in-continuity bowel for further weight loss. Elongated villi with hyperplasia of cells are seen as adaptation to short bowel. (d) At 47 months there was intussusception of jejunal stump, which was resected. Mucosa is markedly atrophic (hematoxylin and eosin, reduced by 49% from ×25).

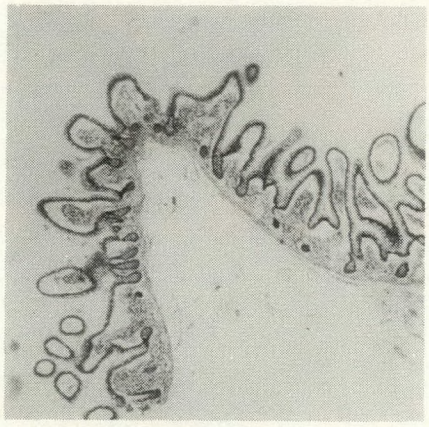

Fig. 11d

FIG. 12—Severe diffuse fatty metamorphosis (>75% of liver infiltrated). Some portal areas are spared (hematoxylin and eosin, reduced by 49% from ×25).

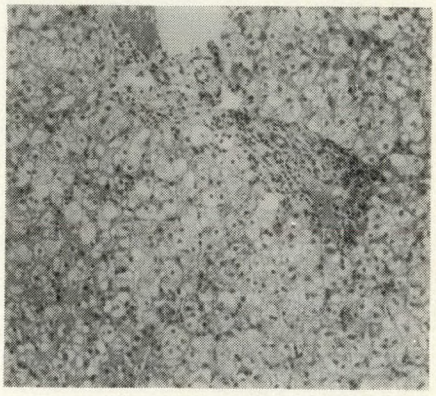

Fig. 13a

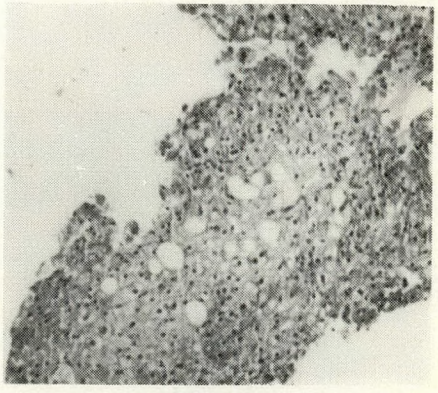

Fig. 13b

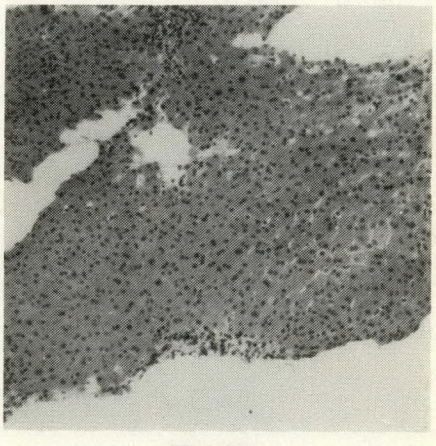

Fig. 13c

FIG. 13—(a) Liver biopsy shows diffuse fatty infiltration at operation. (b) Same patient 12 months after jejunoileal bypass shows improvement (<25% of liver occupied by fat). (c) At 36 months liver is normal (hematoxylin and eosin, reduced by 55% from ×100).

These patients can eat only very small soft meals, chewed well, slowly. Fig. 15, right, includes three of our patients, and one from elsewhere, who had undergone previous gastric partitioning with no control over the channel and who subsequently had the channel narrowed and circumferentially imbricated over a bougie.

The postoperative complications in the 110 patients are shown in Table VI. The five patients requiring reoperation had circumferential imbrication initially against a no. 32 F bougie and were guilty of dietary indiscretion despite instructions to take tiny liquid meals (e.g., from a 30-ml plastic medicine glass) for 8 weeks postoperatively.[18] At reoperation, the through-and-through and circumferential sutures about the channel were removed; closure of the channel and gastrogastrostomy over a bougie would likely be the procedure in later cases. There was no diarrhea since the vagal trunks were preserved, but constipation occurred occasionally in the first 2 months, because of lack of dietary residue.

Fourteen patients had cholecystectomy for cholelithiasis at the time of gastric partitioning (28 had had previous cholecystectomy), and 4 underwent repair of a hiatal hernia using the Hill technique.

When excess trauma occurred in the stomal area during operation, a gastrostomy was placed in the lower stomach for effective decompression and possible later feeding. This was done in 19 patients. A drain was placed in each patient. There have been no leaks or subphrenic abscesses. There have been no metabolic complications after the procedure. The liver has not shown the abnormalities that occasionally occurred after jejunoileal bypass, but has improved directly with the weight loss.

Discussion

To our knowledge all 174 patients who underwent surgery for morbid obesity are alive. Thoracic epidural anesthesia, supplemented by a light general anesthetic, was usually used, and the patients were awake and extubated at the end of the operation.[28] The epidural was maintained for analgesia up to 3 days after operation Patients were able to breath and move with ease, and did not require intramuscular narcotics, which have a respiratory depressant effect. Further, there is evidence that nitrogen balance is improved with epidural analgesia.[29]

If an epidural catheter is to be inserted, minidose heparin can only be started postoperatively, but patients went to the operating room wearing compression stockings. There were no instances of pulmonary embolism.

Jejunoileal bypass produced satisfactory weight loss, but obligates the patient to years of medical surveillance. Scott and associates[6] showed that as the length of bowel is shortened, metabolic complications increased. On the other hand, increasing bowel length permits future weight gain after bowel adaptation; this is anticipated because of the transport capability of the millions of intestinal absorptive cells involved.

The adaptations that maintain the weight as it stabilizes consist of villus hypertrophy in the in-continuity bowel and also in the immediately adjacent bypassed ileum where intestinal contents reflux.[30,31] We[22] and others[32] have found mucosal atrophy in the remaining bypassed bowel. Friedman and associates[33] found villus height in the proximal jejunum to be almost identical to that before jejunoileal bypass, but others[30-32] have not found this.

The major cause of liver deterioration after jejunoileal bypass is believed to be protein malabsorption, especially of methionine and choline, in the first 2 years.[10,11,22] The lack of this complication in our patients may be due to the liberal use of predigested protein capsules. It is known that infusions of amino acids given intravenously or into bypassed jejunum if exteriorized will rapidly reverse the liver changes.

In gastric partitioning, a small pouch to give the sensation of satiety[34] and a small calibrated outlet that will not expand are important.[20] *Double* application of the stapler is necessary to prevent staple-line disruption.[18] Circumferential Marlex mesh about the outlet has been proposed, but has been followed by delayed progressive scarring and stenosis.[11] Buried nonreactive circumferential Silastic tubing may be advantageous.[35] Techniques have been described to leave a centrally located opening in the partition.[17] However, it is difficult to reinforce this central channel posteriorly to prevent its ultimate enlargement. Furthermore, with a central outlet, one may be unsuccessful in applying the stapler twice with the two openings coinciding to permit a channel. We believe that if suture reinforcement is used along the entire length of the partition, this can be difficult and hazardous. Oblique partitions with the outlet on the lesser curvature side have been proposed,[35] but it is known that food tends to pass readily down the lesser curvature Magenstrasse so that weight loss may be unsatisfactory; furthermore, a fundal pouch for distension and to give a sensation of fullness is helpful. The procedures are in a state of development, and many improvements will likely be devised in the next decade. Comparisons of new techniques are now being reported.[36]

Lately, in patients who are unacceptable operative risks, we have used an intragastric balloon inserted trans-

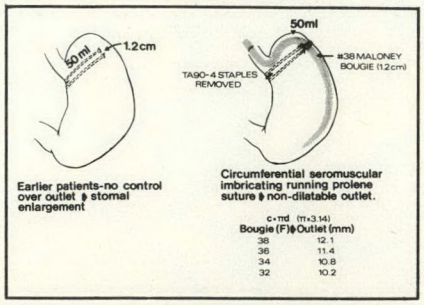

FIG. 14—Left: gastric partitioning as performed on first 56 patients with no control over outlet; this led to stomal enlargement. Right: gastric partitioning with nonexpandable outlet performed in last 54 patients.

Table VI—Complications after Gastric Partitioning

Complication	No.
Early	
Transient outlet obstruction	5
Persistent outlet obstruction requiring reoperation	5
Wound seroma	11
Late	
Ventral hernia	8

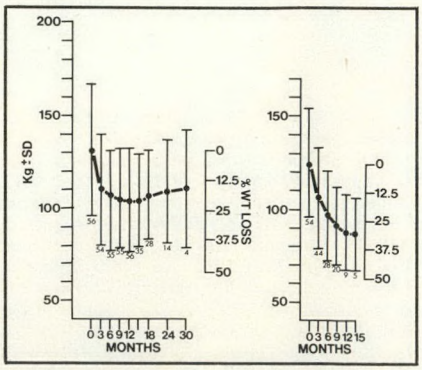

FIG. 15—Weight loss following gastric partitioning without reinforcement of outlet (left) and with reinforcement by nonexpandable imbricating Prolene suture about outlet (right).

nasally (Fig. 16). Control over the volume of air in the balloon has been maintained by a fine nasogastric tube taped externally over the ear. However, the weight loss will likely be limited to the period that the balloon is in situ.

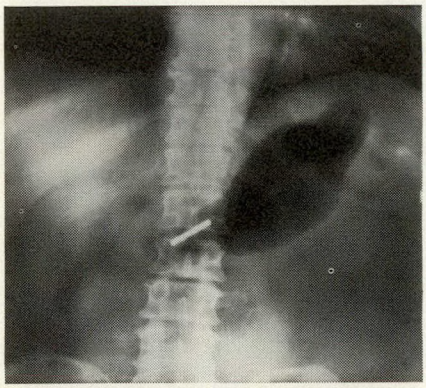

Fig. 16a

Fig. 16b

FIG. 16—Patient with (a) intragastric balloon in place to provide early satiety. (b) This man lost 44 kg in 5 months with resolution of stigmata of morbid obesity.

Conclusions

Jejunoileal bypass, using critical measurements, has provided acceptable and lasting weight loss, but the sequelae require prolonged surveillance. Gastric partitioning with a pouch capacity of 50 ml is not associated with long-term complications but provides unreliable weight loss due to the expanding outlet. The procedure may provide as effective weight loss as jejunoileal bypass if a secure technique is used to prevent subsequent enlargement of an outlet of 1.2 cm or less.

Addendum

After a further 6 months of follow-up, the mean percentage weight loss after gastric partitioning with the reinforced channel is 34% in the 14 patients who have passed 18 months. There continues to be a variation in the percentage weight loss in different patients, despite the calibrated operation. Experience with a further 43 patients suggests that the no. 36 F bougie may give optimal results in the procedure performed.

We thank Drs. Segundo M. Mariz and Frederick Harris of the department of pathology, the department of anesthesia, Dr. Douglas A. Thompson of the division of nephrology, and the departments of photography and medical records for assistance.

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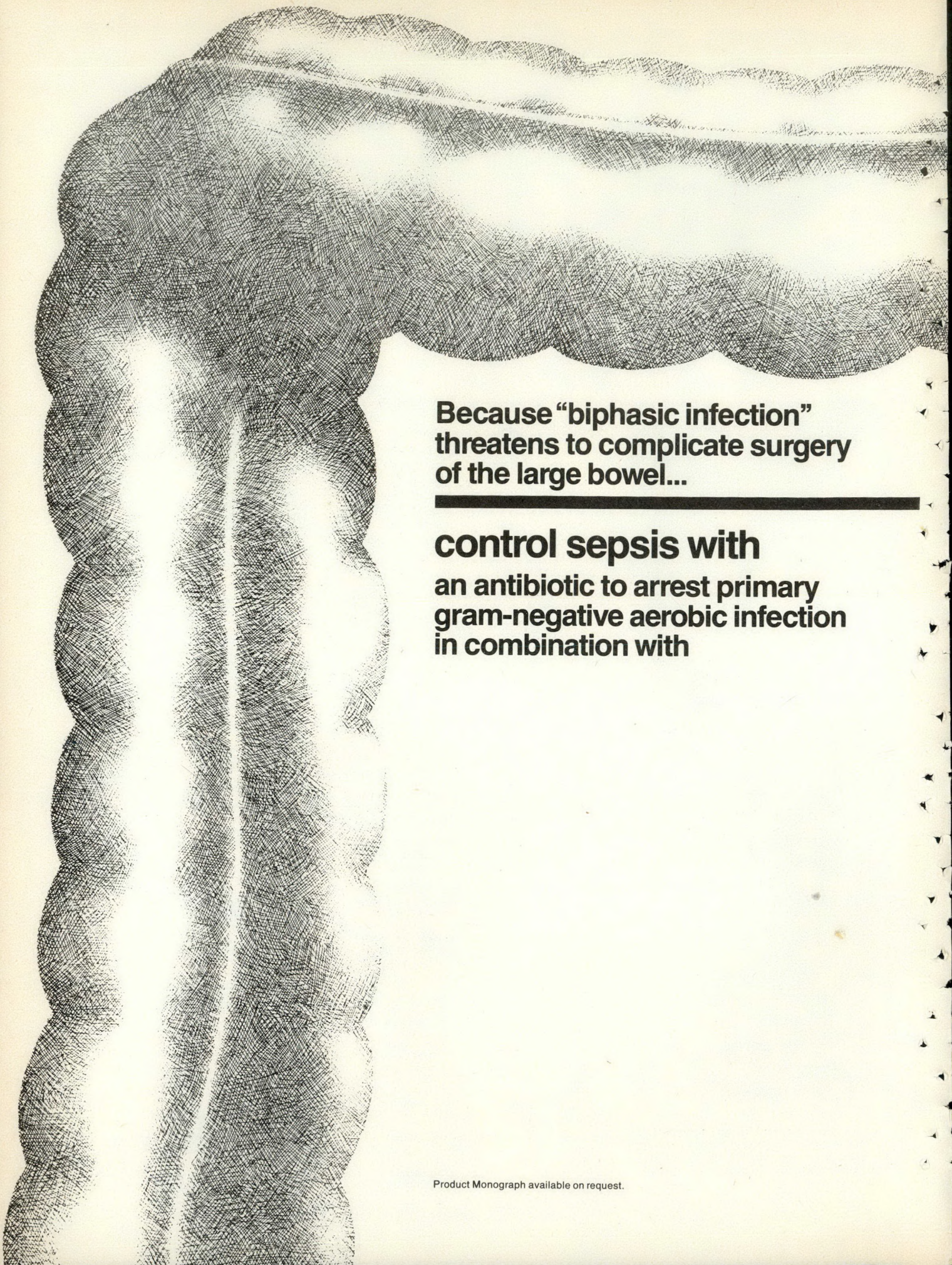

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Treatment Outcome and Efficiency in Surgery

T.H. Brian Haig, MD, PH D, FACS, FRCS[C]

Health gains for patients treated on the surgical service of the University Hospital in Saskatoon have been measured using an illness index matrix and these gains have been related to financial costs. Although many patients benefited, 46.5% did not, either because they suffered self-limiting complaints or because their diseases were beyond the surgeon's ability to help. This 60-bed surgical service generated costs of $8 million in 1979, of which about $3 million were for services from which there was little or no gain for the patient. Since 72% of expenditures were for basic bed, board and nursing costs, more exacting use of hospital beds holds the greatest potential for increasing efficiency. There is evidence, too, that our use of medical manpower may be improvident.

Les bénéfices pour la santé chez les patients traités dans le service de chirurgie de l'Hôpital Universitaire de Saskatoon ont été mesurés à l'aide d'une grille d'indices de maladie, et ces bénéfices ont été rattachés à leurs coûts financiers. Bien que plusieurs patients aient profité des traitements, il n'en a pas été de même pour 46.5%, soit parce qu'ils souffraient d'affections qui évoluent spontanément vers la guérison, soit que leur maladie dépassait la capacité d'intervention du chirurgien. En 1979, ce service chirurgical de 60 lits a entraîné des coûts de $8 millions dont $3 millions sont allés pour des services n'offrant que peu ou pas de bénéfices pour le patient. Comme 72% des dépenses passent pour des coûts de base tels que le gîte, le couvert et le personnel infirmier, la meilleure façon d'améliorer l'efficacité serait de faire un usage plus parcimonieux des lits d'hôpitaux. Il existe aussi des indices d'une utilisation abusive des effectifs médicaux.

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In 1979, Canadians spent $18 579 959 000 on health care.[1] Of this sum 90% went towards personal services for patients, leaving less than $2 000 000 000 for health promotion through social and community interventions. Since, to a very large extent, the determinants of health lie outside the sphere of therapeutic medicine,[2-5] one must question the disproportionate allocation of expenditures towards therapy, even in a developed country like Canada. Indeed, McKinlay and McKinlay[6] contended that despite current levels of health and longevity in the United States, about 92% of all health gains over the past 100 years occurred before the surge in medical spending began in the mid-1950s. The same picture emerges from Britain.[7]

Still, medical care has contributed to the health of our population over the past 50 years,[8] as it has to comforting the sick for much longer than that. What is perhaps less apparent, but more disturbing, is that we seem to be putting more and more of our limited resources into interventions from which expectations of gain are least.[9-13]

What we lack is a good way of measuring the health gains that do accrue from medical interventions and of determining the cost of each increment of gain. Without this knowledge we cannot judge how the many components of medical care compare with alternative ways of allocating resources to improve health. This work reports on one set of techniques that can be used for measuring therapeutic outcome and relates the outcome to cost.

Material and Methods

Our approach was to measure the improvement in health experienced by individuals as a result of treatment in hospital and to tabulate the cost of that treatment; then we calculated the dollar cost of each increment of benefit to give a measure of efficiency. We did this for a random sample constituting 20% of all patients treated on the adult general surgery service of University Hospital in Saskatoon during 1979, using hospital charts as the sole source of information.

Working from concepts espoused by Williams[14] and by Patrick and colleagues,[15] we first defined illness as having three components — *discomfort, dysfunction* and *prognosis*. Independent assessments of discomfort and dysfunction were made for the first day each patient entered the surgical ward in 1979. The discomfort component was measured using a 10-point ordinal scale having three levels of severity and three levels of frequency, with the absence of discomfort and death, as the two extremes. For dysfunction we used the cardinal scale developed and tested by Bush and colleagues,[15-17] but we simplified it to a 10-point scale by grouping entries wherever preference values tended to cluster. We then had two coordinates — one a measure of discomfort and the other a measure of dysfunction — descriptive of each patient's condition on the day of admission to hospital. We plotted these coordinates on an illness index matrix upon which indifference curves had been drawn as described by Williams[14] and by Culyer;[18] each indifference curve was assigned the preference value ascribed to the corresponding dysfunction value. This simplification was considered satisfactory for an initial study of the method and gave us the desirable attribute of preference values on a cardinal scale. Illness index measurements of like character were then made for each day after the patient's admission for 1 year and a time profile curve was drawn that was descriptive of the patient's course for this 1 year. A second curve was then drawn for each patient, as outlined by Williams[14] and by Culyer,[18] descriptive of that patient's probable course had treatment not been undertaken. For this information we went to various sources in the medical literature,

drawing particularly heavily upon Eiseman's recent study[19] of prognosis in surgical disease. Using Williams' technique of subtracting the area under the curve depicting the course with treatment from that under the curve delineating prognosis in the absence of treatment, we derived a value descriptive of the gain that the patient achieved from therapy. We call this the social utility value because it describes the benefits derived by that person as a social being and not simply as a patient with a specific medical diagnosis.

Costs of hospital care, too, were tabulated individually for each patient, but no attempt was made either to assess social costs or to convert these dollar equivalents. The hospital business office gave us a per diem bed cost for each bed, including hostelry, dietary and nursing costs; this base figure was used for each hospital day. To it was added the physicians' fees, and costs of drugs, laboratory and radiologic services and operating room facilities; for these categories each patient was treated individually by listing, then costing out, all of the services and materials applied to that patient while in hospital.

Efficiency was defined as the dollar cost of each social utility unit and was calculated by dividing the total cost of treatment by the social utility value derived from that treatment.

Results

The distribution of social utility values plotted against costs is illustrated in Fig. 1; it shows a skewing to the positive benefit side, but also shows a heavy clustering towards zero. Positive social utility values were attained by 53.5% of patients. For the 46.5% of patients who derived zero or negative benefits, 40.6% of the total resources were expended. This, projected to the whole service, would amount to about $3 205 855 out of a total expenditure of $7 895 918 for the general surgical service in 1979. Thirty-six (11.4%) patients in the sample had costs exceeding $10 000 and consumed a total of $566 472, which amounted to 36% of the total resources for this sample. These patients were a decade older than average and their mean gain was about half that of the whole sample, but 61% of them did benefit.

Table I shows that basic bed costs accounted for 72% of the total, while investigative services cost about 8% and therapeutic materials and services took almost 20%. The proportion attributable to basic bed costs was somewhat greater for those who did not benefit, as were the investigative costs, while therapeutic services were proportionately a little higher for the group achieving positive health benefits from their stay in hospital.

Table II records the age and sex distribution for the sample as it was divided, somewhat arbitrarily, into disease categories. The mean social utility value for the entire sample was +56.98, and for 16 of the 18 disease categories the social utility value was positive. The magnitude of benefit did vary substantially between disease categories, but there was little constant relation between the degree of benefit and either age or sex. The proportion of patients who benefited varied appreciably between disease categories as well, but in 13 of the 18 categories more people benefited than did not, with an average of 75% having a positive social utility value.

Few patients with benign breast or endocrine disease benefited, principally because of the relatively trivial nature of their illnesses. Treatment outcome was disappointing also for those with disease of the lungs or thorax, exclusive of carcinoma, but in these cases it was usually because they suffered from serious diseases for which little could be done. While many patients with peripheral arterial disease did gain substantially from treatment, a relatively large proportion of persons with these chronic, disabling diseases gained nothing.

Of all patients treated on this service, 45% had complaints referable to the gastrointestinal system, exclusive of cancer. Most who suffered from the common specific diseases — cholelithiasis, anal and perianal disease, hernia, bowel obstruction and appendicitis — gained substantially from treatment. However, 40% of this large group had a variety of more nebulous complaints and only a quarter of these were helped as a result of their hospital treatment; often the cause was not discovered. Efficiency in this group, as in almost all others, was substantially greater in those patients who were operated on. This reflects the fact that overall, more could be achieved when operation was possible.

Table III catalogues the total cost of treatment per patient for each disease category and shows the derivation of these costs. Overall expenditures generally reflect the complexity of therapeutic requirements, so illnesses requiring sophisticated technology, highly skilled nursing and ancillary care and very specialized medical procedures cost most. In all

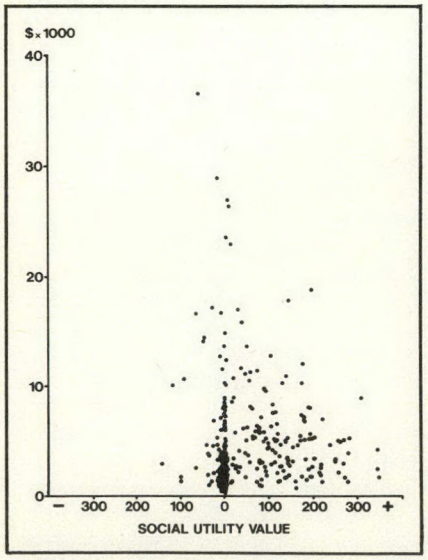

FIG. 1—Scattergram showing cost–benefit coordinates for each of 316 patients.

Table I—Costs

Costs	Total sample (N = 316)	Positive SUV* (n = 169)	Negative SUV† (n = 147)
Total, $	1 579 183	938 012	641 171
Bed/board/nursing, $ (%)	1 146 791 (72.62)	(71.75)	(73.89)
Investigation, $ (%)			
Laboratory	69 512 (4.40)	(4.40)	(4.40)
Radiology	43 142 (2.73)	(2.55)	(3.00)
Miscellaneous	10 343 (0.66)	(0.64)	(0.67)
Therapy, $ (%)			
Physicians	168 378 (10.66)	(11.17)	(9.93)
Drugs	76 007 (4.81)	(4.97)	(4.59)
Operating room/ miscellaneous	64 961 (4.11)	(4.50)	(3.54)

*Patients who derived a social utility value (SUV) greater than zero.
†Patients who derived a social utility value of zero or less.

categories, however, basic bed costs accounted for by far the largest expenditure and investigative procedures the smallest. For most groups of patients the tabulations reflect quite well the entire dollar cost of treatment for the particular illness. This would not be true for cancer patients, however, as some will have received intensive courses of chemotherapy or radiotherapy after leaving hospital. Similarly, expenditures for cardiac patients will be biased towards the low side because virtually all these patients were investigated on the cardiology service during an admission before, and distinct from, their admission to the surgical service. These services would add from $1000 to $2000 to the overall costs generated by each of these patients

Table II—Demographic and Efficiency Characteristics

Category of disease	No. of patients	Age, yr*	Sex ratio male : female	Social utility value*	% deriving positive social utility value	Efficiency†	% undergoing operation	Efficiency for operation‡
Cholelithiasis	19	46 (20)	2 : 17	84.14 (85.66)	68	44.12	95	44.14
Anal/perianal	15	54 (16)	9 : 6	68.35 (68.25)	80	41.58	93	40.34
Groin hernia	13	55 (15)	13 : 0	101.19 (101.52)	77	20.33	85	19.54
Small bowel obstruction	13	45 (22)	5 : 8	95.30 (94.62)	77	60.96	54	52.13
Appendicitis	12	25 (13)	8 : 4	167.46 (148.63)	75	12.55	100	12.55
Peptic ulcer disease	12	52 (19)	11 : 1	67.92 (119.72)	42	56.65	25	40.52
Other gastrointestinal/ abdominal§	58	52 (24)	24 : 34	21.32 (61.82)	26	176.10	36	117.63
Trauma	28	41 (21)	22 : 6	74.64 (90.32)	64	84.45	71	82.01
Coronary artery disease	17	51 (8)	14 : 3	77.85 (72.65)	71	93.64	100	93.64
Valvular heart disease	13	57 (16)	8 : 5	57.92 (97.55)	54	137.00	92	133.69
Other cardiac disease	8	72 (13)	5 : 3	98.24 (54.23)	100	39.48	88	33.59
Peripheral artery disease	34	67 (12)	29 : 5	45.92 (101.84)	47	169.52	88	164.85
Venous disease	9	52 (17)	3 : 6	58.46 (70.97)	56	61.23	44	29.19
Carcinoma	36	65 (16)	20 : 16	46.77 (60.95)	58	148.97	75	138.41
Soft tissue tumour	3	46 (18)	1 : 2	139.70 (71.85)	100	8.26	100	8.26
Breast/endocrine§	18	49 (11)	0 : 18	−22.84 (41.82)	11	N/A‖	83	N/A
Lung/thorax§	9	57 (11)	6 : 3	−3.65 (6.87)	22	N/A	44	N/A
Renal§	3	75 (15)	1 : 2	95.34 (69.17)	100	98.73	67	101.30
Total sample	316¶	53 (19)	179 : 137	56.98 (89.38)	54	87.69	72	79.60

*Mean (± standard deviation).
†Efficiency = mean total cost ÷ mean social utility value = the mean dollar cost for purchase of each social utility unit.
‡Efficiency calculations confined to those patients in this category who underwent operation.
§Excluding malignant neoplasms and renal transplants.
¶Total sample slightly less than total for categories because a few lesions fell neatly into more than one category.
‖This calculation is meaningless when social utility value is zero or less.

Table III—Distribution of Costs within Illness Categories

Category of disease	Total cost/patient, $*	Bed costs†		Investigation costs‡		Therapeutic costs§	
		$*	%	$*	%	$*	%
Cholelithiasis	3712 (1328)	2864 (1109)	77.16	158 (87)	4.26	690 (195)	18.58
Anal/perianal	2841 (2374)	2212 (1909)	77.8	87 (68)	3.05	541 (427)	19.04
Groin hernia	2058 (859)	1541 (632)	74.87	78 (66)	3.80	448 (230)	21.77
Small bowel obstruction	5810 (3535)	4532 (2701)	78.01	367 (447)	6.32	930 (619)	16.01
Appendicitis	2102 (1161)	1534 (898)	72.98	103 (133)	4.88	464 (139)	22.07
Peptic ulcer disease	3848 (2666)	2984 (2250)	77.55	316 (173)	8.21	548 (373)	14.24
Other gastrointestinal/ abdominal¶	3721 (3221)	2996 (2638)	80.51	237 (243)	6.37	491 (468)	13.21
Trauma	6303 (5608)	4176 (3496)	66.25	652 (785)	10.35	1446 (1560)	22.94
Coronary artery disease	7290 (5807)	4593 (4993)	63.01	642 (332)	8.81	2053 (537)	28.17
Valvular heart disease	7935 (3728)	4373 (2683)	55.11	1346 (900)	16.96	2239 (805)	28.21
Other cardiac disease	3879 (1936)	2908 (1593)	74.97	216 (153)	5.59	755 (403)	19.46
Peripheral artery disease	7784 (7444)	5718 (6208)	73.45	471 (549)	6.05	1595 (1281)	20.49
Venous disease	3579 (5591)	3057 (5169)	85.41	91 (53)	2.55	431 (417)	12.04
Carcinoma	6967 (5659)	5321 (4339)	76.38	534 (633)	7.67	1109 (919)	15.92
Soft tissue tumour	1154 (183)	877 (117)	75.93	44 (31)	3.81	234 (148)	20.25
Breast/endocrine¶	1866 (1265)	1259 (805)	67.47	121 (162)	6.47	486 (346)	26.06
Lung/thorax¶	5472 (5724)	4203 (4419)	76.81	403 (463)	7.37	866 (900)	15.82
Renal¶	9413 (3792)	6945 (2413)	73.78	703 (343)	7.47	1765 (1166)	18.75
Total sample	4997 (4884)	3629 (3737)	72.62	389 (312)	7.79	979 (607)	19.58

*Mean (± standard deviation).
†Bed, dietary, nursing and related support costs.
‡Laboratory, radiologic, electrocardiographic and related costs.
§Physicians' fees, drugs, operating room and related costs.
¶Excluding malignant neoplasms and renal transplants.

and since they are not included in our figures, efficiency calculations for both of these groups is somewhat overstated.

Discussion

The crux of this work is the measurement of treatment outcome. Over the past three or four decades several methods have become available to accomplish this; some are useful, though of restricted applicability. Culyer[18] discussed a number of these in some depth and delved into the conceptual difficulties encountered with them. In addition, Sullivan[20] and Torrance[21] pointed out problems inherent in developing health status indices of the sort used in this study. Certainly the obstacles are real, but they must be overcome if we are to measure the benefits provided by our therapeutic interventions. The method used in this work lacks polish, but it has two outstanding virtues. First, it is applicable to all categories of illness and to all types of therapy, and second, its measurements are blind to medical diagnoses. This latter attribute has several important implications. Complications and other unsought outcomes are accounted for easily and their effects weighted no differently from the primary complaints. Moreover, when the methodology is refined sufficiently for prospective studies, nonmedical personnel will be able to take the required measurements, thereby reducing costs and, perhaps more important, shielding patients from medical influence in making their assessments of outcome.

Of greatest concern with the methodology is our rudimentary ability to predict the natural history of untreated disease. For short-term analyses, such as in this study, the medical literature supplemented by first-hand experience works reasonably well, but is less rigorous than one would wish. For long-term estimates, the literature seems inadequate and personal experience unreliable. Some alternative tactic may be necessary. The assignment of preference values requires more study and refinement, too, for while the dysfunction scale of Bush and his colleagues[15-17] is a tested and very good starting point, it is not comprehensive enough, and it was calibrated using healthy young people rather than a patient population. The discomfort scale we used is crude and has not been rigorously tested. The cost calculations are undoubtedly subject to some error as well, particularly with reference to the per diem component for bed, board and nursing services: there is bound to be some variation in requirements between patients. By sequestering and independently tabulating investigative and therapeutic costs, however, we have fairly accurately calculated the real costs engendered by each patient studied.

While recognizing and accepting the above caveats, we can learn several useful things from this first run at measuring treatment outcome and efficiency. First, it is evident that bed costs are by far the greatest of the cost components and investigative costs the smallest. So, in considering cost containment, keeping people who do not need acute care out of hospital would be by far the most effective way of reducing the expense of medical care. Given our system of publicly funded personal medical services, however, incentives for patients, for physicians and for hospitals all tend to increase rather than to limit hospital use. On our particular service the miscellaneous cases of gastrointestinal and abdominal disease are those for which we might improve our performance most in this regard. Second, limiting laboratory and radiologic investigations likely has merit in protecting patients from unnecessary discomfort and harm, but the economic impact of these efforts will be modest. A third observation is that therapeutic services, including the professional component, were much less intensely applied to the usual general surgery patients than they were to trauma patients — now largely taken over by subspecialists — and to patients with cardiac and arterial disease. While it is recognized that a tertiary care hospital is a poor reflection of the larger community, this observation must make us wonder whether very expensive medical manpower is being as well used as it should be or whether our general surgeons are being overtrained and underused.

Conclusions

The measurement of therapeutic outcome in surgery is possible and can be combined with cost analysis to evaluate efficiency. The application of a prototype measure of this sort allows us to see that we can probably improve the use we make of hospitals and of medical personnel.

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Gastric Variceal Bleeding Due to Occlusion of Splenic Vein in Pancreatic Disease

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Nine patients with gastric varices secondary to occlusion of the splenic vein, which resulted from pancreatic disease, were treated by the authors between 1973 and 1981. Profuse hemorrhage, recurrent bleeding and hypochromic anemia were investigated by endoscopy, gastrointestinal roentgenography and selective angiography. Pancreatic disease was defined by ultrasonography, endoscopic retrograde pancreatography and operation. There were three cases of chronic pancreatitis, three of pancreatic abscess, two of pseudocyst and one of carcinoma of the pancreas.

Definitive control of upper gastrointestinal bleeding from gastric varices was achieved by combining splenectomy with selective pancreatic surgical procedures. Two patients did not undergo operation. One death resulted from an unresectable carcinoma. Bleeding did not recur in six patients followed for up to 8 years after operation.

Entre 1973 et 1981, les auteurs ont traité neuf patients atteints de varices gastriques secondaires à une occlusion de la veine splénique causée par une maladie du pancréas. Une hémorragie abondante, des saignements répétés et une anémie hypochrome étaient investigués par endoscopie, radiographie gastro-intestinale et angiographie sélective. La maladie pancréatique était précisée par échographie ultrasonique, pancréatographie endoscopique rétrograde et à l'opération. On identifia trois cas de pancréatite chronique, trois d'abcès pancréatique, deux de pseudo-kyste et un cancer du pancréas.

L'arrêt définitif des hémorragies gastro-intestinales supérieures dues à des varices gastriques fut obtenu par une splénectomie associée à des interventions chirurgicales sélectives du pancréas. Deux patients n'ont pas subi d'opération et un décéda d'un cancer non résécable. Il n'y eut pas de récidives des saignements chez six patients suivis jusqu'à 8 ans après l'opération.

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Upper gastrointestinal hemorrhage is one of the most common reasons for emergency admission to a gastrointestinal service. Endoscopy facilitates early identification of the source of bleeding in most patients. However, in a minority no active bleeding site is identified. The cause of the hemorrhage is frequently attributed to gastritis. Such gastric bleeding may be associated with pancreatic disease. Varices may result when splenic vein occlusion occurs from the extrinsic effects of such disease. This represents a form of localized, or left-sided, portal hypertension. The management of gastric variceal bleeding from this cause is quite different from that related to cirrhosis. In this paper we discuss the etiology, diagnosis and management of this unusual cause of gastrointestinal hemorrhage.

Patients and Treatment

The series comprised nine patients, seen between 1973 and 1981 (Table I). Five presented with frank upper gastrointestinal bleeding, three with hypochromic, microcytic anemia and one was found incidentally to have gastric varices at operation for chronic pancreatitis. All had pancreatic conditions documented by ultrasonography, endoscopic retrograde pancreatography and operation. Disease included pancreatic abscess, pseudocyst, chronic pancreatitis and carcinoma of the pancreas.

Gastrointestinal roentgenography was not diagnostic of gastric varices in eight patients who underwent this investigation. Endoscopy similarly was nondiagnostic (Table I). Celiac angiography diagnosed the condition in the seven patients who were studied, and in two cases the diagnosis was established at operation.

Six patients had definitive surgical procedures and have had no recurrence of their gastrointestinal bleeding during follow-up from 6 months to 8 years. One patient (no. 9, Table I) with extensive carcinoma of the body of the pancreas underwent a palliative gastroenterostomy and died 5 months later.

Two patients did not have definitive treatment. One patient (no. 8, Table I) had only hypochromic anemia and refused operation. The second (no. 5), aged 75 years, had moderate bleeding that ceased spontaneously. Because of age and associated illnesses, she was managed conservatively and splenectomy will be performed only if bleeding recurs.

Discussion

Many disease processes may cause occlusion of the splenic vein with preservation of flow in the remaining portal venous system. The causes may be intrinsic or extrinsic. Intrinsic causes, which are extremely rare, include myeloproliferative disorders and umbilical vein catheterization.[1,2] Extrinsic causes relate to structures contiguous to the splenic vein — the pancreas anteriorly and the retroperitoneum posteriorly. Rarely, retroperitoneal fibrosis, lymphoma or hematoma may produce compression of the splenic vein resulting in thrombosis.[2,3] More commonly, the pancreatic lesion obstructs flow. Pancreatic diseases include chronic pancreatitis, congenital cyst, pseudocyst, abscess and carcinoma.[2,4-6] Splenic vein thrombosis is not uncommon with chronic pancreatitis. Rignault and colleagues[7] described 20 patients with chronic pancreatitis who underwent percutaneous splenic portography; 8 were found to have isolated splenic vein thrombosis. Similar results have been reported by Salam and associates.[8]

Table I—Clinical Features of Nine Patients Presenting with Gastric Varices

Patient no.	Age, yr	Sex	Clinical presentation	Pancreatic disease	Previous surgery	Gastro-intestinal radiologic findings	Endoscopic findings	Angio-graphic findings	Surgical treatment	Follow-up, mo
1	43	M	Recurrent bleeding	Abscess	Vagotomy and pyloroplasty	ND	ND	SVT	Splenectomy	96
2	47	F	Profuse hemorrhage	Abscess	—	—	ND	—	Distal pancreatectomy and splenectomy	60
3	43	M	Recurrent bleeding	Pseudocyst	Vagotomy and pyloroplasty	ND	ND	SVT	Cystogastrostomy and splenectomy	60
4	55	M	Bleeding	Abscess	Cystojejunostomy	ND	ND	SVT	Distal pancreatectomy and splenectomy	18
5	75	F	Profuse hemorrhage	Pseudocyst	Cystogastrostomy	ND	ND	SVT	—	8
6	37	M	Pain	Chronic pancreatitis	—	ND	ND	—	75% pancreatectomy and splenectomy	8
7	59	F	Anemia	Chronic pancreatitis	External drainage of pseudocyst	ND	ND	SVT	75% pancreatectomy and splenectomy	6
8	57	F	Anemia	Chronic pancreatitis	Pancreatico-jejunostomy	ND	ND	SVT	—	6
9	69	F	Anemia, cachexia	Carcinoma	—	ND	—	SVT	Gastroenterostomy	Dead, 5

ND = nondiagnostic, SVT = splenic vein thrombosis.

Once the splenic vein has become occluded, the venous flow may take various routes (Fig. 1). The short gastric veins may drain to the gastroepiploic arcade or across the fundus of the stomach to the left gastric vein (coronary vein). The fundal veins may drain through the esophageal venous plexus, producing portosystemic collaterals, which may also form between the spleen and the retroperitoneum. Previous operation may have altered the venous anatomy, producing additional collateral pathways. Burbige and associates[9] described a case of bleeding from colonic varices when thrombosis of the splenic vein from chronic pancreatitis occurred after previous gastrectomy had disrupted gastric collateral vessels.

Variceal hemorrhage secondary to splenic vein occlusion should be considered in any patient with pancreatic disease who presents with upper gastrointestinal bleeding (Table II). On physical examination, splenomegaly in the absence of other signs of portal hypertension or cirrhosis may be noted in about 50% of patients.[10] A pancreatic lesion may be confirmed promptly by abdominal ultrasonography, enhanced by grey scale and real time scanning. Endoscopic retrograde pancreatography will define any abnormalities of the pancreatic ducts. Air contrast roentgenography after barium ingestion will rarely confirm the diagnosis. Gastric fundal varices may assume either a localized polypoid configuration or produce multiple rounded submucosal projections, which may simulate tumour or hypertrophic rugal folds (Fig. 2).[11-13] Endoscopy is extremely accurate in demonstrating esophageal varices but usually cannot identify gastric varices because of the thickness of the gastric mucosa. Endoscopically, gastric varices may mimic the appearance of hypertrophic folds. In the absence of ulcer, gastritis or other causes of upper gastrointestinal bleeding, the endoscopist should suspect gastric varices.

Celiac arteriography is the definitive procedure for identifying gastric varices and splenic vein occlusion. Any patient with upper gastrointes-

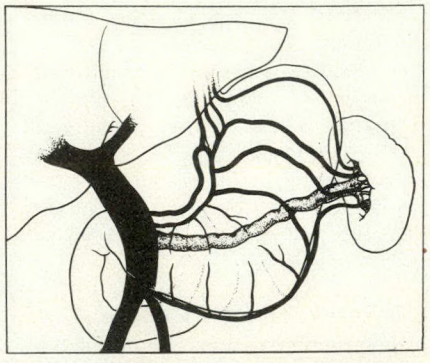

FIG. 1—Collateral venous return from spleen may become varicose following thrombosis of splenic vein.

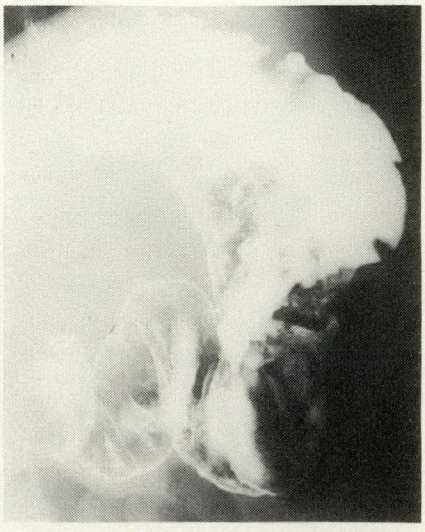

FIG. 2—Gastric barium roentgenogram shows fundal varices which simulate polyps or hypertrophic gastric rugae.

Table II—Investigations Recommended for Bleeding from Suspected Gastric Varices

Gastric bleeding	Endoscopy Air contrast upper gastrointestinal roentgenography
Pancreatic disease	Ultrasonography or computerized axial tomography Endoscopic retrograde pancreatography Operation
Splenic vein occlusion	Selective arteriography and venography Measurement of hepatic venous pressures

tinal bleeding of unknown cause should undergo angiography.

The venous films, following selective splenic artery injection, will demonstrate filling of the splenic parenchyma but not of the main splenic vein (Fig. 3). Subsequently, the collateral venous flow will be seen. Usually this flow is to the portal vein by way of the gastroepiploic or coronary veins. The contributing varices in the gastric fundus will be easily identified, contrasted against an air-filled stomach (Fig. 4). Venous phase films after injection of the superior mesenteric artery can be used to demonstrate the anatomy of the portal vein and rule out extrahepatic obstruction of the portal vein. The gastric and esophageal collateral veins will not be opacified by this injection.

Measurement of wedged and free hepatic venous pressures will give normal results, ruling out portal hypertension from chronic liver disease. This procedure should always be completed at the time of angiography except when profuse hemorrhage necessitates emergency operation.

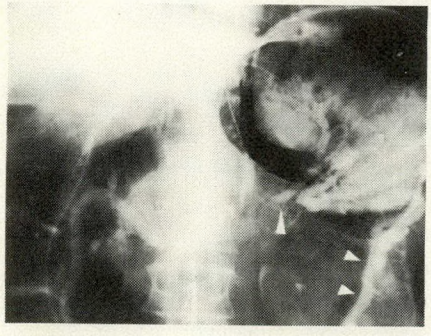

FIG. 3—Venous phase angiography shows complete occlusion (arrow) of splenic vein at hilum with collateral return through gastroepiploic veins (arrows).

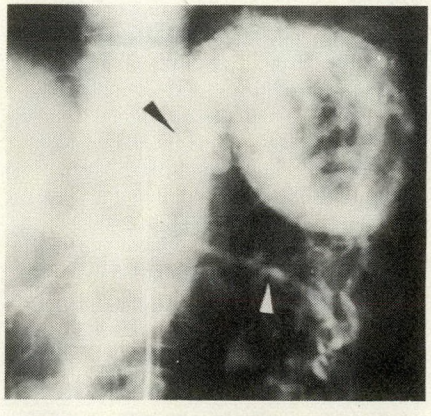

FIG. 4—Massive fundal varices drain to portal system through coronary (black arrow) and gastroepiploic (white arrow) veins.

Pancreatic disease may cause either gastric variceal bleeding from occlusion of the splenic vein, usually at a nonexsanguinating rate, or brisk arterial hemorrhage from rupture of a pseudoaneurysm. Patients with chronic pancreatitis are particularly at risk, because splenic vein occlusion is common in this group. Moreover, 10% of these patients have pseudoaneurysms of pancreatic arteries (splenic artery, gastroduodenal artery secondary to pseudocyst formation).[14]

In patients with severe upper gastrointestinal bleeding who undergo emergency surgery, the diagnosis of splenic vein occlusion may be established at operation. The operative findings have been described by Salam and associates.[8] The triad of dilatation of the gastroepiploic vein, enlargement of the spleen and a normal liver, associated with gastric or gastroesophageal varices, is highly suggestive (Fig. 5). The diagnosis may be confirmed by demonstrating pancreatic disease and by finding a normal portal vein pressure. Portal manometry and venography can be carried out by threading a catheter into a tributary of the superior mesenteric vein.

Once the diagnosis of bleeding varices secondary to splenic vein occlusion is established, the definitive surgical procedure is splenectomy. There may be extensive portosystemic collaterals in the perisplenic ligaments, so it is wise to ligate the splenic artery before mobilizing the spleen. Little and Moossa[15] described an anterior gastrotomy for oversewing the varices, because in their series active bleeding was not stopped by simple splenectomy. This has not been our experience and rebleeding has not been a problem in other published series.[4,8,16-18]

Patients with pancreatic disease associated with occlusion of the splenic vein may require splenectomy combined with selective pancreatic surgery. Occlusion associated with a pancreatic pseudocyst has been reviewed by Salam and associates.[8] Cystogastrostomy should be avoided because of possible hemorrhage from the stomach. Roux-en-Y cystojejunostomy is the procedure of choice for draining the pseudocyst. Pseudocyst decompression alone will rarely relieve splenic vein occlusion, so splenectomy should also be carried out, if technically feasible. When the spleen forms part of the wall of the pseudocyst, the surgeon should consider splenectomy with external drainage of the pseudocyst or possibly distal pancreatectomy including splenectomy.

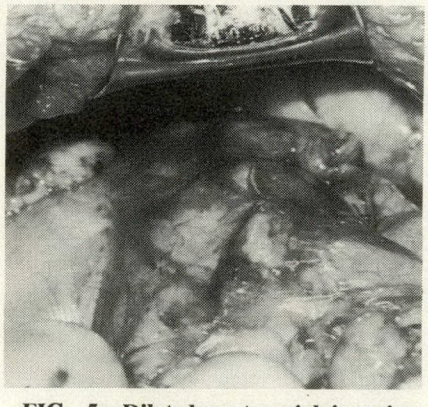

FIG. 5—Dilated gastroepiploic veins coursing through gastrocolic ligament.

Management of peripancreatic abscess producing splenic vein occlusion may present difficult technical problems. With disease limited to the body or tail of the gland, distal pancreatectomy with splenectomy would be the procedure of choice. However, this procedure may be compromised by the severity of subacute and chronic inflammation. Resection under such circumstances may contribute to postoperative complications such as hemorrhage, pancreatic fistula or subphrenic abscess. Débridement and external drainage of the necrotic, peripancreatic tissue combined with splenectomy will control bleeding from the gastric varices and will limit septic complications of the pancreatic abscess. If the patient is not actively bleeding, splenectomy should be reserved for a second operation following control of peripancreatic sepsis.

Patients with chronic pancreatitis and gastric varices present a complex problem, combining acute gastrointestinal blood loss and a chronic pain syndrome. If bleeding is the sole problem, splenectomy alone should suffice. If the pain of chronic pancreatitis is associated with bleeding from the varices, then resection including splenectomy should be considered, based on the extent of disease affecting the gland. Obliteration of surgical planes posterior to the gland occurs in such cases and vascular anatomy can be substantially altered. Care should be taken to prevent injury to the celiac axis and hepatic artery.

Carcinoma of the pancreas producing splenic vein occlusion and bleeding gastric varices presents an almost hopeless situation because the carcinoma is invariably unresectable. The surgeon must consider the course of the malignant disease and the severity of the bleeding when decid-

ing whether splenectomy is indicated. In these patients there may be a place for simple ligation of the splenic artery or percutaneous embolization of the artery. The risks of splenic infarction with subsequent sepsis or revascularization with rebleeding have not been reported.

Conclusions

Occlusion of the splenic vein with gastric varices should be considered in any patient with gastrointestinal hemorrhage and proven pancreatic disease. If angiograms confirm the diagnosis, splenectomy will control bleeding. Most patients will also require a selective surgical procedure to treat the associated pancreatic disease.

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The Conduct of Cholecystectomy: Incision, Drainage, Bacteriology and Postoperative Complications

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The benefits of some ancillary techniques of cholecystectomy are exaggerated by retrospective study of selected patients. Therefore, the authors performed a prospective, randomized study of 100 consecutive patients who underwent simple elective cholecystectomy for chronic cholecystitis and cholelithiasis. No patient was excluded because of incomplete hemostasis or fear of bile leakage. The frequency of pulmonary complications and wound infections was independent of the type of incision — vertical or subcostal. Peritoneal drainage was found to be unnecessary. Short-term drainage may increase the frequency of postoperative fever, but did not increase pulmonary complications or wound infections. In these patients, intra-abdominal sepsis is rare; wound infections were uncommon and the gallbladder bile was usually sterile and not the cause of postoperative infection.

Les études rétrospectives chez des patients choisis exagèrent les avantages de certaines techniques auxiliaires de la cholécystectomie. Les auteurs ont réalisé une étude prospective et randomisée de 100 patients consécutifs qui ont subi une cholécystectomie simple non urgente pour cholécystite chronique et cholélithiase. Aucun patient ne fut exclu en raison d'une hémostase incomplète ou d'une fuite de bile appréhendée. La fréquence des complications pulmonaires et des infections de plaie n'était pas influencée par le type d'incision (verticale ou sous-costale). Le drainage péritonéal fut jugé inutile. Le drainage de courte durée peut augmenter la fréquence de la fièvre postopératoire mais il n'a pas augmenté le nombre des complications pulmonaires ou des infections de plaies. Chez ces patients, les infections intraabdominales sont rares; les infections de plaies furent peu fréquentes et la bile dans la vésicule biliaire était habituellement stérile et elle ne causa pas d'infections postopératoires.

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Although cholecystectomy is the second commonest major operation performed today,[1] the ideal ancillary procedures have not been settled. In 1882, Carl Langenbuch devised the technique of cholecystectomy by careful cadaver dissection and performed the first such procedure on a living person.[2] He opened the abdomen

through a vertical lateral rectus incision with a subcostal T-extension; after removing the gallbladder, he inserted a peritoneal drain before closing the abdomen. Thirty-one years later, a group of German surgeons introduced the "ideal" cholecystectomy;[3] it is usually performed through a subcostal incision, and includes "subserous" cholecystectomy[4] and abdominal closure without peritoneal drainage.[5,6] Sporadic reports since then have claimed for that technique greater patient comfort, a shorter operating time and hospital stay,[7-9] fewer wound infections and respiratory complications,[10] and even lower operative mortality.[11] However, since few careful prospective studies have been done, we designed a study to examine the effect of the type of incision and the insertion or omission of peritoneal drains on the frequency of postoperative complications after simple elective cholecystectomy and to review the nature of infectious complications after routine cholecystectomy.

Patients and Methods

Starting in December 1979, 100 consecutive patients underwent elective cholecystectomy for chronic cholecystitis. Patients with acute cholecystitis or jaundice and those requiring exploration of the common bile duct or an additional procedure on the stomach or colon were not included. However, no other exclusions, such as fear of bile leakage or incomplete hemostasis, were permitted. The patients were randomly assigned by sealed envelope technique to one of three groups: no drain, Penrose drain, or closed suction (Hemovac drain). A subcostal or a vertical abdominal incision was employed depending on the usual practice of the surgeon. Forty-nine procedures were performed by four surgeons through subcostal incisions and 51 operations by three surgeons through vertical abdominal incisions.

The age, sex, weight and height of each patient and the duration of operation were noted. Ideal weight for height and body build values were obtained from standard tables.[12] A risk index of wound infection was calculated as suggested by Davidson and associates.[13] This index relates the risk of postoperative wound infection to the age of the patient, duration of operation, wound class and presence or absence of organisms on culture of the subcutaneous fat at the end of operation. The total blood lymphocyte count was obtained preoperatively by multiplying the total leukocyte count by the percentage of lymphocytes seen on differential smears. Determinations of the serum bilirubin, alkaline phosphatase, glutamic pyruvic transaminase (SGPT) and lactic dehydrogenase (LDH) were made preoperatively and 2 and 4 days postoperatively by standard methods using the Bichromatic Autoanalyser (Abbott Laboratories, Diagnostics Division, Toronto, Ont.). The normal values for total serum bilirubin are 0.3 to 1.4 mg/dl (5.13 to 23.9 μmol/l), alkaline phosphatase 90 to 280 IU/l, SGPT 4 to 25 U/l and LDH 52 to 142 U/l.

At operation, the degree of wound contamination was noted. The cystic duct was doubly ligated by ligature and metal clip, and the serosa of the gallbladder bed in the liver was closed by continuous catgut suture. A culture was obtained from the inner surface of the gallbladder and from the subcutaneous fat of the abdominal wall before skin closure.

Patients were carefully followed postoperatively for development of wound or intra-abdominal complications and for the development of nonspecific pulmonary or urinary tract infections. A wound infection was diagnosed when pus drained from the wound and a sample of the pus was then sent for culture. Wounds that were indurated, reddened or tender were followed until the condition resolved or there was drainage. A further report was obtained 1 month after operation from the patient's physician. The volume of daily drainage from the suction drain was recorded. Drains were removed on the second postoperative day unless the volume of drainage was excessive. Oral temperatures were recorded every 6 hours and a fever index was calculated for each patient as the number of postoperative days that the temperature was higher than 37.5°C after the first 24 hours.

Statistical evaluation was carried out where indicated by Student's *t*-test and χ^2 analysis.

Results

There were no deaths in the series and no patient required reoperation. One patient, operated upon through a vertical incision and closed without peritoneal drainage, sustained intraoperative injury to the portal vein. This injury was identified and the vein repaired. Transient intra-abdominal bleeding occurred postoperatively necessitating blood transfusion before vital signs were stabilized on the day of operation.

The 49 patients who underwent operation through subcostal and 51 through vertical abdominal incisions were compared with respect to mean age, sex, preoperative blood lymphocyte count, percentage of ideal weight for height and the Davidson risk index of wound infection (Table I). The groups were evenly matched. Postoperative complications were equally frequent in patients with subcostal incisions and those with vertical incisions. The mean postoperative fever indices were 2.2 ± 0.26 and 1.98 ± 0.20 days respectively ($P > 0.1$, $t = 0.79$); seven patients had pneumonia (four with subcostal and three with vertical incisions) and wound infections developed in one patient with a subcostal incision and three with vertical abdominal incisions ($P > 0.1$, $\chi^2 = 0.96$).

Patients with and without peritoneal drainage were compared with respect to mean age, sex, preoperative blood lymphocyte counts, the percentage of ideal weight for height and the Davidson risk index of wound infection (Table II). These groups, too, were well matched. Postoperative complications between the groups are compared in Tables III and IV.

Table I—Comparison of Patients According to Type of Incision*

Factor	Type of incision: Subcostal (n = 49)	Type of incision: Vertical (n = 51)	t value†
Age, yr	51.3 ± 2.5	50.5 ± 2.2	0.22
Sex, male/female	6/43	8/43	—
No. of lymphocytes,/mm³	1715 ± 92	1782 ± 100	0.49
Weight, actual/ideal (%)	119.8 ± 3.2	140.9 ± 19.8	1.16
Risk index, %	15 ± 2.6	17 ± 2.44	0.53

*Mean values ± standard error of the mean.
†Not significant.

Postoperative fever occurred more frequently in patients with peritoneal drainage, but no significant difference in fever index was observed between patients with (1.8 ± 0.23) and those without (2.3 ± 0.23) a history of chronic obstructive pulmonary disease and cigarette smoking; postoperative pneumonia was equally common in patients with drainage and those without. Wound infections were slightly more frequent in patients with drainage than in those without, but the difference was not statistically significant. The frequency and level of increase of postoperative serum bilirubin between patients with and those without peritoneal drainage, and between patients with Penrose and those with Hemovac drains did not show any significant differences.

The striking results of cultures obtained at operation from the gallbladder and subcutaneous fat of the wound and from the wounds of the four patients who had wound infections were the low frequency of positive cultures of the gallbladder bile and the lack of correlation of the gallbladder organisms with those found in the subcutaneous fat and in infected wounds (Fig. 1). No patient was given antibiotics prophylactically. The Davidson risk index of wound infection was significantly higher ($P < 0.02$) in the four patients who had wound infections than in those who did not, but the factors constituting the index did not show similar differences (Fig. 2).

Table II—Comparison of Patients According to Use of Drainage

Factor	Drain (n = 50)	No drain (n = 50)	P value (t value)
Age, yr	49.8 ± 2.2	51.5 ± 2.7	NS (0.48)
Sex, male/female	6/44	8/42	NS
No. of lymphocytes,/mm³	1764 ± 108	1744 ± 84	NS (0.15)
Weight, actual/ideal (%)	123.3 ± 3.8	121.6 ± 3.3	NS (0.45)
Risk index, %	13.3 ± 2.2	17.7 ± 2.5	< 0.1 (1.3)

NS = not significant.

Table III—Drain vs. No Drain Postoperative Complications

Factor	Drain (n = 50)	No drain (n = 50)	P value
Postoperative fever index, d >38.5°C	2.5 ± 0.2	1.7 ± 0.2	<0.01 (t = 2.28)
Wound infection, no.	3 (2 Hemovac, 1 Penrose)	1	>0.3 (χ^2 = 1.04)
Postoperative pneumonia, no.	4	3	NS

Table IV—Postoperative Bilirubinemia vs. Drainage

Factors associated with bilirubinemia	Drain (n = 50)	No drain (n = 50)	P value (t value)
Abnormal postoperative serum bilirubin, no.	9	11	NS
Preoperative serum bilirubin, mg/dl	0.56 ± 0.04	0.45 ± 0.03	<0.05 (1.94)
Postoperative serum bilirubin, mg/dl	0.92 ± 0.09	0.87 ± 0.08	NS (0.56)
Postoperative increase bilirubin, mg/dl	0.36 ± 0.08	0.42 ± 0.08	NS (0.50)
With Hemovac drain, mg/dl (n = 25)	0.24 ± 0.05		
With Penrose drain, mg/dl (n = 25)	0.42 ± 0.15		NS (0.30)
Corresponding hepatic bile, ml	94	110	
Corresponding peritoneal fluid, ml	940	1100	
Hemovac drainage, ml	59.2 ± 12.3 (median 30, range 0–450)		
Drains retained more than 2 d	5 (2 Hemovac, 3 Penrose)		

Discussion

Although much has been written on the effect of ancillary techniques of cholecystectomy on postoperative complications, few prospective studies have been done. Our study examines the problem in a prospective, controlled manner. The allocation of incisions was not truly random, but the matching of patients was.

Which Incision is Preferable?

Some surgeons[14] report fewer complications and others[15] more pulmonary complications with subcostal incisions than with vertical abdominal incisions. In keeping with the spirometric studies of Williams and Brenowitz[16] we found no difference in the frequency of postoperative pneumonia or in the fever index between the two incisions. Haight and Ransom[17] have reported similar results.

Is It Safe to Omit Peritoneal Drainage?

Our results indicate that peritoneal drains may be omitted safely in cases of uncomplicated elective cholecystectomy. There were no op-

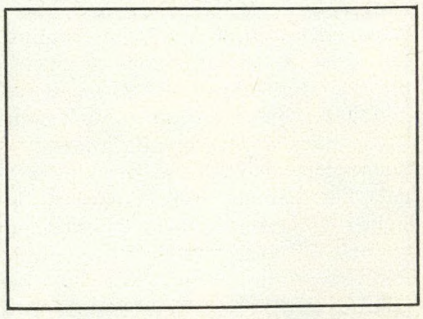

FIG. 1—Bacteriology: gallbladder, subcutaneous fat, infected wounds.

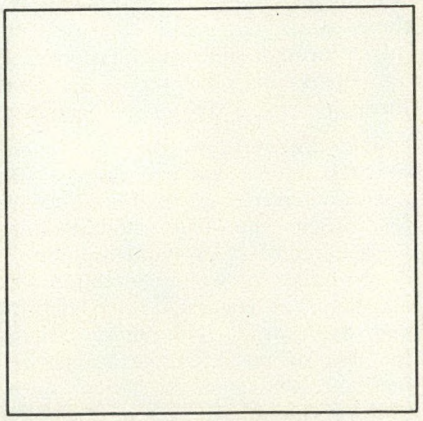

FIG. 2—Risk index: factors in wound infections.

erative deaths and no patients required reoperation. Glenn[18] has stated that subhepatic accumulations occur after the majority of cholecystectomies and are of clinical importance in 7% to 10% of cases. We should therefore have expected at least three instances of such accumulations in 50 cholecystectomies that were not drained. None was suspected or diagnosed.

Does Any Less Obvious Morbidity Result from Omitting a Drain?

Edlund and associates[19] found a transient abnormal increase in serum bilirubin levels after undrained cholecystectomy and attributed this to resorption of bile that would otherwise have been discharged by the drain. Our results show that an abnormal postoperative bilirubin level was equally common in patients with and without drains. The extent of the postoperative increase in serum bilirubin level was also similar in the drained and undrained groups. Based on the average concentration of bilirubin in hepatic bile and peritoneal drainage fluids,[20] large amounts of bile would have to be lost and fluid resorbed to produce this postoperative increase in serum bilirubin level. It is therefore unlikely that this mechanism alone explains the increase. Nor is pericholangitis[21] a satisfactory explanation, for no significant changes occurred in the concentrations of SGPT, LDH and serum alkaline phosphatase.

Polk[22] has recommended sump suction drainage of the subhepatic space after cholecystectomy. Sump drains without bacterial filters, although more efficient than Penrose drains,[23] entrain more organisms from the patient's skin, dressings and environment.[24] If drainage is required, closed suction seems preferable, but, surprisingly, van der Linden and associates[25] recently found that closed suction drains impair rather than enhance intraperitoneal drainage. We found no difference between our patients who had Penrose drains and those who had Hemovac drains. Indeed, since patients without drains did as well as those with drains, we would expect little difference between the two types of drains.

Are There Any Advantages to Omitting the Drain?

We found increased frequency of postoperative fever in patients with peritoneal drains. Maull and associates[26] reported similar results, but two other prospective studies[19,27] found no increase.

No significant increase in wound infection occurred in patients with peritoneal drains. In contrast to the findings in numerous retrospective studies, no prospective controlled clinical trial has demonstrated such an association.

What Is the Frequency and Source of Postoperative Infection in Uncomplicated Elective Cholecystectomy?

In our study there were four wound infections, all due to *Staphylococcus pyogenes;* there was no intra-abdominal sepsis. Only 11% of gallbladder cultures grew organisms and none of them *S. pyogenes.* The light growth of *Staphylococcus epidermidis* from the subcutaneous fat of 20% of operative wounds was probably insignificant. The lack of correlation between the results of cultures of the gallbladder, subcutaneous fat and infected wounds suggests that in the truly "low-risk" patient[28] with uncomplicated cholelithiasis who undergoes elective operation, culture of the gallbladder bile and subcutaneous fat is not useful. Since cultures obtained from the nasal mucosa and skin of two of these four patients were negative for *S. pyogenes,* it is unlikely that they were carriers of this organism. These findings are in sharp contrast to what occurs in "high-risk" patients.[28] There, the source of postoperative infection is endogenous, usually from the streptococcal or coliform bacteria in the bile, and postoperative intra-abdominal infection occurs more frequently. These differences have not been adequately emphasized, but have been noted previously by Stone and associates.[29]

Conclusions

In simple elective cholecystectomy for chronic cholecystitis and cholelithiasis the choice of abdominal incision, vertical or subcostal, does not influence the development of postoperative pulmonary or infectious complications. Peritoneal drainage may be omitted safely without increasing morbidity. Postoperative fever occurs less often when no drainage is used but the frequency of respiratory complications and wound infections is not altered. Gallbladder bile is usually sterile and is not the source of wound infection.

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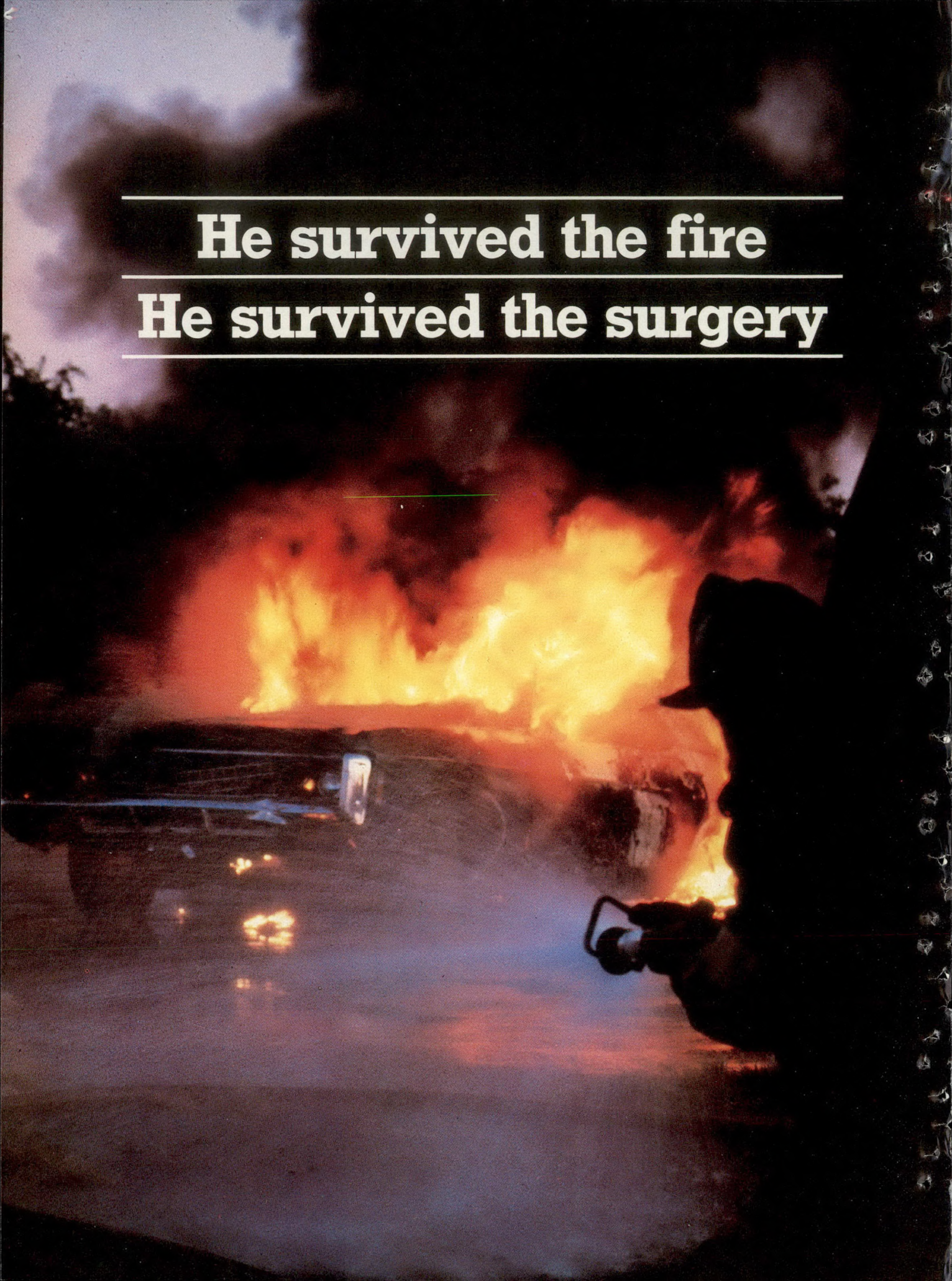
He survived the fire
He survived the surgery

Management of Duodenal Diverticula

C.H. SCUDAMORE, MD, FRCS[C], R.C. HARRISON, MD, FACS, FRCS[C] AND T.T. WHITE, MD, FACS

Duodenal diverticula are not uncommon. Uncomplicated diverticula require no treatment. In the period 1970 to 1980, 104 patients with symptoms presented at the Vancouver General Hospital and the Swedish Hospital in Seattle. Of these, 26% presented with pain and 24% with anemia. Malabsorption and benign tumours were noted infrequently. Fifteen patients were treated surgically for pain, perforation, hemorrhage, tumour, blind loop syndrome and obstruction of the duodenum, biliary tract or pancreatic duct. Two patients died.

The second part of the duodenum was involved in 82% and the third part in 10%. Anatomical variations are common in the area of the ampulla; these should be anticipated before operation. Surgical procedures in the area may be technically demanding and associated with high mortality and morbidity. The choice of procedure depends on the urgency and nature of the complication of the diverticulum. Endoscopic retrograde cholangiopancreatography can play a role in evaluating the possible significance of the diverticulum and the anatomy of the area. In some cases endoscopic sphincterotomy may be possible. In some situations diverticulectomy is contraindicated.

Il n'est pas rare de rencontrer des diverticules duodénaux. Lorsqu'il n'y a pas de complication, ils ne nécessitent pas de traitement. Durant la période allant de 1970 à 1980, 104 de ces patients souffrant de symptômes ont été reçus au Vancouver General Hospital et au Swedish Hospital de Seattle. Parmi ceux-ci, 26% manifestaient de la douleur et 24% étaient anémiques. De la diarrhée et des tumeurs bénignes étaient rarement observées. Une intervention chirurgicale fut pratiquée chez 15 patients présentant de la douleur, une perforation, une hémorrhagie, une tumeur, un syndrome de l'anse borgne ou une obstruction du duodénum, des voies biliaires ou du canal pancréatique. Deux patients sont décédés.

Il y avait atteinte de la seconde partie du duodénum dans 82% des cas et de la troisième partie dans 10%. Les variations anatomiques de la région ampullaire sont fréquentes et elles devraient être anticipées avant l'opération. Les interventions chirurgicales pratiquées dans cette zone peuvent être techniquement difficiles et elles sont associées à un fort taux de mortalité et de morbidité. Le choix du type d'intervention dépend de l'urgence et de la nature des complications reliées au diverticule. La cholangiopancréatographie endoscopique rétrograde peut jouer un rôle quand il s'agit d'évaluer l'importance du diverticulum et l'anatomie de la région. Dans quelques cas, la sphinctérotomie est possible. Dans certaines situations, la diverticulectomie est contre-indiquée.

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Duodenal diverticula are found in 2% to 5% of all individuals who undergo upper gastrointestinal contrast studies.[1,2] The duodenum is the second most frequent location for gastrointestinal diverticula after the colon.[3] Symptoms attributed to duodenal diverticula are nonspecific and seldom severe, and it is difficult to identify the diverticulum as the source. The usual presentation of acute complicated duodenal diverticular disease is that of a surgical emergency in an elderly patient, with either an acute abdomen or an active gastrointestinal hemorrhage. The diagnosis is seldom considered preoperatively. The condition is difficult to manage surgically and is associated with appreciable morbidity and mortality.[2,4-6]

The majority of reports dealing with complicated duodenal diverticula were published before the era of modern fiberoptic endoscopy. The investigation and treatment of duodenal diverticular disease has been altered by the use of the side viewing duodenoscope and endoscopic retrograde cholangiopancreatography.[7,8] It now may be possible to investigate and treat some nonemergent complications of duodenal diverticula entirely endoscopically.[7]

Emergent complications must be recognized early and dealt with by a surgeon who is familiar with the complex anatomical relations of the diverticulum that may be obscured by inflammation or bleeding.

Patients and Method

The records of patients seen at the Vancouver General Hospital for complications arising from duodenal diverticular disease were reviewed for the 10-year period 1970 to 1980. Those who had treatment or surgical procedures for coexisting conditions and were found incidentally to have duodenal diverticular disease were excluded. Two cases from the Swedish Hospital in Seattle, Washington were also included. There were 104 patients in all.

Case Reports

The following four cases from the Vancouver General Hospital are presented to illustrate the problems of the behaviour, diagnosis and treatment of complicated duodenal diverticula.

Case 1

An 85-year-old man complained of generalized abdominal pain, which had started suddenly in the right upper quadrant and flank 4 hours earlier. Examination revealed generalized abdominal tenderness, bowel sounds were absent and the abdominal wall was rigid. His temperature was 37.4°C, blood pressure 140/90 mm Hg, pulse rate 90 beats/min and irregular. The leukocyte count was 8.9×10^9/l with 39% polymorphonuclear cells and 58% lymphocytes, the total serum bilirubin level was 1.4 mg/dl (23.9 μmol/l), serum alkaline phosphatase was 122 IU/l (normal to 125 IU/l), serum amylase was 140 IU/l, (normal to 80 IU/l). The prothrombin time was normal. Roentgenogram of the abdomen showed multiple air–fluid levels throughout the large and small bowel. There was air in the retroperitoneal space.

A diagnosis of a ruptured viscus was made; supportive measures were instituted and antibiotics given. At laparotomy the common bile duct was found

to be dilated and a greenish staining was noted in the periduodenal tissues. The duodenum was kocherized. A perforation was noted in a duodenal diverticulum that measured 3 cm. The common bile duct was explored, stones were removed from it and a probe was passed through into the duodenum. The duodenum was opened and the gallstones were removed from the diverticulum. The diverticulum was totally resected and the duodenum closed primarily. A T-tube was placed in the common duct. The patient had previously had a cholecystectomy.

The postoperative course was complicated by mild pancreatitis. A T-tube cholangiogram obtained 7 days after operation appeared normal.

Case 2

An 87-year-old Caucasian woman with no history of abdominal complaints had acute epigastric pain beginning several hours before admission. The pain was steady, generalized and radiated to the back. She had vomited bile-stained fluid twice. She had had a normal bowel movement that morning. The blood pressure was 180/100 mm Hg, the pulse rate was 100 beats/min and regular and the temperature was 38°C. Bowel sounds were absent and her abdomen was distended and rigid. No abnormality was found on rectal examination. Abnormal laboratory results included a leukocyte count of 8.7×10^9/l, 25% polymorphonuclear cells, 63% lymphocytes and occasional metamyelocytes. The blood glucose level was 249 mg/dl (13.7 mmol/l). (The serum bilirubin, creatine phosphokinase, glutamic oxaloacetic transaminase and lactic dehydrogenase levels were all normal.) The serum albumin level was 2.7 g/dl, calcium 9.8 mg/dl (2.43 μmol/l), phosphate 2.6 mg/dl (0.85 mmol/l) and amylase 471 IU/l. The urine amylase level was 1624 IU/l. The prothrombin time was 70% of normal. Abdominal roentgenograms did not reveal free air or retroperitoneal gas shadows. An abdominal tap produced cloudy fluid in copious quantities. On Gram-staining the fluid was found to contain *Clostridium* sp. and large numbers of leukocytes and erythrocytes and had an amylase concentration in excess of 5000 IU/l.

A diagnosis of perforated or infarcted viscus was made. Supportive measures were instituted and antibiotics given. At laparotomy the periduodenal tissues were bile-stained and a perforated diverticulum of the second part of the duodenum was found. The biliary system was normal to palpation.

The diverticulum was patched with omentum, a large T-tube was brought out through the lateral duodenal wall as a controlled fistula, and a gastrostomy and feeding jejunostomy were fashioned. A Salem sump was placed in the subhepatic space near the diverticulum.

Postoperatively she had prolonged bile-stained drainage from the controlled duodenal fistula site. This closed after approximately 1 month.

Case 3

An 84-year-old man complained of epigastric pain and weakness. His appetite was diminished and he had noted a weight loss of 6 kg over the previous 4 months. His pain was intermittent, worse between meals, most severe in the epigastrium and radiated to the back. He had noted black stools on and off for a few weeks. He had had his gallbladder removed 15 years before. He was a thin, pale man in no acute distress. His blood pressure was 160/90 mm Hg supine and 140/70 mm Hg sitting, accompanied by faintness and a pulse rate of 110 beats/min. Tenderness but no mass was noted in the epigastrium. Abnormal findings on laboratory studies were a hypochromic microcytic anemia, blood urea nitrogen value of 50 mg/dl (18 urea, mmol/l) and serum creatinine value of 1.2 mg/dl (106 μmol/l). The serum amylase level was normal and the prothrombin time was 90% of normal. The aspirate from the nasogastric tube was bile-stained. Upper gastrointestinal roentgenography after barium meal demonstrated a large duodenal diverticulum. Two days after admission the patient's blood pressure dropped to 90/60 mm Hg and his heart rate climbed to 140 beats/min. Bright red blood appeared in a nasogastric aspirate. At operation a bleeding duodenal diverticulum was found with a large enterolith in its lumen. The remaining duodenum was normal. The common duct was probed and was normal. The stone was removed and the ulcer in the diverticulum, which was actually bleeding, was oversewn. The patient did well postoperatively.

Case 4

A 74-year-old woman presented with a long history of intermittent epigastric and right upper quadrant pain, usually postprandially. On occasion she would vomit bile-stained material which frequently brought relief of her symptoms. Antacids were of no help. She was slightly obese but in no acute distress. The findings on abdominal examination were normal. Laboratory values included a hemoglobin level of 13.7 g/dl, leukocyte count of 6.7×10^9/l with 70% polymorphonuclear cells and 12% lymphocytes, serum alkaline phosphatase 120 IU/l, prothrombin time 85% of normal, total serum bilirubin of 1.4 mg/dl (23.9 μmol/l) and direct value of 0.6 mg/dl (10.3 μmol/l). The serum glutamic oxaloacetic transaminase, lactic dehydrogenase and amylase levels were normal.

Upper gastrointestinal roentgenograms after barium meal showed a diverticulum in the medial portion of the second part of the duodenum and the oral cholecystogram showed nonfunctioning gallbladder after a double dose of contrast material. Endoscopic retrograde cholangiopancreatography demonstrated the duodenal diverticulum with compression of the bile duct. Stones were demonstrated in the gallbladder and common duct. An endoscopic sphincterotomy was done and stone extraction was carried out with a balloon catheter (Fig. 1). The patient remains free of symptoms and has not had to have her gallbladder removed.

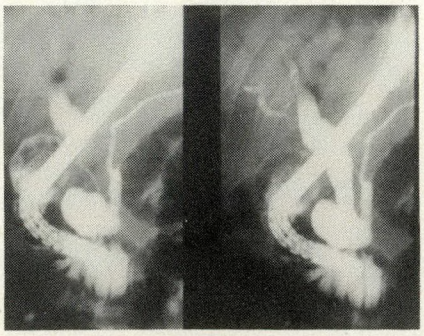

Fig. 1a Fig. 1b

FIG. 1—(a) Duodenal diverticulum obstructing common bile duct. (b) Following endoscopic sphincterotomy and stone removal.

Results

Our experience was essentially the same as that of others. The male to female ratio of the 104 patients was 2:3. The average age was 71 years (range from 27 to 96 years). Multiple duodenal diverticula were found in 22% of the patients. The first part of the duodenum was involved in 6%, the second in 82%, the third in 10% and the fourth in 2%; laterally projecting diverticula were found in 2%. Active associated diseases that could be responsible for presenting symptoms were frequently found (Table I). The presenting signs and symptoms were pain in 26% (with no associated disease in 4%), hemorrhage in 10% (with no other source in 4%), anemia in 24% (with no other source in 6%) and diarrhea in 4%. Once the associated diseases were

Table I—Associated Disease

Disease	% of patients
Biliary tract	24
Hiatus hernia	20
Peptic ulcer disease	20
Myocardial disease	18
Diverticula elsewhere in gastrointestinal tract	16
Gastric carcinoma	3
Pancreatitis	2
Aortic aneurysm	2

ruled out as a cause for their complaints, only about 25% of these patients still had unexplained symptoms.

Only 15 of the 104 patients underwent operation and for varying indications (Table II). Two patients died in hospital, one of sepsis and pulmonary embolism and the other of sepsis and pancreatitis. The hospital stay was prolonged in two patients because of high enterocutaneous fistulas.

Discussion

Diverticular Types

Duodenal diverticula may be true (full thickness) or false (mucosal). True diverticula may be congenital or be acquired traction diverticula secondary to chronic peptic ulcer disease. False diverticula usually occur on the medial or pancreatic aspect of the second part of the duodenum[8] (Table III). Much less frequently they are found in the third or fourth part or at multiple sites. The usual position for second-part diverticula is intimately related to the entrance of the common bile duct into the duodenum. Most open within 2.5 cm of this point, hence the name "pervaterian".[1,9] These are the diverticula associated with most complications and that cause the most problems for both patients and surgeons.[2,6] The commonly accepted theory explaining this position is the presence of a congenital weakness in the duodenal wall allowing the entry of the pancreatic and common bile ducts into the duodenal lumen. Entrance of larger penetrating blood vessels may provide a similar weakness.[10]

Table II—Indications for Operation in 15 Patients

Indication	No. of patients
Pain	5
Hemorrhage	3
Localized perforation	3
Free perforation	1
Enterolith	1
Malabsorption	2

Table III—Location of False Diverticula

Location	%
Periampullary	85
Lateral (torsion commoner)	8
Posterior	4
Multiple	10

Complications and Treatment

There are two situations that give rise to most diverticular complications (Table IV): stones passing down the common duct may become trapped in the lower end of the common bile duct or in the diverticulum itself and enteroliths can form in the amuscular diverticular pouch from food and stasis.[11,12] Obstructive complications include jaundice, pancreatitis and, rarely, duodenal obstruction. Stones in the diverticular lumen can erode through the thin wall causing bleeding or perforation, but they need not be present for perforation to occur.[2,5,13] Large diverticula have a tenuous blood supply, and according to Laplace's law the diverticulum is the vulnerable point if intraluminal duodenal pressures rise.

Perforation of the diverticulum usually presents as sudden pain in the right upper quadrant or epigastrium radiating to the back. This pain progresses rapidly and there is generalized abdominal rigidity. Accompanying these findings are those of toxicity.[10,13] The important differential diagnoses include biliary tract disease, pancreatitis, a perforated viscus and mesenteric infarction.[5,14] The patient is obviously very ill and often profoundly hypovolemic. Plain radiologic studies may demonstrate retroperitoneal gas shadows but seldom free air.[15] Contrast studies demonstrate the presence of the diverticulum but may fail to show the perforation. The most reliable operative clue is that of a bile-stained periduodenal phlegmon.[2] The perforation can be demonstrated after careful kocherization of the duodenum. Diverticulectomy with primary closure of the duodenal wall, followed by periduodenal drainage, has lowered the morbidity and mortality rates compared with drainage alone because the diverticulum may become necrotic or the source of a high enteric fistula.[2] Before wide kocherization or diverticulectomy is performed, care must be taken to identify the pancreatic and common bile ducts. Rarely, they may enter the diverticulum itself and thus would become disconnected at the time of resection. This would be disastrous.[16] Lateral drainage of the duodenum itself as a controlled fistula is not usually necessary; it resulted in a duodenal fistula of long duration in our case 2.

Table IV—Complications

Stomal obstruction
Duodenal obstruction
Bleeding
Perforation
Enterolith — secondary small bowel obstruction (rare)
Biliary obstruction
Pancreatitis
Malabsorption
Tumour (rare)

Elective surgical treatment of periampullary diverticula is seldom required.[17] In the past, elective diverticulectomy for nonspecific indications resulted in a mortality as high as 20% and persistent symptoms in as many as 50%.[10] For patients with serious unexplained symptoms, such as those from a dilated common duct or recurrent pancreatitis, we advocate endoscopic retrograde cholangiopancreatography to delineate the ductal anatomy and to detect the presence of stones in the ducts or diverticulum once the usual contrast studies of the biliary tree and upper gastrointestinal tract have been performed.

It is important to know that the majority of pancreatic and common bile ducts come together and gain entrance into the duodenum at the superior margin of the diverticulum. Although conventional contrast studies may give the impression that the ducts are entering the diverticular dome, this is exceptional but important.[1] Sphincterotomy will permit drainage of the common duct which is compressed by the diverticulum.[8] Sphincterotomy and endoscopic retrograde cholangiopancreatography should not be done when there is poor patient cooperation, when there are medical contraindications, when the duct enters the dome and when perforation is suspected. Acute pancreatitis is a relative contraindication.[8,17,18]

If the ampulla enters the dome of the diverticulum when there is common duct obstruction, diverticulectomy is contraindicated and choledochoduodenostomy is much preferred. Reimplantation of the ducts carries a mortality rate of more than 30%.[16] There are three rare situations when the common bile duct, pancreatic duct or ampulla must be divided. The first is when there is a tumour in the diverticulum whose prognosis is worse than the expected mortality of duct repair. The others are when life-threatening hemorrhage and perforation are present and they cannot be managed by other means. If disconnection is essential or if the dam-

age is done inadvertently the common duct and pancreatic duct should be reconstituted by separate anastomoses into a Roux-en-Y loop.

Conclusions

Elective treatment of uncomplicated duodenal diverticula is not usually justified. Perforation is best managed by resection and primary closure. A diverticulum, with or without common duct stones, may play a role in obstructing the common duct.

At operation a bile-stained periduodenal phlegmon is the diagnostic clue. The anatomy of the common duct, major pancreatic duct and the diverticulum may be difficult to determine. If the ducts enter the diverticulum itself choledochoduodenostomy should be done and the diverticulum not be disturbed.

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Resection and Primary Anastomosis for Diverticulitis with Perforation and Peritonitis

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Primary resection is the treatment of choice for diverticulitis of the colon with perforation and generalized peritonitis. Although there has been controversy concerning the management of the bowel ends after resection, for the last 20 years immediate anastomosis has been gaining increasing support.

Between 1970 and 1981, at the Hôpital du Sacré-Coeur in Montreal, 15 patients having diverticulitis with perforation and diffuse spreading peritonitis who fulfilled specific criteria were treated by primary resection of the perforated segment of bowel and immediate anastomosis. The criteria for operation were: (a) the bowel must not be distended; (b) the bowel should be empty of feces; (c) edema of bowel wall at the resection site must be minimal; (d) the distal segment of colon should be above the peritoneal reflection; (e) there should be no fecal contamination; (f) the patient's general condition should be reasonably good. If these criteria were met, the procedure was safe and gave satisfactory results. In the authors' series, postoperative hospital stay was reduced considerably (it averaged 11 days). There was one anastomotic leak and one death from acute pulmonary edema.

La résection primaire constitue le traitement de premier choix dans les cas de diverticulite du côlon avec perforation et péritonite généralisée. Bien que la conduite à tenir après la résection avec les deux bouts de l'intestin ait fait l'objet d'une controverse, l'anastomose immédiate s'est gagnée de plus en plus d'appuis au cours des 20 dernières années.

Entre 1970 et 1981, à l'Hôpital du Sacré-Coeur à Montréal, 15 patients qui souffraient de diverticulite avec perforation et péritonite diffuse envahissante, et qui rencontraient certains critères spécifiques ont été traités par résection primaire du segment intestinal perforé et anastomose immédiate. Les critères retenus pour l'opération étaient les suivants: a) absence de distension de l'intestin; b) absence de fèces dans l'intestin; c) oedème minime de la paroi intestinale au lieu de résection; d) le segment distal doit être situé au-dessus de la réflexion péritonéale; e) absence de contamination fécale; f) état du patient raisonnablement bon. Quand chacun de ces critères était rempli, l'opération se révéla sûre et donna des résultats satisfaisants. Chez la série de patients traités par les auteurs, la durée du séjour à l'hôpital fut réduite considérablement (une moyenne de 11 jours après l'opération). Il y eut une fuite d'anastomose et un décès causé par un oedème pulmonaire aigu.

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Perforation with diffuse peritonitis is a rare but serious complication of diverticular disease of the colon. Its incidence in most published reports is about 2%.[1] Although infrequent, this condition, once diagnosed, must be treated as an emergency. Hughes and associates[2] in 1963 and Hinchey and associates[3] in 1978 recognized four clinical stages of diverticular disease with perforation: stage I — pericolic abscess, confined by the mesentery of the colon; stage II — pelvic abscess, resulting from local perforation of a pericolic abscess; stage III — diffuse generalized peritonitis that usually results from the rupture of either a pericolic or pelvic abscess into the peritoneal cavity; stage IV — fecal peritonitis, usually secondary to perforation of a diverticulum, with feces escaping from the lumen of the colon into the peritoneal cavity. This report is concerned with patients belonging to stage III (those who had diverticulitis with perforation and diffuse spreading peritonitis). We intend to demonstrate the safety of resection and primary anastomosis in managing this serious complication, provided that the patients are carefully selected and certain criteria are fulfilled.

Patients and Methods

At the Hôpital du Sacré-Coeur in Montreal, we reviewed all patients with acute diverticulitis of the colon seen between Jan. 1, 1970 and Aug. 31, 1981. Fifty-one had diverticular disease with perforation, 26 with generalized peritonitis (stages III and IV) and 25 with localized peritonitis (stages I and II).

Of the 26 patients with generalized peritonitis, 15 underwent primary resection of the perforated segment of bowel with immediate anastomosis, 4 had primary resection and no anastomosis (Hartmann procedure), while 7 patients were treated by a three-stage procedure (proximal colostomy, resection, closure of colostomy).

Of the 15 patients treated by resection and primary anastomosis (Table I), 6 were men and 9 were women. The average age was 57 years (range from 33 to 77 years). In some cases, emergency laparotomy was indicated from the start, either because the patient was admitted with features of diffuse generalized peritonitis or, on rare occasions, because the flat plate of the abdomen showed a pneumoperitoneum (Fig. 1). However, most of the patients were admitted with clinical features suggestive of localized peritonitis of the left iliac fossa. They were initially managed by a medical regimen consisting of nasogastric suction, intravenous administration of fluids and antibiotics. Operation became imperative if, despite this regimen, the general condition of the patient continued to deteriorate or signs of spreading peritonitis appeared. Thus, the time of observation before operation during which the patient was kept on medical treatment varied from a few hours to a few days (maximum 10 days).

In general the leukocyte count was elevated and only a few patients had a count lower than 10×10^9/l. At laparotomy the perforated segment of the colon was resected, followed by immediate end-to-end anastomosis of the bowel, using the one-layer technique, with 3-0 Prolene sutures. Lavage of the peritoneal cavity with large amounts of isotonic saline solution was carried out before the abdomen was closed; Penrose and sump drains were inserted. We did not perform a proximal colostomy. All our patients received antibiotic therapy. We used a combination of clindamycin and gentamicin. The former is effective against anaerobic bacteria, while the latter is especially effective against gram-negative aerobes. Occasionally we added a third antibiotic, either penicillin or ampicillin, for the control of the gram-positive aerobes.

Results

The results of resection and primary anastomosis in our series were satisfactory. There was only one complication, a colocutaneous fistula, indicating a small anastomotic leak. This healed spontaneously without the need for a proximal colostomy. Wound infection was not a problem since delayed wound closure was used. There was one death (6.6%) from acute pulmonary edema, which was confirmed by autopsy. The average period of hospital stay after operation was 11 days (range from 7 to 15 days).

Table I—Diverticulitis of the Colon with Perforation in 15 Patients Treated by Primary Resection and Anastomosis

Sex	Age, yr	Leukocyte count, $\times 10^9$/l	Time before operation, d	Complication	Death	Hospital stay, d
M	47	17.7	1	Fistula	—	8
F	54	23.0	1	—	—	12
F	73	18.0	3	—	—	12
M	55	7.9	3	—	—	10
F	77	7.5	10	—	Acute pulmonary edema	15
M	56	15.5	5	—	—	14
M	69	19.2	1	—	—	9
M	55	21.7	<1	—	—	14
F	68	15.9	2	—	—	10
F	65	14.6	6	—	—	12
F	72	7.4	3	—	—	10
F	45	9.4	5	—	—	10
F	33	18.4	1	—	—	7
F	47	9.6	3	—	—	13
M	56	15.6	5	—	—	11

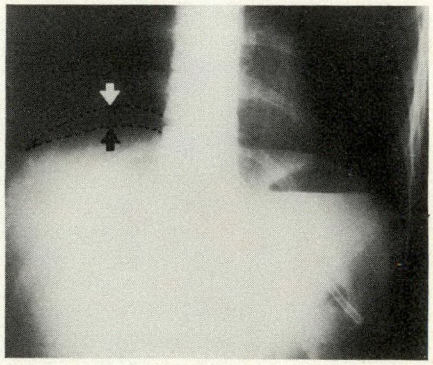

Fig. 1a

Fig. 1b

FIG. 1—(a) Upright position, (b) lateral supine position. Arrows point to free air in peritoneal cavity.

Discussion

Controversy has existed for many years concerning the best procedure for the treatment of diverticulitis with perforation. There are two types of procedure: the three-stage procedure and primary resection with or without anastomosis.

Traditional teaching favoured the three-stage procedure. In the first stage proximal colostomy is done with drainage of the perforated segment of bowel; the diseased segment is resected 2 to 6 months after the colostomy and a third operation is done to close the colostomy. Although the three-stage procedure was the standard treatment for diverticulitis with perforation and peritonitis for a long time, the mortality, about 30%,[1,4-7] is unacceptable.

We believe, as do many others,[7-11] that resection of the perforated segment of bowel should be the primary goal of therapy, since it removes the source of peritoneal contamination. Whether to do an immediate anastomosis or a colostomy with delayed anastomosis remains controversial. Because of the high risk of anastomotic leak, some[12-14] disagree with the trend to perform primary anastomosis on unprepared bowel in the presence of diffuse peritonitis. After resection of the perforated segment of colon, many surgeons prefer either to establish a proximal colostomy with a mucous fistula, or to do the Hartmann procedure — proximal colostomy and intra-abdominal closure of the rectal stump. In both cases anastomosis of the bowel ends is postponed until the conditions are more favourable.[7,10,15,16]

The results obtained in our series suggest that primary colonic resection with immediate anastomosis as advocated by Madden and Tan[1] has its place in the management of diverticulitis with perforation and diffuse peritonitis. Occasionally a proximal colostomy may be performed if the integrity of the anastomosis is in doubt. In our series no colostomies were done.

Since Gregg[17] in 1955 reported his series on diverticulitis with perforation, primary resection of the diseased segment of bowel with immediate anastomosis has gained increasing support. Although most of the published series[4,18-23] are small, the results in general are quite satisfactory. Of 73 patients reported only 6 died, a mortality of 8.2% (Table II).

Although it is tempting to perform this procedure more often, it should be emphasized that it is only suitable for patients in whom the following criteria are fulfilled: (a) the bowel should not be distended; (b) the bowel should be empty of fecal material; (c) there should be minimal edema of the bowel wall at the site of resection; (d) the distal segment of the colon should be above the peritoneal reflection; (e) there should be no fecal contamination of the peritoneal cavity and (f) the patient should be in fairly good general condition.

If all these criteria are respected, primary colonic resection with immediate anastomosis is a safe and satisfactory alternative for the management of the perforated diverticular colon with spreading peritonitis.

Table II—Reported Deaths from Primary Resection and Anastomosis in Patients with Diverticulitis of the Colon with Perforation

Authors	No. of patients	No. of deaths
Gregg, 1955[17]	7	0
Belding, 1957[18]	3	0
Large, 1964[19]	13	2
Madden, 1965[21]	7	1
Smiley, 1966[4]	3	0
Roxburgh and associates, 1968[22]	8	1
Dandekar and McCann, 1969[23]	7	1
Ryan, 1974[20]	10	0
Present series	15	1
Total	73	6 (8.2%)

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Iatrogenic Bile-Duct Strictures

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Management of iatrogenic stricture of the bile ducts is difficult and is associated with substantial morbidity and mortality. At the Toronto General Hospital, 61 patients with strictures following operative trauma underwent a total of 75 repairs. The overall success rate was 80%. End-to-end duct repair is preferred when feasible; 8 of the 61 patients were treated this way, with success in 7. In the majority of patients, however, extensive duct scarring and shortening preclude its use, necessitating Roux-en-Y hepaticojejunostomy, done in 53 cases. If the anastomosis is adequate, with good mucosal approximation, a stent is not essential and of 12 such operations, 10 were successful. Even when the mucosa-to-mucosa apposition is less than optimal, the use of a straight-tube stent or changeable U-tube stent may produce a satisfactory end result. Of 41 such operations, 31 were successful.

Since iatrogenic bile-duct strictures lead to high morbidity and frequent recurrence despite seemingly adequate repair, continued emphasis should be placed on prevention of injury, especially during routine cholecystectomy. Experience, careful dissection, knowledge of anatomic variations and good judgement concerning the performance of a cholecystostomy in difficult situations, should all reduce this complication to a minimum.

Le traitement des rétrécissements iatrogènes des voies biliaires extra-hépatiques est difficile et il est relié à une morbidité et une mortalité importantes. Soixante-et-un patients du Toronto General Hospital qui souffraient de rétrécissement secondaire à un traumatisme opératoire ont subi un total de 75 réparations. Le taux global de réussite a été de 80%. Une réparation bout-à-bout est préférée chaque fois qu'elle est possible; 8 des 61 patients furent traités de cette façon, dont 7 avec succès. Toutefois, chez la majorité des patients un tissu cicatriciel étendu ou en raccourcissement du canal en empêche le recours et nécessite une hépatico-jéjunostomie de Roux-en-Y. Cette opération fut pratiquée dans 53 cas. Quand l'anastomose est adéquate et que le rapprochement des muqueuses est bon, un gabarit n'est pas essentiel et sur 12 opérations de ce genre, 10 ont réussi. Même quand l'apposition des muqueuses n'est pas idéal, l'emploi d'un tube droit ou d'un tube en U variable comme gabarit peut donner des résultats satisfaisants. Sur 41 opérations de ce genre, 31 ont été couronnées de succès.

Comme le rétrécissement iatrogène des voies biliaires entraîne une forte morbidité et de fréquentes récidives en dépit de réparations apparamment adéquates, l'accent doit être mis de façon soutenu sur la prévention des lésions, particulièrement durant les cholécystectomies ordinaires. L'expérience, une dissection soigneuse, la connaissance des variations anatomiques et un bon jugement concernant la réalisation des cholécystostomies dans les situations difficiles devraient réduire cette complication au minimum.

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A benign, extrahepatic stricture of the bile duct presents one of the most difficult management problems in abdominal surgery. It is associated with high morbidity and appreciable mortality. The commonest cause is surgical trauma to the bile duct during operations on the biliary tract, stomach or pancreas.

Numerous techniques of reconstruction have been described. The long-term results are far from satisfactory, as many patients require multiple repairs. We review the operative management of iatrogenic biliary strictures in patients seen at the Toronto General Hospital over a 15-year period.

Patients and Methods

All patients operated upon between 1966 and 1980 for benign biliary strictures resulting from surgical trauma were reviewed. Sixty-one patients (43 men, 18 women) were studied. The mean age was 49 years (range from 17 to 79 years). Patients were followed up either by the surgeon or, for geographic reasons, by the referring doctor. The follow-up ranged from 3 to 144 months (mean 26.5 months, median 24 months).

The commonest procedure during which bile-duct injury initially occurred was cholecystectomy (58 patients). Ductal operation and gastrectomy were the causes in two and one patient respectively. In the majority of the original operative reports, the cholecystectomy was reported as "routine". The injury was noted at the time of original operation in only 15 patients (25%) and all underwent immediate repair; 14 were operated on at another institution and were later referred to the Toronto General Hospital for further assessment and management. One primary repair at the time of injury was performed at this institution.

The sites of bile-duct injury are depicted diagrammatically in Fig. 1, which shows their relative frequency in the intrahepatic, common hepatic and common bile ducts and at the bifurcation.

The 61 patients underwent 103 operative reconstructions, 75 of which were performed at the Toronto General Hospital. Twenty-eight (75%) of the 37 patients admitted after the original injury were seen within the first 10 days, while the rest presented

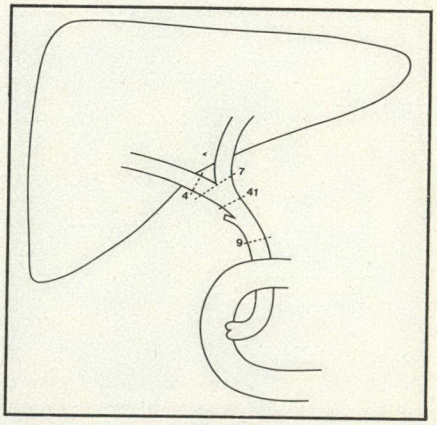

FIG. 1—Location of injury: common bile duct (9), common hepatic duct (41), bifurcation (7) and right hepatic duct (4).

from 1 month to 20 years after injury (mean 6 years). Jaundice (71%) and biliary fistula (50%) were the most common early signs of duct injury.

Patients whose first repair failed usually presented later, at a mean of 2 years after the previous reconstruction. Jaundice (84%) and cholangitis (67%) were found most commonly in this group.

Investigations

The investigative techniques used to diagnose the obstruction changed over the course of the review. At present, patients with a suspected extrahepatic biliary obstruction initially undergo ultrasonography to check for dilated extra- and intrahepatic ducts (Fig. 2). If a bile fistula is present, a sinogram is carried out. If there is no bile fistula, percutaneous transhepatic cholangiography is performed, accurately demonstrating the site of obstruction and the anatomy of the upper biliary tree (Fig. 3). A catheter may be left in place after percutaneous transhepatic cholangiography to decompress the proximal ducts temporarily in preparation for operation, and is sometimes necessary for drainage of an infected biliary tree. Endoscopic retrograde cholangiopancreatography can be useful in determining the distal limit of the obstruction (Fig. 4), as the stricture is often long. It is also valuable in early duct injuries when dilatation of the intrahepatic ducts has not yet occurred, making percutaneous transhepatic cholangiography difficult to perform.

These techniques have replaced laparotomy and operative cholangiography for diagnosis.

Reconstructive Techniques

Table I lists the types of repair used in the 75 reconstructive procedures performed at this institution. Axial (end-to-end) repair was favoured when early reconstruction was carried out using healthy ducts with no dilatation or loss of tissue. It was possible in only nine cases.

Choledochojejunostomy or hepaticojejunostomy (listed as hepaticojejunostomy in Table I) using a Roux-en-Y loop of jejunum was used in 53 cases; stents were placed in 41. Transhepatic U-tube changeable stents were used in 17 instances and straight tubes in 24 (Table II). In three repairs with a straight tube, the mucosal graft technique, to be described in more detail, was used. The U-tube stents were left in, with replacement, for a mean period of 9 months. The straight-tube stents were left in place for a mean period of 5 months. Simple dilatation with stenting and choledochoduodenostomy were employed in 3 and 10 cases respectively.

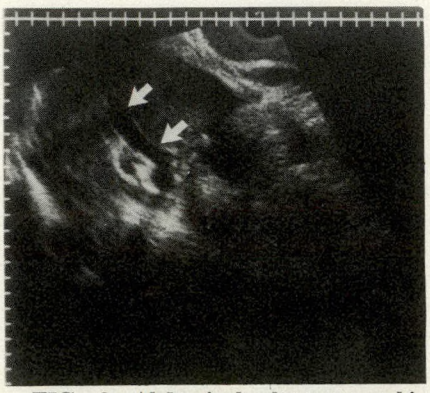

FIG. 2—Abdominal ultrasonographic findings in 67-year-old woman with obstructive jaundice, 5 months after cholecystectomy for cholelithiasis. Arrows indicate dilated intrahepatic bile ducts.

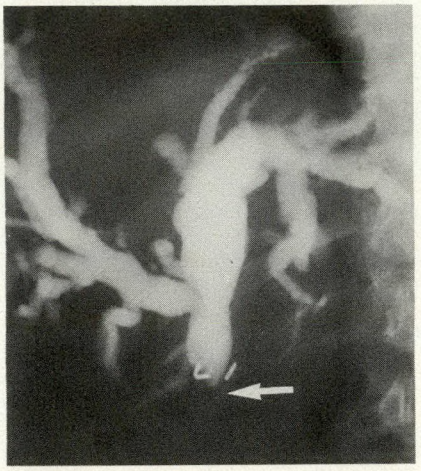

FIG. 3—Percutaneous transhepatic cholangiogram from same patient as in Fig. 2. Dilated intrahepatic ducts narrow to obstruction of common hepatic duct at site of surgical clips (arrow).

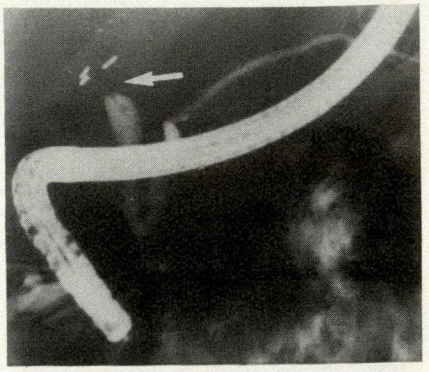

FIG. 4—Endoscopic retrograde cholangiopancreatogram from same patient shows obstruction of common bile duct 1 cm distal to surgical clips (arrow).

Hepaticojejunostomy

Unstented.—At laparotomy, proximal healthy duct is identified, cleared and transected. Proximal duct is then anastomosed, end-to-side, to a Roux-en-Y jejunal loop, using one layer of interrupted, fine, catgut sutures. We prefer catgut to silk because silk may act as a nidus for the formation of common bile-duct stones. If precise mucosa-to-mucosa apposition is accomplished using healthy duct, a stent is not necessary. However, if severe scarring of proximal duct prevents such precise anastomosis, a U-tube changeable stent or a straight rubber transhepatic stent is used.

U-tube technique (Fig. 5).—After the intrahepatic ducts have been dissected as far as safety allows, a mal-

Table I—Results of 75 Bile-Duct Reconstructions

Operative repair	No.	Successful outcome, no. (%)
Dilatation and stent	3	2 (67)
Axial repair	9	8 (89)
Choledochoduodenostomy	10	9 (90)
Hepaticojejunostomy	53	41 (77)
Total	75	60 (80)

Table II—Results of 53 Hepaticojejunostomies

Technique	No.	Successful outcome, no. (%)
Unstented	12	10 (83)
Stented	41	31 (76)
Straight tube	24	15 (63)
U-tube	17	16 (94)
Total	53	41 (77)

leable trochar is introduced into the left or right hepatic duct and brought out through the anterior surface of the corresponding lobe of liver. A no. 18, long, red rubber catheter is then tied to the end of the trochar, retracted back through the liver and withdrawn from the open end of the hepatic duct. The same procedure is then carried out for the other hepatic duct if necessary. The transhepatic tube is threaded into a Roux loop through a small incision on the antimesenteric border, and leaves the loop through an additional opening approximately 15 cm from its entrance. The lower end of the stent is brought through the abdominal wall to lie comfortably in relation to the Roux loop and the upper end is brought out through a separate stab incision near the exit from the liver surface. Side holes are cut in the tube before positioning so that bile can drain from the bile duct into the Roux-en-Y loop (an additional tube with identically located side holes is prepared at the same time for future exchange). The loop is then tacked to the undersurface of the liver with interrupted catgut sutures so that the jejunal mucosa abuts the intrahepatic duct where the rubber tube enters the jejunum from the liver. This procedure may be repeated for the other intrahepatic duct and the tube secured at its jejunal exit with a fine catgut pursestring suture.

The advantage of U-tube stenting in such high strictures with extensive scarring is that both ends of the tube lie outside the abdomen. If the tube becomes blocked with sludge it can easily be replaced.

Mucosal graft technique.—This operation is a refinement of the U-tube technique. Instead of a simple opening in the antimesenteric border of the Roux-en-Y loop, a 1-cm rim of seromuscular layer is removed, leaving exposed mucosa. A no. 18 red rubber tube is placed transhepatically as described for the U-tube technique. The end of the tube is then brought through a small opening in the exposed mucosa and secured there with a fine chromic catgut suture. With a combination of gentle manipulation of the jejunal loop from below and traction on the transhepatic tube above the liver, the exposed mucosa is gradually approximated to the intrahepatic duct. This may be secured in place as well, using interrupted fine catgut sutures between the seromuscular layer surrounding the mucosa and the scar tissue around the intrahepatic duct. The distal end of the tube sits free in the jejunal loop and the proximal end is brought out through a stab wound near the hepatic exit. This technique provides mucosa-to-mucosa apposition under very difficult circumstances and has the advantage of requiring stenting for a shorter time than with the U-tube technique.

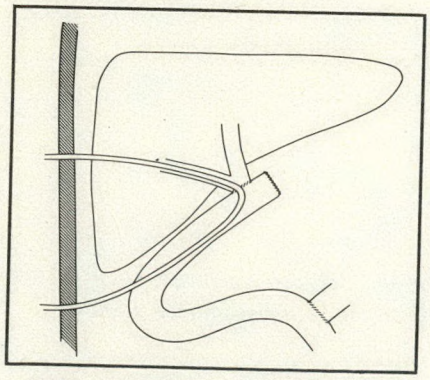

FIG. 5—U-tube technique. Single replaceable U-tube stent is placed across hepaticojejunostomy at bifurcation.

Results

There were no operative deaths. One patient died of liver failure and secondary biliary cirrhosis 2 months after his third biliary reconstruction. The overall complication rate following these 75 operations was 24.3%. Wound infection (33.3%) and persistent bile leak around the drains (22.2%) were encountered most frequently.

A repair was considered successful if the patient was asymptomatic at follow-up or could be managed conservatively when mild, intermittent symptoms were present. The result was deemed unsatisfactory if reoperation was necessary.

Overall, 60 of the 75 repairs (80%) were successful; 52 patients were asymptomatic and the remaining 8 complained of only occasional mild symptoms. The mean follow-up in this group was 27 months (range from 3 to 144 months). The overall failure rate was 20%. The mean period between operation and reoperation was 11 months (range from 2 to 42 months). Reoperation was prompted by frequent recurrence of jaundice or cholangitis, or both.

The success rate for each type of reconstruction performed is shown in Table I. Of the 22 short strictures, 3 were managed by dilatation and intubation only, 9 by axial repair and 10 by choledochoduodenostomy. The success rate of these procedures was 86% overall. Most patients, however, had long or high strictures that required hepaticojejunostomy using a Roux-en-Y loop. Forty-one (77%) of 53 patients who underwent hepaticojejunostomy had a successful result at a mean follow-up of 28 months (range from 3 to 144 months); 7 (13.5%) of these patients have occasional mild symptoms that have not necessitated reoperation. In 12 patients whose operation failed, recurrent jaundice or cholangitis, or both, necessitated reoperation within a mean period of 13 months (range from 3 to 43 months).

Hepaticojejunostomies were stented with straight tubes in 24 cases (Table II). Fifteen of these patients had satisfactory results. Sixteen of the 17 patients receiving U-tube stents have done well, with a mean follow-up of 38.8 months. Twelve patients had unstented hepaticojejunostomies and 10 of these have had good results (mean follow-up of 17 months).

Discussion

The various causes of bile-duct obstruction were recently reviewed by Smith.[1] He reported on 3527 patients presenting with biliary obstruction between 1946 and 1977, operative trauma being the cause in 44% of cases. The preventable aspects of injury to the extrahepatic bile ducts have been dealt with in several reports.[1-5] Fundamental knowledge of normal anatomy and its variations as well as good surgical judgement are necessary to avoid operative bile-duct trauma. As Smith summarized: "The more common causes of bile-duct injury include an inadequate incision and poor exposure, poor illumination, poor anaesthesia, poor assistance, ignorance of possible anatomic anomalies, and a failure to make certain identification of the anatomical structures."[1] The injury may be obvious at the time of operation (in 25% of our patients) or, if not recognized initially, may become evident after a variable period.

Bile-duct reconstruction is associated with moderate morbidity and mortality. Warren and Jefferson[5] reported 1552 such operations at the Lahey Clinic between 1940 and 1967, with a 25% morbidity and 13% mortality. The success of bile-duct repair is often difficult to predict and depends on several technical factors, including the location of the stricture, the number of previous attempted repairs, and the presence of surrounding adhesions and acute inflammation. Poor mucosa-to-mucosa apposition

usually results in recurrent stenosis. Excess tension or overzealous mobilization of the ducts may impair blood supply and result in stenosis. Other factors, such as intrahepatic bile-duct fibrosis, local infection, impairment of hepatic function and the patient's overall clinical status, may also affect the result.

The procedure of choice for serious bile-duct injury is axial repair (choledochocholedochostomy). However, this operation is only possible when duct loss is minimal, such as at the time of initial trauma, when both proximal and distal ends are healthy. We performed nine axial repairs and eight were successful. In most circumstances an end-to-end anastomosis cannot be performed without tension. In this situation, choledochoduodenostomy or hepaticojejunostomy is preferred.

In our early experience, Roux-en-Y hepaticojejunostomy was used only for high strictures, but more recently it has become the preferred type of reconstruction for all injuries except those amenable to primary axial repair. There are distinct advantages with this technique: it can always be performed without tension regardless of the level of the anastomosis; an anastomotic leak is less hazardous with a Roux loop than with the duodenum; there is virtually complete protection against reflux of undigested intestinal contents into the biliary tree and finally, if reoperation is necessary, revision of hepaticojejunal anastomosis is far easier to perform.

Bismuth and associates[6] recently reported their long-term results using a Roux loop hepaticojejunostomy in 117 patients with benign strictures. Eighty-six percent had no symptoms attributable to biliary disease at follow-up (53.7% after 6 years). The operative morbidity was 17.8%, largely from wound sepsis; there were no deaths.

The technique of transhepatic stenting of the biliary–intestinal anastomosis in order to prevent progressive stenosis was initially described by Smith.[7,8] Saypol and Kurian[9] modified this approach by bringing both ends of the rubber stent through the abdominal wall, facilitating tube replacement. We reported our experience with this technique in eight patients and concluded that U-tube stenting was particularly useful when extensive scarring of the proximal duct remnant made mucosa-to-mucosa anastomosis to jejunum difficult.[10] We currently have 17 patients who underwent transhepatic U-tube stenting, and only 1 has required reoperation after 1 year. These patients were intubated for a mean duration of 9 months.

The importance of long-term stenting is also emphasized in a number of reports from the Lahey Clinic.[4,5,11]

For high strictures with appreciable proximal duct scarring, an alternative to the U-tube stent operation is the jejunal–mucosal graft technique described by Wexler and Smith[12] that we performed in three patients of this series. Smith[1] leaves his transhepatic tube in place for approximately 4 months. He reported success in 70% of patients who have undergone the procedure.

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SESAP III Question

626. A 20-year-old man has a painless, smooth, firm unilateral scrotal mass of six weeks known duration. It does not transluminate. The best management would consist of

(A) Inguinal incision with orchiectomy if a tumor is found
(B) administration of antibiotics and observation for two to four weeks
(C) needle biopsy
(D) scrotal incision to excise the mass
(E) scrotal incision and open biopsy

For the incomplete statement above select the one answer that is best of the five given.
For the critique of Item 626 see page 340 of this issue.

The Sugiura Procedure for Bleeding Esophageal Varices: a Modification

R.J. GINSBERG, MD, FRCS[C], P.F. WATERS, MD, FRCS[C] AND S.M. STRASBERG, MD, FRCS[C]

The Sugiura procedure is an operation for control of bleeding esophageal varices. Splenectomy, extensive paraesophagogastric devascularization, and transection and anastomosis of the esophagus are performed through two incisions. The modifications described by the authors, such as the single thoracoabdominal incision and use of a stapler, simplify and shorten the operation. Preliminary results in nine patients are encouraging.

L'opération Sugiura sert à arrêter le saignement des varices oesophagiennes. Une splénectomie, une dévascularisation para-oeso-gastrique étendue et une dissection transversale avec anastomose de l'oesophage sont pratiquées à travers deux incisions. Les auteurs décrivent des modifications telles qu'une incision thoraco-abdominale simple et l'utilisation d'une agrafeuse, qui simplifient l'opération et en racourcissent le temps. Les premiers résultats chez neuf patients sont encourageants.

The Sugiura procedure is a new technique for treating esophageal varices. It was developed in response to poor results obtained by standard portacaval shunts.[1-3] Starting initially with esophageal transection as described by Walker,[4] the procedure was modified by Sugiura in stages to include an extensive paraesophagogastric devascularization and splenectomy. The results obtained by Sugiura[3] have been outstanding, with an actuarial survival rate of 83%, more than double that for standard shunts.

We were attracted to the operation not only by the results reported by Sugiura, but also by the fact that the operation approached the problem of varices in a manner that was in harmony with our understanding of their pathogenesis. Portal hypertension should be viewed as a beneficial response to the block in blood flow to the liver due to cirrhosis, since portal hypertension maintains liver blood flow. When portal pressure reaches about 20 cm saline, portacaval shunts develop. As the block in the liver worsens, the shunts increase in size, and there is probably little or no further increase in portal pressure. This concept is in keeping with the observation that portal pressure and the degree of shunting are largely unrelated.[5] Portacaval shunts may develop in many areas, but the most prominent and clinically important one is along and through the esophagus. Why this should be is unknown; however, the tendency to form this shunt is great. Ablation of this shunt, for example by classic portoazygous disconnection, is not followed by compensatory dilatation of other shunts that take over its role, but by a tendency for the shunt and for bleeding to recur. Selective decompression of varices, which recognizes the benefit of portal hypertension, is also followed by a substantial incidence of recurrent bleeding, probably because the part of the operation that disconnects the main portal system from the varices eventually fails.

The approach taken by Sugiura to deal with this situation is unique. He recognized that the shunt through the hiatus has two parts, an intraesophageal component and a paraesophageal component. The Sugiura operation obliterates the intraesophageal shunt, while carefully preserving the paraesophageal shunt. Theoretically, the paraesophageal shunt is sufficient to satisfy the need for a shunt through the hiatus and the intraesophageal shunt does not reform. No other operation incorporates this rationale.

The Sugiura operation has three parts: splenectomy, extensive paraesophagogastric devascularization and division and suture of the esophagus. Sugiura performs the operation through separate thoracic and abdominal incisions, does a selective vagotomy as part of the devascularization, which then requires a pyloroplasty, and divides and sutures the esophagus by hand. It is in these areas that we have modified the procedure. Sugiura's description and diagrams of the method used to preserve the paraesophageal shunt are schematic and we have endeavoured to provide a description from our experience that may be a more practical guide for the surgeon.

Modifications

Incision

A thoracoabdominal incision is used (Fig. 1). With the patient in the right lateral decubitus position, the incision is made in the seventh or eighth intercostal space (i.e., that which, if extended, would cross just above the umbilicus). Posteriorly, the incision reaches the erector spinae, but anteriorly it extends only 3 to 5 cm beyond the left costal margin and usually not beyond the edge of the left rectus abdominis muscle. The diaphragm is incised circumferentially at about 1.5 cm from its costal attachment for a distance of 2 to 3 cm on each side of the incision, although extensions may be made to facilitate exposure in short, thick-chested persons. The incision is closed in layers starting with the diaphragm, which is resutured to its costal attachment with no. 1 Vicryl, beginning at the extremity of each incision and tying the sutures to each other and to that of the internal oblique-transversus abdominis muscle layers. The costal margin is not sutured, but the ribs are approximated by pericostal sutures.

Comment.—This incision provides superior access to the left upper qua-

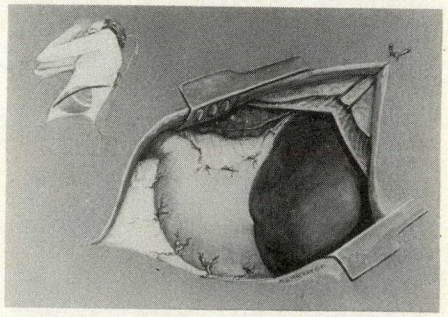

FIG. 1—Incision used for modified Sugiura procedure.

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drant of the abdomen (Fig. 1). Removal of any spleen, particularly a large spleen with body wall adhesions, is considerably easier than through a midline incision. The esophagogastric junction and intra-abdominal esophagus, which are the focal points of the subdiaphragmatic portion of the operation, are easy to see and to reach. Little retraction is needed, as the liver, even when large, sinks down to the right. Access to the intrathoracic esophagus up to the inferior pulmonary vein is ample and one may simply and quickly inspect the esophagus on both sides of the diaphragm, an important advantage when completing the devascularization right at the hiatus. Closure of the incision is straightforward, preserves diaphragmatic function and results in few problems as long as the costal cartilages are not sutured.

Subdiaphragmatic Portion of the Operation

Splenectomy is performed by standard techniques. Sugiura does not state explicitly how far down the greater curvature the gastric devascularization is carried, but it appears from his diagrams that only the short gastric vessels are taken,[1,3] although the omentum may be opened below the gastroepiploic arcade. Sugiura also states that the upper half of the stomach is stripped of venous collaterals.[3] We have used this as a guide and removed gastroepiploic branches from the upper half of the greater curvature.

The remainder of the devascularization is best viewed as if making a longitudinal incision on one side of a banana, then lifting the skin in front and behind, exposing and dividing attachments between pulp and skin until the whole midsection of the pulp is free and exposed. To do this the left lateral portion of the esophagus is exposed and the nerves, vessels, peritoneum and fat are lifted off the esophagus in front and behind as two flaps, and only those branches that directly enter the muscle of the esophagus or bare area of the stomach posteriorly are ligated and divided. Eventually, the right lateral border of the esophagus is reached and cleared, and the flaps of nerves, fat, peritoneum and vessels fall away to the right. At this point the large paraesophageal veins (now completely detached from the esophagus) can be seen running up to the hiatus. This portion of the operation is completed by the technique used in highly selective vagotomy to clear the lesser curvature down to the "crow's foot" (Fig. 2). Again it is essential to stay on the gastric wall to avoid injuring the longitudinal veins.

Comment.—Sugiura's diagrams in the 1973 and 1977 papers[2,3] differ slightly. In his latter paper it appears that the major paraesophageal vein is a continuation of the venous arcade along the lesser curvature (i.e., right gastric and left gastric veins that appear to drain into the portal vein by way of the right gastric vein). We believe that the major shunt is by way of the coronary vein, since following completion of the abdominal portion of the operation we have been able to see a large vein passing through the hiatus on the right side of the esophagus in a plane posterior to the lesser curvature of the stomach (Fig. 2).

Sugiura continues the devascularization for 7 cm along the lesser curvature from the esophagogastric junction. Our point of reference is the "crow's foot" since we wish to preserve pyloric function.

Thoracic Portion of the Operation (Fig. 3)

The inferior pulmonary ligament is divided up to the inferior pulmonary vein. A longitudinal incision is made in the mediastinal pleura over the esophagus. The esophagus is devascularized by the technique described above, first exposing the left lateral edge and peeling the esophagus away from its covering of veins, fat, nerves and pleura, dividing only those vessels that directly enter the muscle. The dissection of the anterior and posterior flaps is carried from the hiatus to the inferior pulmonary vein (12 to 18 cm). This is facilitated by dissecting the midportion first and passing a Penrose drain around the first section of cleared esophagus. Again great care is taken to preserve the longitudinal veins. The final portion of the devascularization is performed at the hiatus. Working from both sides the surgeon can free the esophagus from all venous attachments, often without destroying the phrenoesophageal membrane. If the dissection is performed from one side only, this is difficult to accomplish.

Comment.—Although Sugiura obviously intends the paraesophageal venous plexus to be preserved, it is our impression that this is not well understood and that coronary and other longitudinal veins have been ligated in the course of a "Sugiura procedure". It is possible that preservation of the paraesophageal longitudinal veins is not necessary. However, in reporting results it is important to indicate whether or not the longitudinal plexus was preserved, since the results may be different, as would be the rationale for the operation.

As in the abdominal part of the operation it is possible to see the large longitudinal paraesophageal vein, which is usually posterior and just to the right of the esophagus; Sugiura also shows a left longitudinal vein in the thorax, but in our experience this is far less prominent.

In both abdominal and thoracic portions of the operation, the two technical features that are most important are careful hemostasis and meticulous development of the flaps, otherwise longitudinal veins will be interrupted.

Division and Repair of the Esophagus

The circular stapler (EEA Auto

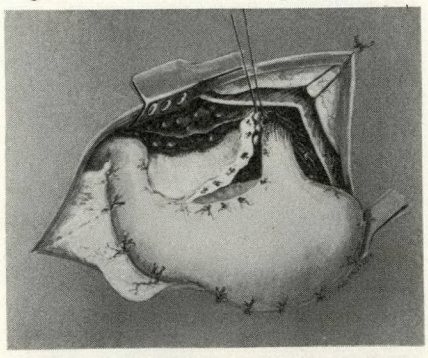

FIG. 2—Completion of abdominal dissection. Esophagus has been cleared from left to right and highly selective vagotomy performed, clearing lesser curvature to "crow's foot". Large vein (lifted forward), usually seen posterior to plane of lesser omentum, is major portoazygous connection. It is probably continuation of coronary vein.

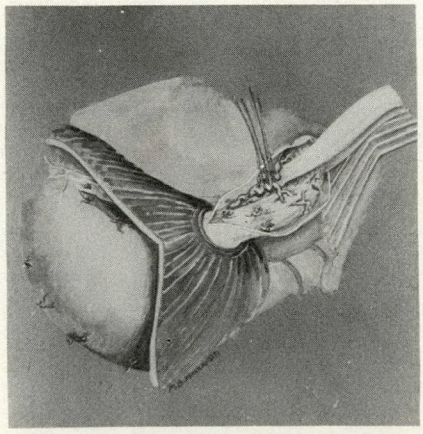

FIG. 3—Thoracic portion of procedure. Veins entering esophageal wall are ligated. Longitudinal veins are preserved. Large longitudinal vein shown is continuation of that in Fig. 2.

Stapler, Auto Suture Company, Division of United States Surgical Corp., Stamford, Conn.) is inserted through a small gastrotomy and an absorbable suture used to tie down the esophagus (Fig. 4). The point of transection is 2 to 3 cm above the esophagogastric junction. After removal of the instrument a D-tube is carefully guided through the anastomosis into the stomach. The gastrotomy is closed and the operation is completed by a loose Nissen fundoplication.

Comment.—The circular stapler makes a difficult procedure easy and quick. The Nissen wrap is used to protect the anastomotic line and as an antireflux procedure. It must be loose.

Results

To determine the scope of this procedure, we decided to accept for operation all patients referred with proven varices that had bled. All patients had serious variceal hemorrhage requiring transfusion of several units of blood. The term emergency was applied to those patients in whom bleeding was active at the time of operation, and the term elective to all other patients, including those who had stopped bleeding only 24 hours before operation and who had a Blakemore tube still in position (two patients).

Five patients had alcoholic cirrhosis and four had cirrhosis due to other causes.

Seven elective procedures were performed over 22 months. One patient had had two previous procedures, a Warren shunt and a splenectomy. Operations were performed in combination by two surgeons, a thoracic surgeon and an abdominal surgeon with a major interest in liver disease. The operating time was about 3 hours and the blood loss about 1500 ml. All patients had an excellent recovery, without postoperative encephalopathy and surprisingly small changes in liver function. At the time of writing there has been no hemorrhage, during a follow-up that has ranged from 1 to 22 months. Several patients have complained of dysphalgia to solid food at 4 to 6 weeks and have required one or two dilatations with a Maloney bougie. In one patient dilatation resulted in a small leak at the anastomosis 6 weeks after operation; the leak was managed conservatively with total parenteral nutrition for 2 weeks. Repeated dilatations (i.e., more than two) have not been required. One patient who complained of epigastric pain was found to have a duodenal ulcer 6 months after operation. This patient was treated with cimetidine for 6 months and has now been free of ulcer and off medication for 6 months.

In two patients emergency operations were performed. Both were bleeding actively despite pressure of a Blakemore tube with gastric balloon inflated. Both were encephalopathic, deeply jaundiced and had coagulopathies, one of which was severe. One patient had massive ascites and muscle wasting. Both patients died in the postoperative period, one on the first postoperative day of an uncontrollable coagulopathy, and the other at 14 days, of a combination of sepsis and liver failure.

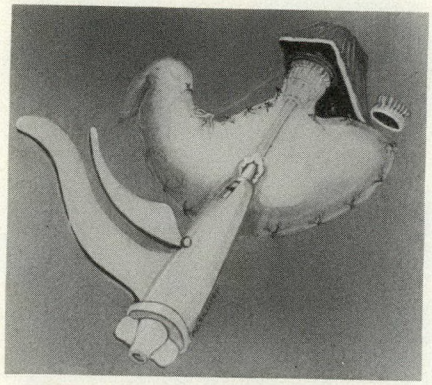

FIG. 4—Transection and anastomosis with EEA circular Auto Stapler. "Button" of tissue removed is shown at right.

Comment

The Sugiura procedure is a new and logical approach to the problem of bleeding varices. We have modified several aspects of the procedure; these modifications will increase the ease, speed and safety of the operation. The results in our series of nine patients are preliminary and permit no objective statements about the efficacy of the procedure. However, these results are sufficiently encouraging that a controlled trial is planned.

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BOOK REVIEWS

continued from page 282

The 16 chapters are systematically laid out in numbered subheadings and each chapter basically follows the same format. A reference and subject index follows at the end of each chapter.

Everything that one would want to know about a particular operation is detailed, including initial comments on anesthesia, positioning, exsanguination and skin preparation. The book contains 425 figures, most in colour, all of which are artist's drawings. The figures are clear and concise.

The basic techniques used and described are familiar to all practising hand surgeons and illustrations of their application are numerous; for example, on syndactyly alone there are 58 pages with half of that number dedicated to illustrations, on right-hand pages, of all types and degrees of syndactyly and the left-hand pages related to a textual description with timely advice regarding the particular procedure.

This book is an atlas of the techniques found most useful by the authors and no time is wasted on extensive discussion of alternative techniques.

Omitted are references to the role of microvascular surgery of free tissue transfers because this text is a result of work done before this development.

I found very little in this book to argue with except perhaps the use of the tubed pedicle in restoring or reconstructing a web space, but even this was restricted to one particular situation.

This is a classic atlas and a must as a reference for anyone doing hand surgery particularly that involving congenital deformities.

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GASTROINTESTINAL HEMORRHAGE. Edited by Richard G. Fiddian-Green and Jeremiah G. Turcotte. 429 pp. Illust. Grune & Stratton, Inc., New York; Academic Press Canada Ltd., Don Mills, Ont., 1980. $57.85. ISBN 0-8089-1267-4.

This book resulted from a symposium on gastrointestinal hemorrhage held at the University of Michigan. The clinical evaluation and investigation of gastrointestinal hemorrhage provides a good introduction. A separate chapter is devoted to bleeding in infants and children. The section on chronic gastrointestinal blood loss features an easy-to-follow "diagnostic flow chart". Diagnostic endoscopy and angiography are discussed thoroughly and the chapter on angiography details several typical radiologic findings that would be of particular interest to the non-radiologist.

continued on page 339

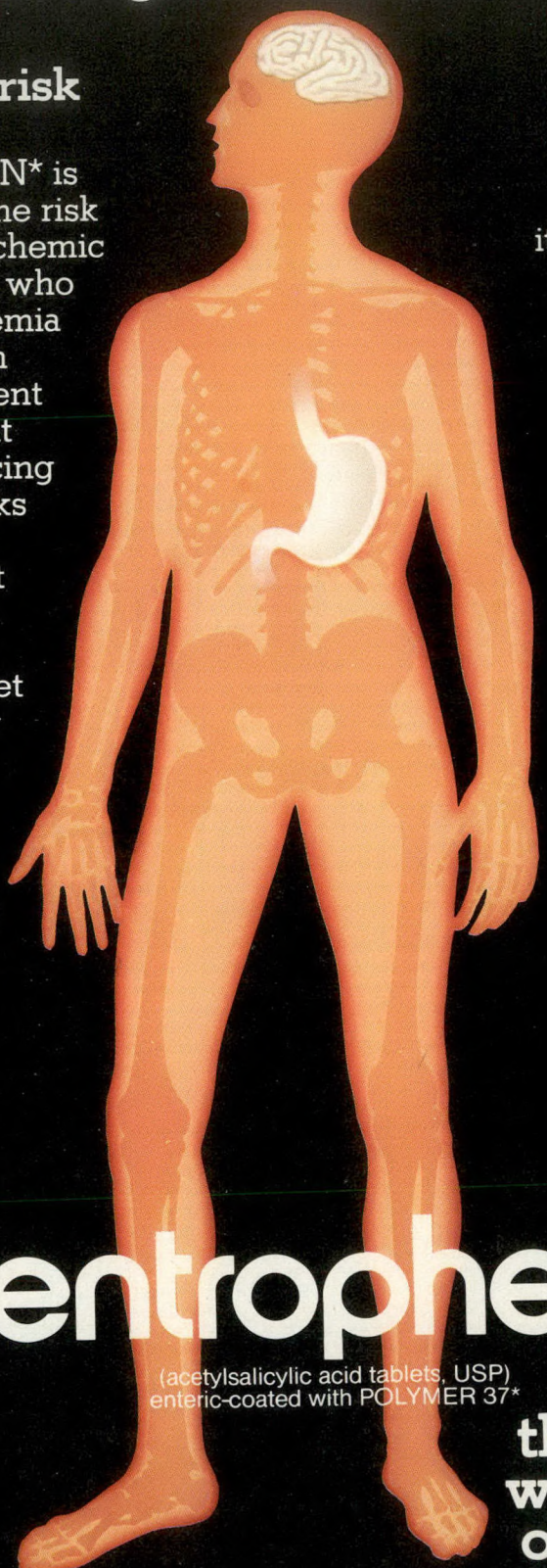
NOW IN STROKE

The Advantages of ENTROPHEN*

To reduce the risk of stroke

Now, ENTROPHEN* is indicated for reducing the risk of recurrent transient ischemic attacks or stroke in men who have had transient ischemia of the brain due to fibrin platelet emboli. At present there is no evidence that ASA is effective in reducing transient ischemic attacks in women, or is of benefit in the treatment of completed strokes in men or women.

Inhibition of platelet cyclooxygenase activity by a single dose of ENTROPHEN*-10 was comparable to that of plain ASA, although the effect was delayed, reflecting the delayed appearance of ASA in the plasma.[1]



with reduced risk of stomach upset

When you prescribe ASA for long-term use, it is important not to create additional problems for your patients.

While they may benefit from the therapeutic effect of ASA, there is still a potential for gastric irritation and upset, particularly when the regimen calls for continuous daily dosage.

Clinical experience has shown that ENTROPHEN*, coated with POLYMER 37* reduces gastric distress in long-term treatment with high doses of ASA.

entrophen*

(acetylsalicylic acid tablets, USP)
enteric-coated with POLYMER 37*

To reduce the risk of stroke with reduced risk of stomach upset

Frosst
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1. Ali, M. et al.: Plasma acetylsalicylate and salicylate and platelet cyclooxygenase activity following plain and enteric-coated aspirin,

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TABLETS

entrophen*

(acetylsalicylic acid tablets, USP)

Enteric-coated with POLYMER 37*

Anti-inflammatory—Analgesic Agent

Platelet Aggregation Inhibitor

DESCRIPTION

ENTROPHEN* is an enteric-coated tablet containing acetylsalicylic acid coated with POLYMER 37*, a partially esterified polyvinyl alcohol.

ACTION

Acetylsalicylic acid (ASA) has analgesic, antipyretic and anti-inflammatory properties.

In rheumatic diseases, although the analgesic and antipyretic effects are useful, the major purpose for which ASA is used is to reduce the intensity of the inflammatory process. Inhibition of prostaglandin synthesis may be involved in the anti-inflammatory action of ASA.

ASA also alters platelet aggregation and release reaction by inhibiting prostaglandin synthesis. Thromboxane A_2 is an essential step in platelet aggregation. ASA prevents Thromboxane A_2 formation by acetylation of platelet cyclooxygenase. This inhibition of prostaglandin synthesis is irreversible and affects platelet function for the life of the platelet.

The POLYMER 37* coating substantially resists disintegration in aqueous fluids having a pH lower than 3.5 for a period of at least 2 hours and is capable of disintegrating in aqueous fluids having a pH of at least 5.5 in from 10 to 30 minutes. Thus, POLYMER 37* coating effectively inhibits the release of ASA in the stomach, whilst allowing the tablet to dissolve in the upper portion of the small intestine for absorption from the duodenal area.

Clinical experience has shown that POLYMER 37* coated acetylsalicylic acid diminishes or eliminates gastric distress during long-term treatment with high doses of ASA.

INDICATIONS

ENTROPHEN* is indicated whenever gastric intolerance to ASA is of concern.

ENTROPHEN* is indicated for the relief of signs and symptoms of the following:

Osteoarthritis
Rheumatoid arthritis
Spondylitis
Bursitis
and other forms of rheumatism
Musculoskeletal disorders

Rheumatic fever, however, penicillin and other appropriate therapy should be administered concomitantly.

ASA is generally considered to be the primary therapy for most forms of arthritis.

ENTROPHEN* is also indicated for reducing the risk of recurrent transient ischemic attacks or stroke in men who have had transient ischemia of the brain due to fibrin platelet emboli. At present there is no evidence that ASA is effective in reducing transient ischemic attacks in women, or is of benefit in the treatment of completed strokes in men or women.

CONTRAINDICATIONS

Sensitivity to the ingredients
Active peptic ulcer

Patients who had a bronchospastic reaction to ASA or non-steroidal anti-inflammatory drugs.

WARNINGS

ASA is one of the most frequent causes of accidental poisoning in toddlers and infants. ENTROPHEN* should, therefore, be kept well out of the reach of all children.

PRECAUTIONS

Salicylates should be administered with caution to patients with asthma and other allergic conditions, with a history of gastrointestinal ulcerations, with bleeding tendencies, with significant anemia or with hypoprothrombinemia.

Salicylates can produce changes in thyroid function tests.

Acute hepatitis has been reported rarely in patients with systemic lupus erythematosus and juvenile rheumatoid arthritis with plasma salicylate concentrations above 25 mg/100 mL. Patients have recovered upon cessation of therapy.

Use in Pregnancy

ASA does not appear to have any teratogenic effects. ASA has been found to delay parturition in rats. This effect has also been described with non-steroidal anti-inflammatory agents which inhibit prostaglandin synthesis.

High doses (3 g daily) of ASA during pregnancy may lengthen the gestation and parturition time.

Because of possible adverse effects on the neonate and the potential for increased maternal blood loss, ASA should be avoided during the last three months of pregnancy.

Drug Interactions

Caution is necessary when ENTROPHEN* and anticoagulants are prescribed concurrently, as ASA may potentiate the action of anticoagulants.

Salicylates may potentiate sulfonylurea hypoglycemic agents. Large doses of salicylates may have a hypoglycemic action, and thus, affect the insulin requirements of diabetics.

Although salicylates in large doses are uricosuric agents, smaller amounts may depress uric acid clearance and thus decrease the uricosuric effects of probenecid, sulfinpyrazone and phenylbutazone.

Sodium excretion produced by spironolactone may be decreased in the presence of salicylates.

Salicylates also retard the renal elimination of methotrexate.

ADVERSE REACTIONS

Gastrointestinal reactions: nausea, vomiting, diarrhea, gastrointestinal bleeding and/or ulceration. Ear reactions: tinnitus, vertigo, hearing loss. Hematologic reactions: leukopenia, thrombocytopenia, purpura. Dermatologic and Hypersensitivity reactions: urticaria, angioedema, pruritus, various skin eruptions, asthma and anaphylaxis. Miscellaneous reactions: acute reversible hepatotoxicity, mental confusion, drowsiness, sweating and thirst.

SYMPTOMS AND TREATMENT OF OVERDOSAGE

Symptoms

In mild overdosage these may include rapid and deep breathing, nausea, vomiting (leading to alkalosis), hyperpnea, vertigo, tinnitus, flushing, sweating, thirst and tachycardia. (High blood levels of ASA lead to acidosis.) Severe cases may show fever, hemorrhage, excitement, confusion, convulsions or coma, and respiratory failure.

Treatment

Treatment is essentially symptomatic and supportive. Administer water, universal antidote and remove by gastric lavage or emesis. Force fluids (e.g., salty broth) to replace sodium loss. If the patient is unable to retain fluids orally, the alkalosis can be treated by hypertonic saline intravenously. If salicylism acidosis is present, sodium bicarbonate intravenously is preferred because it increases the renal excretion of salicylates. Vitamin K is indicated if there is evidence of hemorrhage. Hemodialysis has been used with success.

Respiratory depression may require artificial ventilation with oxygen. Convulsions may best be treated by the administration of succinylcholine and artificial ventilation with oxygen. Central nervous system depressant agents should not be used.

Hyperthermia and dehydration are immediate threats to life and initial therapy must be directed to their correction and to the maintenance of adequate renal function. External cooling with cool water or alcohol should be provided quickly to any child who has a rectal temperature over 104°F.

DOSAGE AND ADMINISTRATION

Analgesic; antipyretic

Up to 2.925 g daily as necessary.

Anti-inflammatory

Because the suppression of inflammation increases with the dose of salicylate even beyond the point of toxicity, the therapeutic objective is to employ as large a dose as possible short of toxicity. Most patients will tolerate blood salicylate levels in the range of 20 to 25 mg per cent. The most common reason for failing to obtain a therapeutic response to ASA is the administration of inadequate doses.

The generally accepted way to achieve effective 'anti-inflammatory' salicylate blood levels of 20 to 25 mg per cent is to titrate the dosage by starting with 2.6 to 3.9 g daily, according to the size, age and sex of the patient. If necessary, the dosage is then gradually adjusted by daily increments of 0.65 g until symptoms of salicylism e.g., auditory symptoms, occur. Then, the dosage is decreased by 0.65 g daily until these symptoms disappear and maintained at that level as long as necessary.

In adults the median dose at which tinnitus develops is 4.5 g per day, but the range extends from 2.6 to 6.0 g per day.

Intermittent administration is ineffective. Patients should be advised not to vary the dose from day to day depending on the level of pain because that often fluctuates independently of the intensity of the inflammation. A continuous regimen of 0.65 g four times daily is considered to be minimum therapy for adults. ENTROPHEN* should be administered four times daily. For nighttime and early morning benefits, the last dose should be given at bedtime.

Once maintenance dose is established, ENTROPHEN*-15 may be useful to encourage patient compliance.

Optimally, salicylate therapy should be monitored by periodic blood salicylate level determinations. If this is not practical, the appearance of auditory symptoms in the form of tinnitus or deafness are acceptable as an indication of the maximum tolerated salicylate dose.

There is an inverse relation between blood salicylate levels at which auditory symptoms appear and the age of the patient. In the young adult, this is usually in the range of 20 to 30 mg per cent. In children, however, the level may be much higher, or the effect apparently absent. Because salicylate toxicity may appear without such warning in children, the usual practice is to give ASA in a daily dose of 50 to 100 mg per kilogram of body weight and to follow blood levels aiming for a concentration of about 30 mg per cent.

Rheumatic Fever

A total daily dosage of 100 mg per kilogram of body weight administered in divided doses to allay the pain, swelling and fever.

Cerebral ischemic attacks (men)

The recommended dosage is 1,300 mg per day (650 mg twice a day or 325 mg four times a day).

AVAILABILITY

No. 472—ENTROPHEN*-15 tablets containing 975 mg of acetylsalicylic acid USP, coated with POLYMER 37*. Oval, pale yellow, film-coated tablets with the FROSST name engraved on one face and 472 on the other and supplied in bottles of 100 and 500.

No. 470—ENTROPHEN*-10 tablets containing 650 mg of acetylsalicylic acid USP, coated with POLYMER 37*. Oval, orange, film-coated tablets, with the FROSST name engraved on one face and 470 on the other and supplied in bottles of 100, 500 and 1,000.

No. 438—ENTROPHEN*-5 tablets containing 325 mg of acetylsalicylic acid USP, coated with POLYMER 37*. Round, brown, film-coated tablets, with the FROSST name engraved on one face and 438 on the other and supplied in bottles of 100, 500 and 1,000.

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Unicameral Bone Cyst

K.S. MORTON, MD, CM, M SC, FRCS[C], FACS

A 28-year experience with 76 unicameral bone cysts is reviewed and compared to other large series and recommended treatments recorded in the English literature. Particular attention is paid to the importance of recurrence in relation to the need for secondary operations and in contrast to the disability caused by enforced restriction of activity. Evidence is presented suggesting that, in spite of residual radiologic changes, the disability is frequently insufficient to warrant a second operation. Most unicameral bone cysts probably run their own course, not always greatly affected by treatment, with very satisfactory end results.

L'expérience de 28 années portant sur 76 kystes osseux uniloculaires est analysée et comparée aux autres grandes séries et aux traitements recommandés dans la littérature de langue anglaise. Une attention particulière est accordée à l'importance des récidives en rapport avec la nécessité de nouvelles opérations et en comparaison de l'incapacité rencontrée lorsqu'une restriction des activités est imposée. Les constatations présentées indiquent qu'en dépit de changements radiologiques résiduels, l'incapacité est fréquemment insuffisante pour justifier une deuxième opération. La plupart des kystes osseux uniloculaires se résolvent probablement spontanément, sans être bien souvent affectés par le traitement et avec un résultat final fort satisfaisant.

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Unicameral bone cyst has been a well-known but little understood entity since it emerged from a conglomerate of cystic and fibrous lesions of bone discussed by authors such as Bloodgood[1] and Elmslie.[2] Jaffe and Lichtenstein[3] established the entity more securely, but as recently as 1977 Cohen,[4] in an excellent summary of the subject, pointed out the continuing gaps in the knowledge of this fairly common clinical problem, particularly in the areas of pathogenesis and treatment.

Cohen's article prompted us to examine our experience of 76 cases of unicameral bone cyst registered with the University of British Columbia Bone Tumour Registry over the 28-year period from 1951 to 1978. It was immediately apparent that no statistical analysis could be undertaken because of the limitations Cohen described: the changing patterns of management, the relatively small numbers in relation to age of patient and bone involved and, inevitably, the inadequate follow-up information. Nevertheless, certain observations are valid and conclusions justified.

Patients

Our 76 patients (51 male, 25 female) all had solitary lesions (Fig. 1). When first seen they ranged in age (Table I) from 2 to 57 years. The commonest age was 12 years and the majority were within the first two decades of life. The common sites were the upper humerus (25 patients) and upper femur (22 patients), but 8 cysts occurred in the humeral shaft and 21 were widely distributed in all bones but the skull.

Of the 76 patients, 64 were treated operatively; in 12 no operation was carried out so a pathologic diagnosis was not made. The type of operation (Table II) reflects the timing of treatment rather than our current practice, for these patients were treated over a period of almost three decades. The most common procedure was curettage and bone grafting (45 cases), in earlier years with frozen bank bone and often accompanied by cautery.

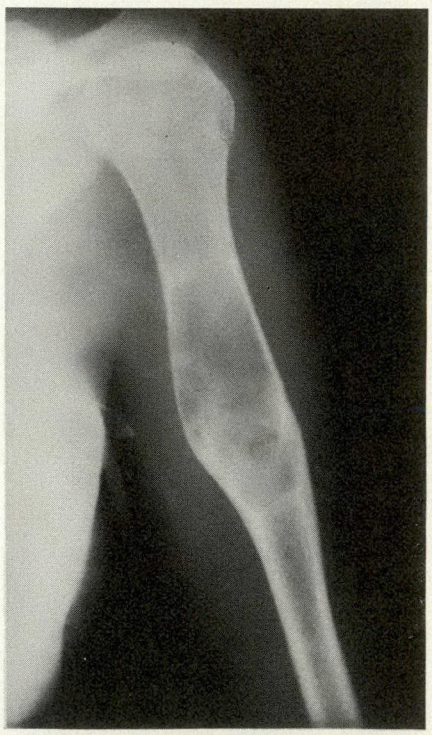

FIG. 1—Typical radiologic appearance of unicameral bone cyst of proximal shaft of humerus in 14-year-old girl.

Table I—Age Distribution of 76 Cases of Unicameral Bone Cyst

Age group, yr	No. of patients
0–10	37
11–20	24
21–30	6
31–40	7
>40	2

Later, autografts were used. The most recent surgical procedure has been subtotal, subperiosteal excision with or without subperiosteal bone grafting (two cases).

Results

On the whole the results were satisfactory in that we have recognized no disability, no loss of limb and no associated malignant conditions. The validity of this conclusion can be judged in part by examination of our follow-up figures (Table III), with only 28 patients followed up for 4 years or more and 37 patients followed up for less than 2 years.

Fracture and Refracture

Forty-four of 76 patients presented with a fracture through, into or near the cyst. The fracture ranged from a fine crack through thinned cortex to, less often, comminuted and displaced fractures requiring treatment. Eleven patients were known to have suffered a second fracture; 1 of these had three and another four fractures.

Reoperation

Six patients underwent two operations. In all instances the initial procedure was curettage and grafting with banked bone. In three patients the second operation was the same while the remaining three had autografts. All these second operations were done in the 1956 to 1967 period. Another patient had two osteotomies for correction of varus deformity from a cyst in the trochanteric region of the femur.

Table II—Type of Operation Performed in 64 Cases of Unicameral Bone Cyst

Operation	No. of patients
Curettage	13
Curettage and banked bone graft	29
Curettage and autogenous graft	16
Subperiosteal resection	2
Other	1
Not described	3

Table III—Length of Follow-up in 76 Cases of Unicameral Bone Cyst

Follow-up, yr	No. of patients
0–½	23
½–1	5
1–2	9
2–3	10
3–4	1
4–5	8
5–10	15
>10	5

Shortening and Deformity

Three patients, all with cysts of the upper humerus, demonstrated up to 5.1 cm of shortening of the involved bone on follow-up 3 to 9 years after pathologic fracture and operation. Though angular deformity sometimes occurred, usually in humeral cysts, it did not persist and was not a complaint in any patients followed up for 4 years or longer. One patient with a cyst in the trochanteric region of the femur required osteotomy for varus deformity on two occasions, at the age of 6 and 7 years. Three years later the cyst had healed and the result was described as satisfactory though there was some valgus deformity at the hip.

Restricted Activity

Perhaps the greatest problem in these patients is the sometimes lengthy and possibly unnecessary period of restricted activity because of fear of pathologic fracture. We are convinced that any treatment that leads to strengthening of the bone and hence shortening of this period of relative inactivity is to be recommended.

Discussion

In spite of our apparently good results we are aware first that deformity, particularly from injudicious surgery, is a potential problem. This is illustrated by the findings from one patient too recent to include in the current series. Curettings from a unicameral bone cyst of the ankle show epiphyseal plate damage that is expected to produce progressive growth deformity (Fig. 2).

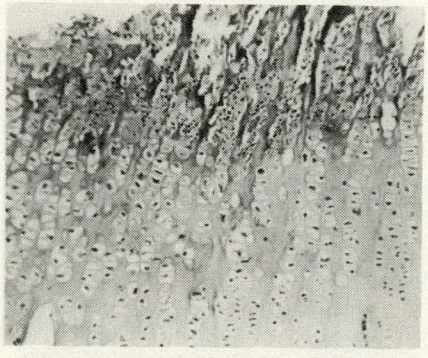

FIG. 2—Curettings from treated unicameral bone cyst showing organized cartilage of epiphyseal plate (hematoxylin and eosin, reduced by 52% from ×125).

Neer and associates[5] defined recurrence of unicameral bone cyst as "continuing expansion and thinning of the cortex" requiring reoperation. On this basis they reported failure rates of initial curettage and grafting as 30% in the upper humerus and 17% in the upper femur.

In another large series, Spence and associates[6,7] categorized any residual cystic defect as a recurrence and found that 64 (36%) of 177 lesions curetted and grafted with freeze-dried cancellous bone allograft recurred to the extent that reoperation was indicated. They also reported greater difficulty with upper humeral lesions. Follow-up ranged from 7 months to 13 years (mean 27 months). In their later paper, reporting 144 lesions curetted and grafted with freeze-dried crushed cortical bone allograft, their overall recurrence rate was 25%; follow-up ranged from 1 to 4 years.

A different surgical approach was reported by Fahey and O'Brien[8] in a small series of 20 patients, that of subtotal resection and autologous or homologous bone grafting. Using strict criteria they had a recurrence rate of 5% at an average follow-up of 3.9 years. McKay and Nason[9] obtained a comparable recurrence rate (9%) in 21 patients treated by subtotal resection without grafting and followed up for a similar period. In both series some patients had had previous surgical treatment.

In the 76 cases reported here, using the criterion of reoperation as the standard for recurrence, our rate was 8%. Clearly this rate would be much higher if the criteria used by Spence and associates,[6,7] and by Fahey and O'Brien[8] were applied, but our series is comparable to that of Neer and associates.[5]

We believe the important feature in judging results of treatment of unicameral bone cyst is the morbidity or disability the individual patient experiences in relation to restriction of his activities, not the roentgenographic appearance following operative treatment and therefore not the "recurrence rate". If, in upper extremity or lower extremity cysts, there is sufficient bone strength to permit unrestricted childhood activities, then the treatment has been successful whether or not there is radiologic recurrence or residual disease. If, on the other hand, there is insufficient confidence

in the strength of the bone to allow the patient reasonable activity, then this may be the most valid criterion of failure and the best indication for a second operation.

As Cohen[4] pointed out, no conclusions are possible in a retrospective review such as this. Our observations, however, support the need for a greater understanding of the natural history of unicameral bone cyst, whether it be untreated, affected by fracture, treated surgically or, as more recently reported, treated by steroid injection.[10]

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Oncocytoma of the Lung

Marco A. Fernandez, md* and J. Nyssen, ba, ph d†

This report describes the clinical, anatomical, histologic and ultrastructural features of a bronchial oncocytoma. Histologically the oncocytes have granular eosinophilic cytoplasm. Ultrastructurally this corresponds to mitochondrial hyperplasia. Because cytoplasmic eosinophilia may be due to an abundance of cytoplasmic organelles other than mitochondria, it is emphasized that electron-microscopic study is an essential component in the diagnosis of oncocytomas.

Cette communication décrit les caractéristiques cliniques, anatomiques, histologiques et d'ultrastructure de l'oncocytome bronchique. Sur le plan histologique, les oncocytes possèdent un cytoplasme granulaire éosinophile. L'ultrastructure correspond à celle d'une hyperplasie mitochondriale. Comme l'éosinophilie cytoplasmique peut être due à une abondance d'organelles cytoplasmiques autres que les mitochondries, on souligne l'importance de considérer l'examen en microscopie électronique comme une composante essentielle du diagnostic des oncocytomes.

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Oncocytoma is a rare variant of bronchial adenoma and is characterized by large cells in contrast to the small cells of the more common carcinoid. Seen under the electron microscope, the eosinophilic cells of the oncocytoma have cytoplasm filled with mitochondria. This tumour may occur in salivary, parathyroid and thyroid glands. Only a few cases diagnosed as bronchial oncocytoma fulfil the criterion of mitochondrial hyperplasia. We present a case of oncocytoma of the lung confirmed by ultrastructural studies.

Case Report

Two weeks before admission to hospital this previously healthy 30-year-old man had sudden onset of cough and hemoptysis. He had recently been on a diet and a program of strenuous exercise in order to lose weight. During the previous 4 months he had lost 8 kg. He had had two episodes of pneumonia in the previous 2 years and had smoked 25 cigarettes daily for 10 years.

The only abnormality found on physical examination was an enlarged thyroid. It felt nodular but was smooth-edged and enlarged, especially on the right. There was no lymphadenopathy.

A chest roentgenogram demonstrated a coin lesion, 3.5 cm in diameter, in the basal segment of the lower lobe of the right lung. Results of skin tests for fungus were negative. A thyroid scan showed many areas of reduced uptake, suggesting a nodular goitre. Iodine-131 uptake indicated a euthyroid state.

Computerized axial tomography of the thorax confirmed the presence of a well-circumscribed mass in the posterior basal segment of the right lower lobe (Fig. 1). Mediastinal nodes were not enlarged.

On bronchoscopic examination a cherry red tumour was found blocking the medial division of the right lower

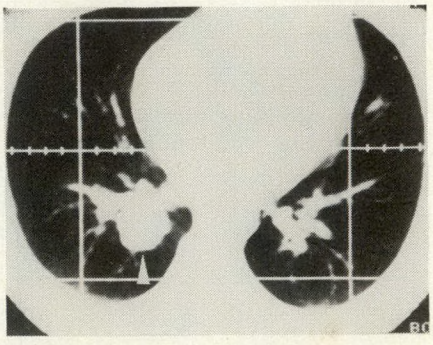

FIG. 1—Computerized tomogram of thorax shows well-circumscribed mass (arrow) which is in posterior basal segment of right lower lobe.

bronchus. The lesion was quite friable and bled easily. No biopsy specimen was obtained. Because of the presence of cold nodularity and the possibility of an occult carcinoma of the thyroid, surgical exploration of the thyroid was carried out and multiple biopsy specimens were obtained that showed chronic thyroiditis. One week after the thyroid exploration, a right lower pulmonary lobectomy was performed.

The gross specimen showed an oval, pink tumour, 1.5 cm in maximum dimension, projecting into the lumen of the bronchus but also having an extrabronchial component. Distal to the tumour the bronchus was markedly dilated forming a cylindrical cavity, 6 × 5 cm, that was filled with old blood (Fig. 2).

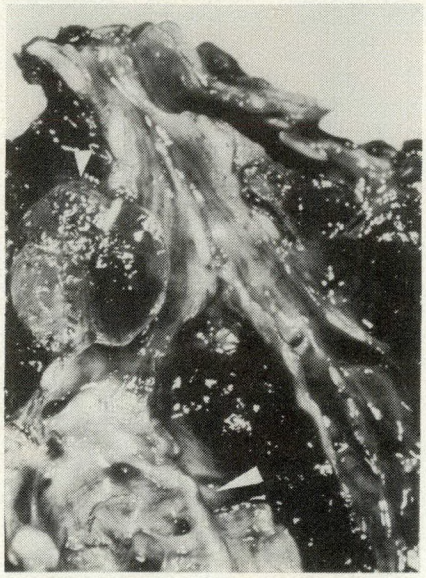

FIG. 2—Oval sessile tumour (small arrow) partially occluding bronchus, and diffuse dilatation (large arrow) distal to lesion.

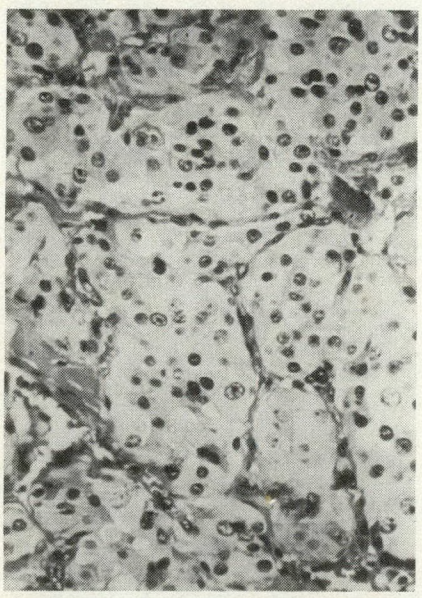

FIG. 3—Polyhedral cells with granular eosinophilic cytoplasm. Nuclei are small with finely-distributed chromatin (hematoxylin and eosin, reduced by 45% from ×400).

Microscopically, the tumour was composed of polyhedral cells with abundant granular eosinophilic cytoplasm. Their nuclei were generally small and vesicular but a few large pleomorphic forms with finely distributed chromatin were present. Mitoses were rare. The cells tended to form solid nests surrounded and outlined by numerous thin-walled blood vessels (Fig. 3). The edge of the tumour was not obviously infiltrative.

None of the lymph nodes contained tumour. Some atelectasis of the parenchyma around the dilated, chronically inflamed bronchus was noted. Hemosiderin-filled macrophages were prominent in that area. Silver stain by the Fontana–Masson method revealed a few argentaffin granules in some of the tumour cells. For electron microscopy the formalin-fixed tissue was sequentially postfixed in 3% cacodylate-buffered glutaraldehyde and 2% osmium tetroxide and was stained with uranyl acetate and lead citrate. Electron-microscopic examination showed that the tumour cells contained numerous mitochondria and a few membrane-bound dense bodies. The appearance was that of an oncocytoma (Fig. 4).

Discussion

The term oncocyte was first used by Jaffé[1] in 1932 to describe a tumour of the salivary gland composed of cells containing large amounts of granular eosinophilic cytoplasm.

Hamperl[2] described a rare variant of bronchial adenoma composed of cells with abundant strikingly eosinophilic granular cytoplasm that he termed oncocytes.

Cells with granular, eosinophilic cytoplasm containing a variety of organelles have been called oncocytes in the past. With the use of electron microscopy it has become clear that very few of these cells are true oncocytes. For example, the eosinophilic granularity of ovarian hilar, corpus luteal and testicular Leydig cells is due to abundant endoplasmic reticulum that fills the cytoplasm.[3] The cells of the granular cell myoblastoma contain lysosomal-like bodies.[4] The granularity of chemodectomas is due in large part to neurosecretory granules.[5]

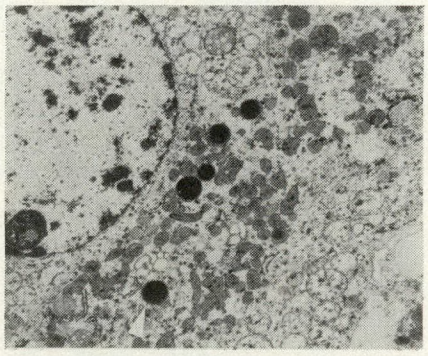

FIG. 4—Electron-microscopic appearance showing marked mitochondrial hyperplasia. Some membrane-bound dense bodies (arrow) are also present (original magnification ×8000).

Mitochondrial hyperplasia is the essential characteristic of oncocytes of the salivary[6] and parathyroid glands,[7] the Hürthle cells of the thyroid gland[8] and the epithelial component of Warthin's tumour[9] and this explains the deep eosinophilia of the cytoplasm seen with hematoxylin and eosin staining.

The main criterion for the diagnosis of the oncocytoma is mitochondrial hyperplasia. Fechner and Bentinck[10] first demonstrated this in a bronchial oncocytoma. Our case of bronchial oncocytoma confirms this observation and re-emphasizes the importance of the ultrastructural appearance.

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Cholestatic Jaundice Caused by Sulindac

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The authors describe a case of cholestatic jaundice in a 66-year-old woman that was caused by sulindac (Clinoril). The diagnosis was confirmed by challenging the patient with the drug. Abdominal pain, nausea, constipation, dizziness, somnolence, vertigo, inhibited platelet activity and hepatotoxicity have all been described in association with sulindac therapy, but this is the first time, to the authors' knowledge, that cholestatic jaundice has been reported as a side-effect.

Les auteurs décrivent un ictère cholestatique apparu chez une femme de 66 ans et causé par le sulindac (Clinoril). Le diagnostic fut confirmé par provocation de la patiente avec le médicament. Douleurs abdominales, nausée, constipation, étourdissements, somnolence, vertige, inhibition de l'activité plaquettaire et hépatotoxicité ont tous déjà été décrits en rapport avec le traitement au sulindac. Toutefois, à la connaissance des auteurs, c'est la première fois qu'un ictère cholestatique est signalé comme effet secondaire de ce médicament.

Sulindac, (z)-5-fluoro-2-methyl-l-{[p-(methylsulfinyl)phenyl] methylene}-lH-indene-3-acetic acid (Clinoril), is an anti-inflammatory and analgesic agent. Its exact mode of action is unknown, but it appears to inhibit the synthesis of prostaglandins. The maximal clinical effect is observed 3 hours after intake.[1] It is not a steroid, and is derived from indene. Its biological activity appears to be localized in the sulfur containing moiety.

On absorption, sulindac is metabolized in the liver, the reduction of sulfoxide to sulfur raising the plasma level of the active principle. Sulindac and its sulfur metabolite are bound to serum albumin and excreted principally by the kidneys, but also in the bile and feces.

Sulindac is used by some in the treatment of osteoarthritis, rheumatoid polyarthritis, ankylosing spondylitis, bursitis, tendinitis and gout (acute phase).

Side-effects include abdominal pain, nausea, constipation, dizziness, somnolence, vertigo and inhibition of platelet activity.[2] Hepatotoxicity[3,4] has been observed.

We present in this article what is, to our knowledge, the first case of cholestatic jaundice associated with the use of this drug. The diagnosis was confirmed by challenging the patient with sulindac.

Case Report

A 66-year-old white woman underwent acromioclavicular arthroplasty on May 14, 1979. When she was discharged from the hospital, sulindac (Clinoril) (200 mg *bid*) was prescribed for persistent pain. A few days after starting treatment she became anorexic, nauseated and feverish but did not seek medical advice. She was also taking isosorbide dinitrate (Isordil), nitroglycerin and flurazepam hydrochloride (Dalmane) as she had been for many years. She did not drink alcohol.

On Sept. 1, 1979 she was admitted for investigation of progressive jaundice and pruritus. Sulindac was discontinued at that time. The total serum bilirubin was 6.9 mg/dl (118 μmol/l) (mainly direct) (normal 0.2 to 1.4 mg/dl [3.42 to 23.9 μmol/l]). There were no physical signs of chronic liver disease. The serum values of amylase, lipase and creatine phosphokinase, and the hemogram and urinalysis findings were all normal as were the blood urea, sugar and electrolyte levels, the prothrombin time, blood platelets, serum iron, iron-binding capacity and serum protein, and the results of electrophoresis. Several serum levels were elevated: glutamic oxaloacetic transaminase was 211 U/ml (normal 8 to 40 U/ml), glutamic pyruvic transaminase was 328 U/ml (normal 9 to 35 U/ml), lactic dehydrogenase was 198 U/ml (normal 109 to 193 U/ml) and alkaline phosphatase was 92 IU/l (normal 9 to 35 IU/l). Ultrasonography was thought to show gallstones, so the patient was operated on. The gallbladder appeared to be normal and there were no gallstones. An intraoperative cholangiogram was normal and the intrahepatic and extrahepatic bile duct system was well visualized, so a liver biopsy was done.

The biopsy specimen showed normal architecture with well-oriented columns, though some of the cells were irregularly shaped. The portal spaces were normal. There were scattered areas of infiltration by lymphocytes (no plasmacytes or polymorphs). No necrosis was found and sinusoids were normal. There were signs of bile stasis; biliary canals were widely dilated and filled with bile, mainly in the centrilobular area. Bile

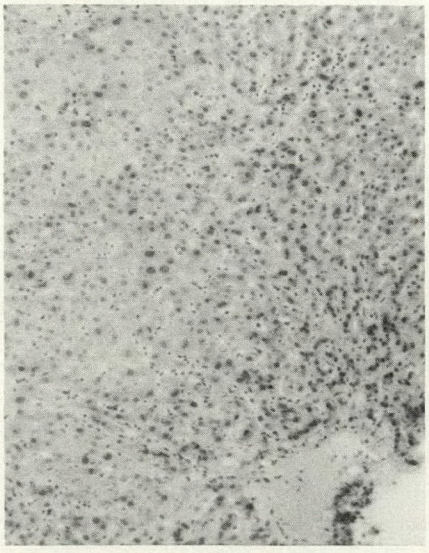

FIG. 1—Biopsy specimen of liver clearly shows well-oriented columns of cells (hematoxylin and eosin, reduced by 52% from ×200).

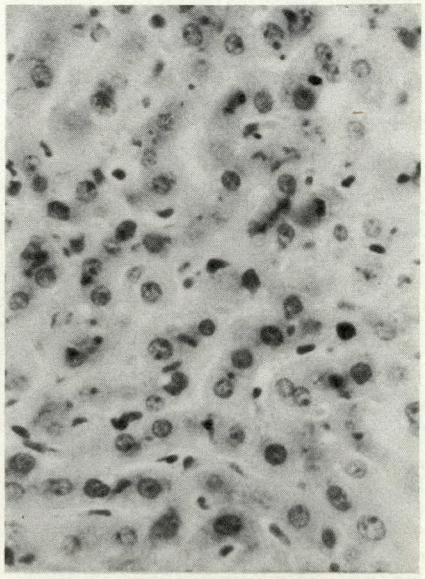

FIG. 2—Biopsy specimen shows bile stasis with bile plugs (hematoxylin and eosin, reduced by 54% from ×1200).

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was also noted in Kupffer cells and some hepatocytes (Figs. 1 and 2). There was no cholangitis but, in some areas, mild fibrosis was noted in the spaces of Disse.

The patient was seen by a gastroenterologist and the diagnosis of cholestatic jaundice, probably secondary to sulindac, was made.

Fifteen days after the operation, the total serum bilirubin value was 20.0 mg/dl (342 μmol/l) (direct 15.4 mg/dl [263 μmol/l]); the serum transaminase levels were normal, as were the prothrombin and partial thromboplastin times. The serum was negative for Australia antigen and the serum creatinine value was normal. The alkaline phosphatase value was elevated to 73 IU/l.

On Sept. 28, the patient was still jaundiced (total bilirubin 16.4 mg/dl [280 μmol/l]; direct 12.6 mg/dl [215 μmol/l]). Because of the persistent cholestasis, she was given prednisone (20 mg/d). The alkaline phosphatase level was 188 IU/l and transaminase levels were normal. There had been a slow drop in bilirubin concentration before the cortisone was given. Steroids were discontinued on Dec. 5, 1979.

On Jan. 23, 1980, the results of liver function tests were normal. The total serum bilirubin level was 1.4 mg/dl (23.9 μmol/l) (half direct). Serum alkaline phosphatase and transaminase levels were also normal.

With the patient's consent, she was challenged with sulindac to determine if that had been the cause of the cholestatic jaundice. On Mar. 5, 1980 she started taking sulindac (2 tabs/d) for 1 week. On the third day she became anorexic, nauseated, jaundiced and suffered pruritus. The total serum bilirubin level rose to 3.2 mg/dl (54.7 μmol/l) (direct 2.2 mg/dl [37.6 μmol/l]) and alkaline phosphatase to 76 IU/l. The serum transaminase levels were slightly elevated.

The medication was discontinued for humanitarian reasons and because it was clear that sulindac was the cause of the jaundice. The liver function test results returned to normal after challenge. Retrograde cholangiopancreatography showed that the pancreas, intrahepatic and extrahepatic bile ducts, cystic duct and gallbladder were all normal.

Discussion

We have demonstrated that sulindac can produce cholestasis similar to that produced by phenylbutazone. To our knowledge, this is the first case reported.

The patient (considering the elevated serum transaminase levels) may have presented a degree of hepatotoxicity following ingestion of sulindac but cholestasis was the primary manifestation. This picture of cholestasis persisted for 3 months. At operation no gallstones were found. The patient was not drinking alcohol. Postoperatively, the transaminase levels rapidly returned to normal and microscopic examination of a liver biopsy specimen showed no evidence of necrosis.

The challenge with sulindac induced a new elevation of serum bilirubin, alkaline phosphatase and transaminase levels — supporting a cholestatic more than a hepatotoxic action.

The mode of action of sulindac is not well understood and the mechanism by which it produces cholestasis remains to be elucidated. We cannot conclude that the steroids helped in the recovery because that could have been the normal response to discontinuing the sulindac.

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NOTICES

Vascular Surgery

Vascular surgery will be the major topic of the 46th annual surgery course sponsored by the department of surgery of the University of Minnesota Medical School. The course will be held on June 14–16, 1982 at the University of Minnesota. For further information contact: Office of Continuing Medical Education, University of Minnesota Hospital Box 293, 420 Delaware St. SE, Minneapolis, MN 55455; (612) 373-8012.

Clinical Application of Hyperbaric Oxygen

The 7th Annual Conference on the Clinical Application of Hyperbaric Oxygen is scheduled for June 9 to 11, 1982 at the Disneyland Hotel in Anaheim, California. Included in the conference will be plenary sessions on the use of hyperbaric oxygen in neurologic disorders and anaerobic infections. Original papers will be presented and there will be workshops and exhibits. For more information, write: Baromedical department, Memorial Hospital Medical Center, 2801 Atlantic Ave., Long Beach, CA 90801-1428, or call (213) 595-3613.

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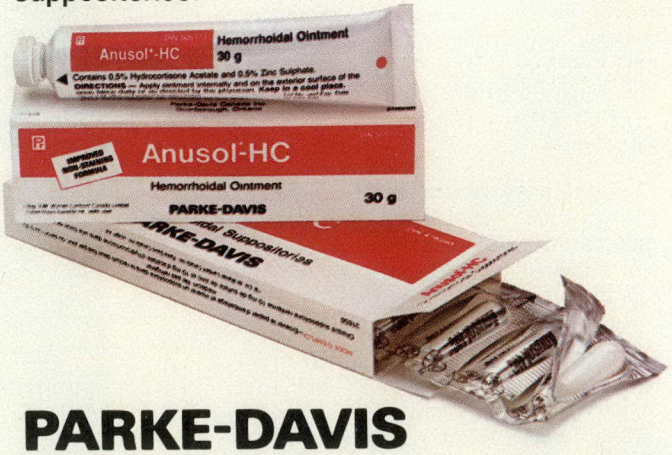

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Alkaline Reflux Gastritis: Roux-en-Y Diversion is Effective

DANIEL TASSÉ, MD, FRCS[C],* PIERRE GHOSN, MD,* MICHEL GAGNON, MD, FRCP[C],† FRANÇOIS MARTIN, MD, FRCP[C],† STEPHEN MORGAN, MD, FRCS[C],* PIERRE POITRAS, MD, FRCP[C]† AND DENIS BERNARD, MD, FRCS[C]*

Even though controversy persists over the physiopathology of alkaline reflux gastritis, Roux-en-Y diversion is a well-accepted surgical treatment and was used in 23 patients over a 6-year period at the Hôpital St-Luc in Montreal. Of the 23 patients, 21 had complete relief of symptoms. Objective criteria for the selection of patients who would benefit from operation are still to be defined. The authors discuss the physiopathology through an extensive review of the literature.

Bien que la physiopathologie de la gastrite alcaline soit encore incomplètement connue, la dérivation sur anse de Roux-en-Y donne d'excellents résultats cliniques. L'objet de cette publication est de rapporter notre expérience dans le traitement chirurgical de cette condition chez 23 patients opérés au cours des 6 dernières années. Une disparition complète des symptômes est observée dans 21 des cas. Des critères objectifs de sélection des patients candidats à une intervention chirurgicale n'ont pas encore été définis. Les mécanismes physiopathologiques de cette entité sont étudiés à travers une revue extensive de la littérature.

Epigastric pain and bilious vomiting are distressing symptoms frequently encountered after gastric surgery. Until recently, these symptoms were included in the postgastrectomy syndromes and were frequently attributed to a chronic afferent loop obstruction syndrome. Two decades ago, a new entity, "alkaline reflux gastritis", was first noted.[1,2] It has been further defined recently.[3,4] Although its physiopathology is not yet clearly understood, treatment by operative methods has gained wide acceptance in the literature.

The purpose of this report is to describe our experience with the surgical management of this syndrome in 23 patients over the past 6 years.

Patients and Methods

From 1974 to 1980, 23 patients (14 men, 9 women) with the diagnosis of alkaline reflux gastritis were admitted for operation. The mean age was 45.1 years (range from 23 to 89 years). The diagnosis was based on symptoms and radiologic, endoscopic and histologic findings.

The most frequent symptom was burning epigastric pain, unrelated to meals, that was found in 95.6% of the patients (Table I). The mean weight loss was 10 kg. Roentgenograms after barium ingestion appeared normal in 15 of 16 patients. Endoscopy demonstrated varying degrees of gastritis in all patients. Bile reflux of varying severity was also observed in all patients; in 16 it was severe with intense staining of the gastric mucosa.

The histologic findings in the antral mucosa in primary reflux gastritis were characterized by edema, congestion and a superficial inflammatory infiltrate (lymphoplasmacytic). In some there was a marked degree of diffuse interstitial fibrosis without evidence of intestinal metaplasia or glandular atrophy.

In secondary reflux gastritis there was edema, congestion, nonspecific inflammatory infiltration of the lamina propria with or without glandular atrophy and intestinal metaplasia but no interstitial fibrosis.

Eight of the patients had an intact pylorus, but only three had not had previous gastrointestinal surgery. Fifteen patients had secondary bile gastritis, following various surgical procedures on the stomach (Table II). The delay between the first operation and the occurrence of symptoms ranged from 1 month to 14 years. These patients had dumping syndrome and one had chronic pancreatitis. All patients had a trial of medical therapy with various drugs, before operation. A little relief of short duration was experienced in seven patients. In most the symptoms were unchanged and sometimes worsened by the medical regimen. The results obtained with different medications are shown in Table III.

Table I—Symptoms in 23 Patients with Alkaline Reflux Gastritis

Symptom	%
Pain	95.6
Bile vomiting	91.2
Diarrhea	39.1
Weight loss	30.0
Headache	26.0

Table II—Previous Operations

Primary gastritis (n = 8)	No.	Secondary gastritis (n = 15)	No.
Cholecystectomy	3	Parietal cell vagotomy	1
Cholecystectomy and transduodenal sphincteroplasty of Oddi	2	Vagotomy and antrectomy	7
		Billroth I	5
		Billroth II	2
No previous operation	3		
		Vagotomy and pyloroplasty	3
		Subtotal gastrectomy	2
		Gastrojejunostomy	1
		Esophagogastrectomy	1

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At surgery, 22 of the 23 patients had a Roux-en-Y diversion and one patient had a previous gastrojejunostomy taken down and then a vagotomy and pyloroplasty done. Additional procedures are listed in Table IV. There were no operative deaths. Two patients were lost to follow-up, while 21 were examined regularly for a mean period of 25.4 months (range from 4 to 62 months).

Eighteen were completely asymptomatic. One patient was relieved of pain and bilious vomiting, but still had the preoperative manifestations of a dumping syndrome and early satiety. This was attributed to a "small reservoir" following a previous subtotal gastrectomy. Two patients were considered as failures, not because of persistence of the same symptoms, but because of emergence of new and different complaints (diarrhea, parietal pain) after the operation. Two patients died in the follow-up period.

In the months following the operation, three patients had stenosis at the anastomosis; this had to be corrected surgically. Five patients showed signs of mild gastric stasis and did not require another operation.

Discussion

Alkaline reflux gastritis is a recently described syndrome that was previously confused with the chronic afferent loop syndrome. It is attributed to the effect of the duodenal contents, primarily the biliary acids, on the gastric mucosa.

The damaging effect of whole bile on gastric mucosa was first noted by Smith in 1914.[5] Interest in this effect was initiated in 1965 by Toye and Williams[6] who demonstrated that bile alone did not produce gastric mucosal injury while diversion of duodenal contents to the stomach produced progressive atrophic gastritis. Byers and Jordan[7] did not find any inflammatory change in isolated gastric mucosa explanted into the gallbladder. Menguy and Max[8] and Delaney and coworkers[9] noted the presence of gastritis when the gastric mucosa was bathed in bile and kept in continuity with the digestive tract. These changes were even greater when the mucosa was exposed to the whole intestinal content.[9]

Delaney and colleagues[10] reported that jejunal contents produced inflammatory changes in the gastric mucosa, but the changes were mild when pure bile or pure pancreatic juice was used.

Subsequently, the effect of biliary acids on gastric mucosa was studied extensively by Silen and Ritchie and others.[11-18] The theory that a mucosal barrier may be damaged by the biliary acids or other components was proposed. The mucosal damage causes a back-diffusion of H^+ ions into the mucosa and this induces the gastritic changes noted. The effect is more severe in the presence of H^+ ions and is maximum in the ischemic mucosa. Acute hemorrhagic gastritis can develop in these circumstances.[5,19-22]

In spite of these extensive studies, the pathophysiology of reflux gastritis is not clearly understood. Organic compounds other than bile, such as lysolecithin, have been incriminated,[23] but the responsible offending agent has not been positively identified.

Clinically the syndrome has been described by Du Plessis,[1,2] van Heerden and colleagues,[3] Bushkin and colleagues[24] and Miranda and associates;[25] it was clearly distinguished from the chronic afferent loop syndrome. Its characteristics are continuous burning pain not relieved by antacids and aggravated by meals. Vomiting has little or no effect on the pain. Vomiting occurs unpredictably and is not as projectile as in the chronic afferent loop syndrome; often the vomitus contains residuals of previous meals.[26] Some degree of malnutrition is present, but is not as severe as in the chronic afferent loop syndrome. No diagnostic test is pathognomonic and the diagnosis remains one of exclusion. However, in most cases, some degree of gastric flaccidity exists and analysis of gastric secretion demonstrates basal hypochlorhydria and minimal response to stimulation. A microcytic hypochromic anemia is usually present.

Endoscopic examination shows diffuse erythematous and friable mucosa that bleeds easily on contact. Areas of superficial erosions and atrophy are often present. Biliary reflux is always noted in the stomach and may be severe in some cases.

Biopsy specimens reveal a decrease in number of the chief and parietal cells, superficial erosions and atrophy of the mucosa and an inflammatory cell infiltration, predominantly of lymphocytes, in the gastric glands. Although this syndrome may be seen in those who do not undergo operation, it is usually encountered after a surgical procedure on the stomach, especially for duodenal ulcer. Its incidence is unknown, although it may be as high as 20%.[27,28] This does not coincide with the incidence of gastritis alone, which is encountered in 64% to 95% of cases[29] after operation for duodenal ulcer. In a recent study of 205 patients, 10 to 22 years after gastric surgery, Malchow and associates[30] found severe chronic gastritis in 40 (32%) of 125 patients after a Billroth I gastrectomy and in 28 (35%) of 80 patients after a Billroth II gastrectomy. Moderate changes were seen in 23.2% and 26%, respectively, in these two groups of patients. Loup and associates[31] observed two cases (0.9%) of reflux gastritis in 248 patients following vagotomy and pyloroplasty.

Conservative management of this condition has been deceiving. Antacids and antisecretory drugs have no effect on the symptoms. Cholestyramine was tried extensively, but little benefit was obtained[32] and it soon fell into disuse. The effect of total parenteral nutrition is incomplete and the symptoms recur with the resumption of oral alimentation.[33]

Table III—Results of Medical Treatment

Treatment	Improved	Worsened	Unchanged
Antacids	7	2	10
Cholestyramine	2	0	5
Metoclopramide	4	2	9
Cimetidine	4	2	7
Diet only	3	0	4
Sucralfate	0	2	0
Propantheline	0	0	1

Table IV—Surgical Procedures Associated with Roux-en-Y Diversion

Procedure	No.
Vagotomy and antrectomy	11
Revision of incomplete vagotomy	2
Revision of incomplete vagotomy and antrectomy	3
Revision of previous gastrojejunostomy	3
Antrectomy	1
Antiperistaltic interposition of a 10-cm segment*	2

*For associated severe dumping.

The reported results of surgical treatment have been conflicting. Some authors found that relief was incomplete.[34,35] However, most reports claim dramatic and nearly complete success after diversion of duodenal contents. Many techniques have been proposed: Henley loop interposition,[26] The Soupault operation,[26] the Tanner 19 procedure[28] and simple dismantling of gastroenterostomy. However, Roux-en-Y loop interposition of a 40- to 50-cm segment of intestine is the most frequently used procedure and the one that uniformly gives the best results. The long-term success rate with this operation varies between 65% and 100%.[36-41] Our own results are similar; 90.5% of our patients became asymptomatic.

Finally, we were particularly surprised to find severe stenosis of the gastrojejunal anastomosis in three patients after a Roux-en-Y procedure for alkaline reflux gastritis. These are the only instances of stenotic anastomosis in almost 400 gastric operations. This is due to the severe inflammatory changes encountered in the mucosa and submucosa of the stomach.

In conclusion, we have found that diversion of duodenal contents using a Roux-en-Y loop is a very satisfactory method of treating alkaline reflux gastritis.

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BOOK REVIEWS

continued from page 327

A substantial portion of the book is devoted to peptic ulcer disease. There is an excellent discussion on the natural history of bleeding peptic ulcers. The value of gastric lavage, antacids and cimetidine in the management of bleeding ulcers may be over emphasized. Surgical therapy is well discussed. The chapter on the pathophysiology of erosive gastritis and stress ulceration is informative. There is mention of therapeutic endoscopy, and the principles involved in laser therapy and photocoagulation are well summarized. The chapter on therapeutic angiography is excellently illustrated. Portal hypertension and bleeding varices are discussed in detail, but the chapter on the use of sclerotherapy is, on account of the timing of this publication, not an up-to-date "state of the art" review. "Miscellaneous" causes of gastrointestinal bleeding are covered in the concluding portion.

This book would be an extremely valuable addition to any medical library.

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SURGERY OF THE AORTA AND ITS BODY BRANCHES. Edited by John N. Bergan and James S.T. Yao. 632 pp. Illust. Grune & Stratton, Inc., New York; Academic Press Canada Limited, Don Mills, 1979. $56.25. ISBN 0-8089-1240-2.

This volume represents the papers presented at a symposium on surgery of the aorta. The editors, well-known vascular surgeons from Northwestern University Medical School in Chicago, have organized the book well. Although there are some areas of overlap because of multiple authorship, the text is readable and presents the information in an orderly and informative fashion.

continued on page 345

Bon Mot

J.L. SHUGAR, MD, FRCS[C]

The pressures of common usage shape and change the quality of words. Sometimes the changes are permanent. New words are born, and eventually their origins are forgotten or are not apparent. Who knows whether "gold bladder" or "very close veins" will eventually stick. Does what seems to be merely mispronunciation contain a kernel of truth, as in "minipause" — let it last just a short time, please — or does the dropping of a syllable really hide a forlorn hope of finding an "anti-inflation pill"? A group of patients can be "separated by sex" for purposes of analysis but what if the author of that study has them "separated *for* sex"? Can investigators carry out "an evaluation of several regiments of breast cancer"? Saved just in time, the March issue of this journal almost had an article on "open hearth surgery" — a cosy topic. How can you contradict your patient when she says that when she bends over, everything "goes out of kilt"? Perhaps it does. An elderly gentleman, who is worried about atheromatous arteries, asks to have his "serum castor oil" level checked. Can you deny him? Watch for changes in "Gray's Anatomy", which will describe a new anatomical entity — "the elbone" and the "appendic" for when only one is meant. In the future, broken bones will be fixed with "scrools". The hurt will be described by a colourful word — "paint". But watch out for pathology reports that say, according to an O.R. nurse, "Dr. Armour, your pants are pressed." That's enough to give you a "migrant" headache.

Can you help me, especially you over-60 types, clear up a mystery? What is a wartime clamp?

My thanks to my mother, my patients, David Woods, Peter Morgan, Gillian Pancirov and Ph. d'Entremont for their contributions.

"Bon Mot" will appear in the Journal from time to time as material becomes available. We earnestly solicit your contributions.

SESAP III Critique

ITEM 626

The possibility that this patient has a testicular tumor must be ruled out. Baseline studies of chest roentgenograms, an intravenous pyelogram, and levels of human chorionic gonadotropin (HCG) and α-fetoprotein should be obtained. If the presence of a tumour is suspected, an inguinal incision with orchiectomy should be done. Treatment with antibiotics is not indicated unless pain and pyuria point to epididymitis. Needle biopsy and scrotal incision are contraindicated because of the risk of local dissemination of the tumour, which changes the route of metastases to include the inguinal lymph nodes. The spermatic cord should be occluded with a noncrushing clamp while the testis is being examined. If a tumor is suspected on visualization and palpation, the orchiectomy should be performed, but not a biopsy.

A

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RHÔNE-POULENC

PAAB CCPP MEMBER PMAC

Maquet Osteotomy for Chondromalacia Patellae: Avoiding the Pitfalls

A. HADJIPAVLOU, MD, M SC, FRCS[C], FACS, H. HELMY, MD, FRCS[C], P. DUBRAVCIK, MD, FRCS[C], L. HELLER, MD, FRCS[C], FACS AND M. KERNER, MD, FRCS[C]

The authors' modification of the classic Maquet procedure of anterior displacement of the tibial tubercle allows effective decompression of the patellofemoral joint. Strict adherence to the modified technique has prevented all major complications. Forty-four patients underwent this procedure. They were divided into a retrospective group of 20 and a prospective group of 24. Nine patients in the prospective group had the modified Maquet procedure following a failed Hauser operation. All had an excellent result due to decrease in the patellofemoral compression force. The modified Maquet procedure was also valuable in those who had an extensor lag following patellectomy (two patients), failed proximal realignment (three patients) and failed patellar shaving procedures (three patients).

Les modifications apportées par les auteurs à l'opération classique de Maquet par déplacement antérieur du tubercule du tibia, permettent une décompression efficace de l'articulation fémoro-patellaire. En s'en tenant de façon stricte à cette technique modifiée, on a pu prévenir toutes les complications majeures. Quarante-quatre patients ont subi cette intervention. Ils ont été divisés en groupe rétrospectif de 20 sujets et en groupe prospectif de 24. Neuf patients du groupe prospectif ont eu l'opération modifiée de Maquet suite à un échec de l'opération de Hauser. Tous ont bénéficié d'excellents résultats grâce à la diminution de la force de compression de l'articulation fémoro-patellaire. L'opération modifiée de Maquet s'est aussi révélée utile chez les sujets présentant un retard d'extension à la suite d'une patellectomie (deux patients), dans les cas d'échec d'un réalignement proximal (trois patients), et lors de l'échec d'une opération de planage de la rotule (trois patients).

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The treatment of patellar chondromalacia is one of the most controversial issues in orthopedic surgery. The term patellar chondromalacia implies damage to the articular cartilage resulting in softening, fissuring or fragmentation. The knee may be asymptomatic or transiently symptomatic with crepitus and pain over its anterior aspect.[1-4] When the symptoms become clinically important or debilitating, conservative management should be used before surgical intervention.

Many surgical procedures have been considered for correcting each of the underlying causes of degeneration of the patellofemoral joint. These include the realignment procedures (proximal[5,6] and distal[7-9]). Soft tissue lining for patellar arthroplasty[10] and excision of the damaged articular cartilage (shaving, chondrectomy,[7,11-14] hemipatellectomy [Sullivan J: personal communication] or patellectomy[11,15-17]) have also been tried. More advanced cases of joint degeneration have also been treated by total replacement of the patella by a prosthesis.[18,19]

Patellectomy may reduce pain at the patellofemoral joint, but retention of the patella is functionally superior as it enhances the power and function of the quadriceps mechanism.[20] Nevertheless, there are occasions when patellectomy is necessary because of persistant pain. Maquet[21] and Bandi[22] have described displacement of the tibial tubercle anteriorly to allow decompression of the patellofemoral joint, an attractive concept in view of the poor results of other procedures.

Patients

We used the Maquet[21] procedure in 44 patients between January 1976 and February 1981. The patients ranged in age from 18 to 60 years (mean 27.1 years). There were 20 men and 24 women divided into two groups. In group 1 we carried out a retrospective study of 20 consecutive patients (11 men, 9 women). Follow-up ranged from 27 to 54 months (mean 35.3 months). Group 2 comprised 24 patients (9 men and 15 women) studied prospectively. Follow-up ranged from 6 to 26 months (mean 18.8 months).

The diagnosis of chondromalacia was made by means of arthrotomy or arthroscopic examination. The chondromalacic lesion in our series was grade 3 or 4 according to the Outerbridge classification.[23]

Indications for Operation

The Maquet procedure was indicated for the following problems: (a) chondromalacia due to chronic malalignment (25 patients), (b) severe chondromalacia of the patella secondary to trauma (12 patients), (c) osteoarthritis of the patellofemoral joint (5 patients) and (d) extensor lag following patellectomy (2 patients).

Of the 25 patients with malalignment, 13 knees had failed to respond to conservative therapy and 12 knees to previous surgical realignment procedures (three proximal and nine distal Hauser realignments). Of the 12 patients with patellar chondromalacia due to patellar trauma, conservative therapy in 8 had failed, and in the other 4, previous surgical procedures (shaving in 3 patients, hemipatellectomy in 1) had failed.

Results

Criteria used to determine the results of the procedure included: (a) the degree of pain at the patellofemoral joint, (b) the range of motion of the knee and (c) the functional capacity of the knee with respect to the activities of daily living (walking stairs, running, sitting and kneeling).

Four groups emerged: no improvement, improvement, marked improvement and asymptomatic knee.

Patients with improved knees could walk longer distances without pain than before operation. The range of motion was normal with mild pain on walking up and down stairs or when kneeling. There was increased pain only when running.

In patients rated as markedly improved, there was only occasional pain and no disability when walking and only infrequent mild pain after long periods of fast walking, climbing or descending stairs or running.

Asymptomatic patients had no pain and could walk up and down stairs, kneel and run and had a normal range of motion of the knee.

Table I sets forth the results in the two groups of patients. Patients with extensor lag were markedly improved. There were no cases of infection or peroneal nerve palsy in the prospective group.

Complications (Table II)

The complications were attributed to certain predictable difficulties and compromised the overall results in our retrospective group of patients. Most of these complications arose within the first 2 months after operation. Of the three patients with nerve palsy, two had completely recovered at 6 months after operation and one had partially recovered but had residual causalgia. In one case of fracture of the distal end of the osteotomized bone, there was penetration of the skin and osteomyelitis, which was finally cured by sequestrectomy. Of two other cases of osteomyelitis with skin necrosis, one was cured, but in the other the site was still draining at the time of follow-up 32 months after operation.

Table I—Results

	Group, no.	
Grading	1 (n = 20)	2 (n = 24)
No improvement	4	0
Improvement	6	1
Marked improvement	5	10
Asymptomatic	5	13

Table II—Complications

	Group, no.	
Complication	1	2
Fracture at distal end of elevated tongue of bone	5	0
Fracture at unsupported proximal end of elevated tongue of bone	1	0
Skin necrosis with osteomyelitis	3	0
Neuroma of infrapatellar nerve	2	0
Common peroneal nerve palsy	3	0
Pain at osteotomy site	5	2
Lateral patellar tilt	0	1
Hemiarthrosis	1	0

Although our technique closely resembles that of Maquet and Bandi, strict adherence to certain minute details previously ignored reduced considerably the number of complications and improved the results in the subsequent 24 patients of the prospective group.

Operative Technique in Prospective Group

Tibial Osteotomy

The plane of the tibial osteotomy is critical. At the proximal end the osteotomy is in the coronal plane. At about 6 cm distal to the tibial tubercle the osteotomy changes its plane and becomes parallel to the anteromedial surface of the tibia (Fig. 1); it is flat and pliable. The original osteotomy resulted in a triangular cross-section of bone at its distal segment and therefore allowed a fracture to occur more readily (Fig. 2). The shape of the tongue of bone is flat and rectangular measuring approximately 10 × 1.5 × 0.5 cm (Fig. 3).

Bone Graft

The bone graft that supports the anteriorly displaced tibial tubercle (Fig. 3) is obtained from the bone bank or the patient's iliac crest (Figs. 4 and 5). It is important that the two longer surfaces of the bone graft be parallel to the two surfaces of the tibial osteotomy within which the graft is to be wedged. The shape of the graft, when viewed from the lateral side, has the appearance of a truncated right triangle. The proximal end or short side is 1.5 cm to 2 cm thick and tapers distally to 0.8 cm to 1 cm. The longer side measures 5 cm to 6 cm lying flat against the osteotomized surface of the tibial diaphysis. The hypotenuse lies flat under the raised tongue of bone; the distal gap is packed with cancellous bone. We have maintained the anterior elevation of 1.5 cm to 2 cm since we believe, as shown by Burke (personal communication, 1979) that decompression of the patellofemoral joint occurs effectively with this amount of displacement.

Incision

Two incisions are used (Fig. 6). One is a long incision over the anteromedial surface of the tibia, extending from the level of the tibial tubercle to the middle third of the leg. This allows the tibial tubercle to be

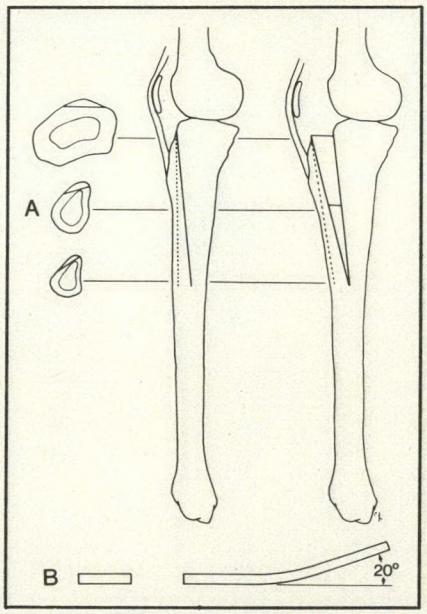

FIG. 1—Plane of osteotomy of tibia. (A) Correct plane of osteotomy with large flat surface to prevent fracture of elevated tongue of bone. (B) Flat cut is more pliable than triangular cut.

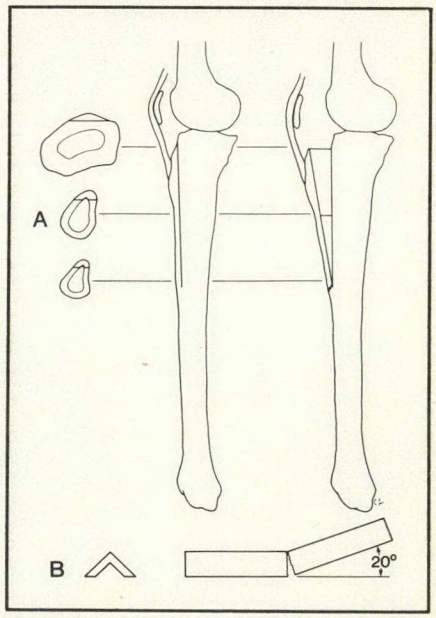

FIG. 2—Plane of osteotomy of tibia. (A) Incorrect triangular configuration. (B) Triangular tongue of bone has no elastic properties.

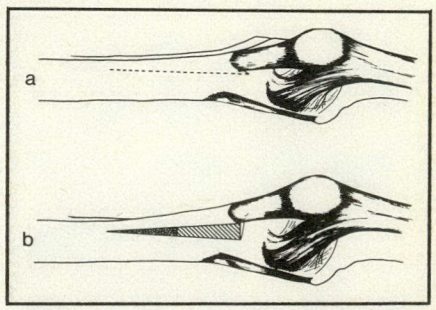

FIG. 3—Lateral view of osteotomized and anteriorly displaced tibial tubercle (a) before, (b) after.

elevated. A large medial skin flap is created by dissecting the deep subareolar space and the tibial crest is not denuded from the overlying soft tissues. A second lateral parapatellar incision, 5 cm long, is made for exploration of the knee joint; this allows good visualization of the joint and bypasses the infrapatellar branch of the saphenous nerve, thus avoiding damage that could result in formation of a neuroma. The lateral retinaculum is released under direct vision and the genicular artery is ligated. Through a lateral incision, the fascia of the anterior compartment can be released with a straight Smillie meniscectomy knife. Care must be taken not to denude the tibial crest.

Discussion

By strict attention to details, the results in our series of patients were markedly improved in those studied prospectively, by preventing troublesome technical complications.

● Skin necrosis can be eliminated by using a long anteromedial incision of the subcutaneous tibial surface. This contributed to better skin closure without tension after the tibial tubercle was elevated, even to the extent of 2.5 cm.

● Two patients in our retrospective series had a troublesome neuroma of the infrapatellar branch of the saphenous nerve. The infrapatellar nerve crosses the medial aspect of the knee joint only slightly distal to the medial joint line. Damage to this nerve may be avoided by using a lateral parapatellar incision for the knee exploration and by beginning the long anteromedial skin incision at the level of the tibial tubercle.

● Through the lateral parapatellar incision, the lateral retinaculum of the knee joint can be released, thereby preventing the tethering effect of the knee joint capsule on the patella (Fig. 7).

● Postoperative hematoma and hemiarthrosis may be averted by tourniquet release before closing the wound and by ligating the lateral inferior genicular artery if severed. Hemovac drainage is used.

● The three instances of peroneal nerve palsy in the retrospective group were attributed to excessive traction on, and displacement of, the anterior fascia of the leg caused by the anterior displacement of the tibial tubercle (Fig. 8). In one case, the common peroneal nerve was found to be displaced anteriorly and angulated when explored. One can prevent this complication by releasing the lateral fascia; this may also prevent anterior compartment syndrome.[14] Fig. 9 shows the path of the common peroneal nerve. The proximal cut shows the nerve at the posterolateral surface of the head of the fibula between fascia and bone. The middle cut shows the nerve anterior to the neck of the fibula and the distal cut shows the nerve remaining anterior to the proximal shaft of the fibula. In the middle and distal cuts the nerve is not lying between fascia and bone.

The suggested shape of the bone graft and osteotomy of the tibia allow a good total contact, a snug fit and no pivoting of the graft when it is wedged between the anteriorly displaced tongue and the remaining tibia. No transfixion screw is necessary. The graft should extend to the bony attachment of the patellar tendon in order to support the tongue fully and

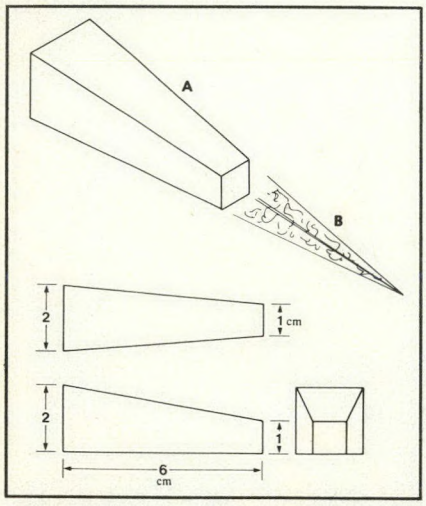

FIG. 4—Bone graft. (A) Graft for proximal osteotomy area. (B) Cancellous bone for distal portion of osteotomy site.

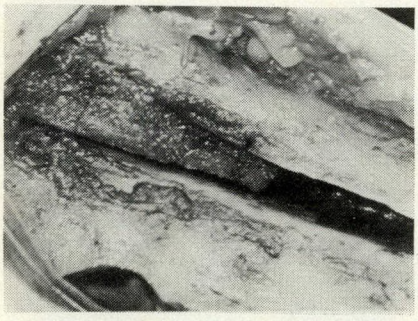

FIG. 5—Bone graft wedged into osteotomy site.

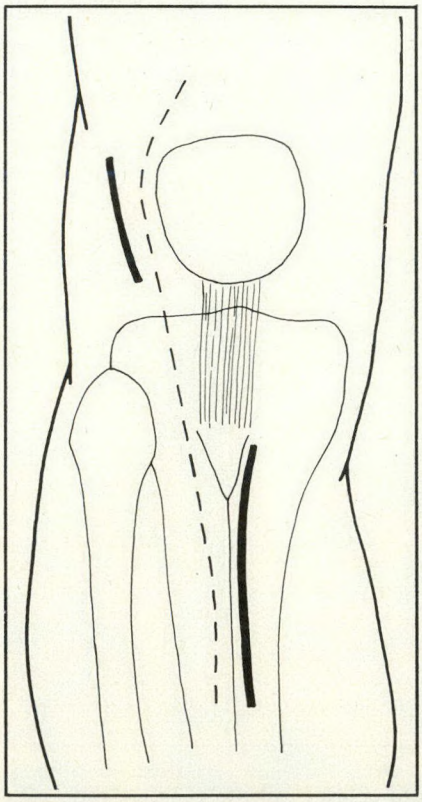

FIG. 6—Incisions. Dotted line shows site of lateral retinaculum release through lateral parapatellar incision.

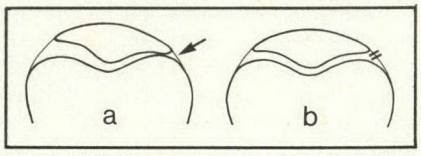

FIG. 7—Release of lateral retinaculum prevents patellar tilt.

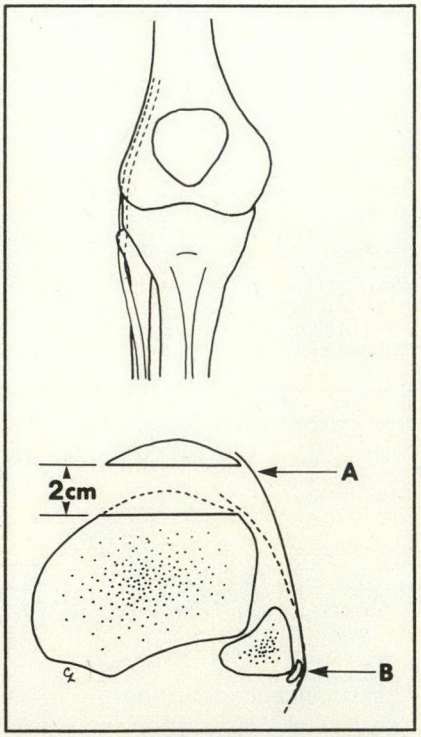

FIG. 8—Anterior displacement of tibial tubercle exerts excessive tension on fascia (A) resulting in traction or compression of peroneal nerve against fibula (B), or both.

so prevent it breaking when it receives a direct blow from a fall. In the prospective study, lateral tilt of the patella occurred in one case because the lateral release was not done at the time of the original Maquet procedure. This was carried out later and corrected. Although follow-up in some cases in our retrospective group was only 6 months, even this short follow-up demonstrated that there are difficulties that can be prevented. The complications in this group arose within 1 to 2 months after operation and the outcome of the procedure was directly related to these complications.

In the retrospective group, the use of banked bone eliminated the need for an iliac incision; there was excellent incorporation of the graft. All cases of distal and proximal realignment fell within the markedly improved or asymptomatic group and the shaving or hemipatellectomy cases within the improved group.

The Maquet osteotomy is indicated for patients with patellofemoral arthritis, chondromalacia of the patella secondary to direct patellar trauma and failed surgical intervention. We also use it for severe patellar chondromalacia with only a mild malalignment that does not require tibial tubercle transplantation to correct the Q angle.

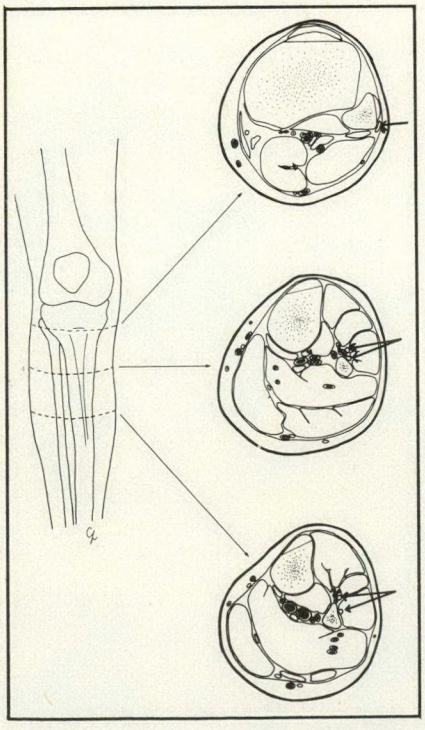

FIG. 9—Common peroneal nerve (arrows) at head of fibula (proximal cut), at neck of fibula (middle cut) and at shaft of fibula (distal cut).

We thank Mr. David Saxe, department of photography, Jewish General Hospital and Miss Christine Lalonde, Lady Davis Institute, for their work on the illustrations and Carmela Iuculano and Barbara Dubravcik for preparing the manuscript.

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BOOK REVIEW\$

continued from page 339

This book is divided into six sections. The first section deals with evaluation and investigation of patients being considered for aortic surgery. It includes a discussion on physiologic changes that occur during aortic cross-clamping.

The second section deals with principles of surgery of the thoracic and abdominal aorta, including some special chapters on the types of bypass systems used during thoracic aortic cross-clamping, "the small aorta syndrome" and the problem of colonic ischemia following surgery on the abdominal aorta. There is excellent discussion on the abdominal aortic aneurysm, from the standpoint of elective and emergency surgery of ruptured aneurysms.

Aortoiliac occlusive disease forms the basis for the third section, which includes a discussion on importance, extra-anatomic bypasses and management of aortocaval problems.

There follow sections on renal vascular hypertension and mesenteric ischemia, and discussions of the roles of percutaneous and iliac occlusive disease.

The last section deals with special problems in aortic surgery and considers such topics as splanchnic artery aneurysms, surgery for infected aortic grafts, spinal cord ischemia after aortic surgery and aortoenteric fistulas.

This book provides an exhaustive outline of the aorta and its surgical problems. One would have hoped that some of these areas would have been dealt with in greater detail, but overall, this book serves as a good guide to current thoughts in the management of aortic diseases and will be of value to students of aortic disease, be they housestaff or vascular surgeons.

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As the treatment of the patient in shock has progressed in recent years, various organ systems have, in turn, been the "weak link" limiting recovery. The deterioration often becomes apparent first in the kidney where disruption of cellular metabolism causes acute tubular swelling with resulting oliguria.

Soon, the pulmonary vasculature begins to exhibit a pattern of interstitial oedema, pooling of blood in the stagnant microcirculation and aggregation of the formed elements of the blood. Diffusion of oxygen from the alveoli is greatly decreased, commonly resulting in arteriovenous shunting – a phenomenon in which blood passes through the lung without oxygenation.

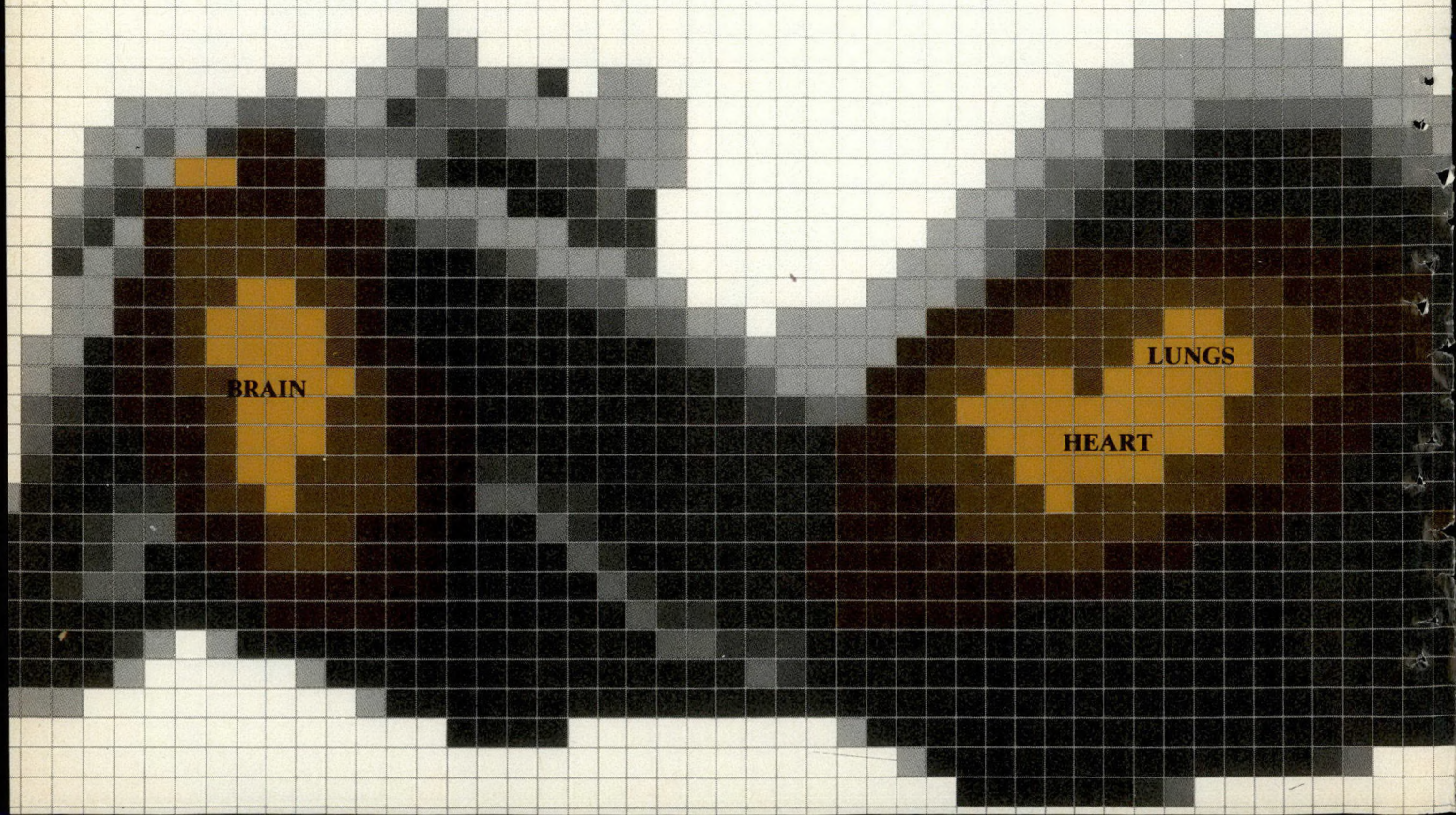
If the shock state continues, lysosomal enzymes in the anoxic pancreas trigger the release of myocardial depressant factor (MDF), a vasoactive peptide which can cause further deterioration of the microcirculation.

Although cerebral blood flow is maintained early in shock at the expense of the other organs, this state of adequate perfusion does not persist for long. Relatively soon, available oxygen becomes critically low, and the brain, too, turns to anaerobic metabolism. Lactate acid levels rise and fluids leak into the intestitial spaces. Continued oxygen deprivation threatens both the cellular integrity of the brain and the central nervous system function.

Once shock has progressed beyond its early stage, fluid administration alone is usu-

severe shock threatens each link in the body's chain of organ systems

Solu-Medrol exerts a protective effect on the cell to preserve tissue function and increase survival rates



ally not sufficient to reverse the haemodynamic and cellular disruptions.

Solu-Medrol, administered in pharmacologic doses as part of a comprehensive treatment regimen, can reduce vasoconstriction and increase tissue perfusion. The multiphasic activity of Solu-Medrol helps restore haemodynamic balance, preserve lysosome and cell membranes, and protect cellular and intracellular structures and function.

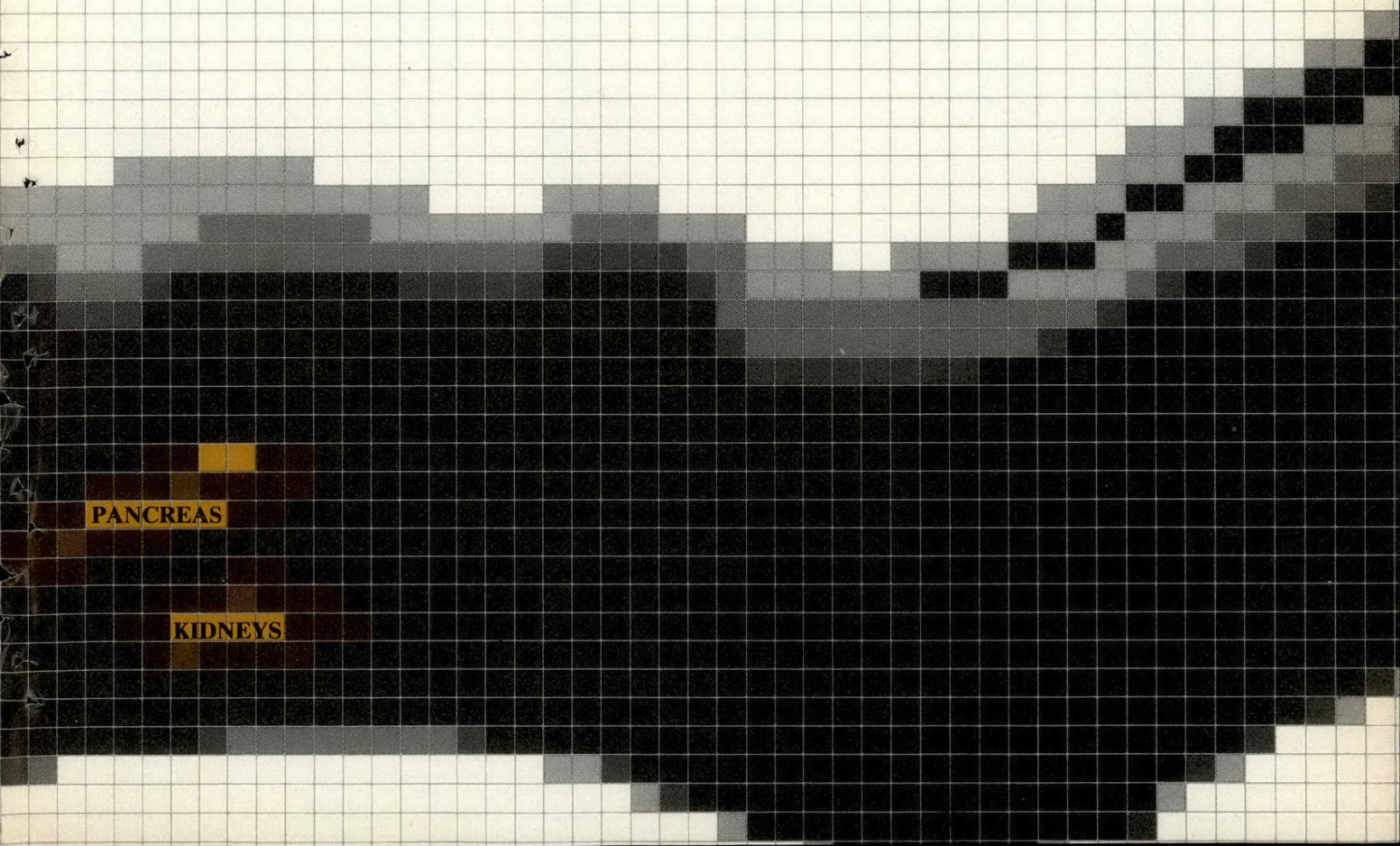
The actions of Solu-Medrol affect all of the body's systems:

- **In the kidney, increased perfusion reverses the anoxic state and improves organ function as evidenced clinically by an increase in urine output.[1]**
- **Improvement in the pulmonary vasculature is demonstrated by a reduction in shunting and an increase in oxygen consumption.[2]**
- **The affinity of haemoglobin for oxygen is reduced permitting increased delivery of oxygen by the blood to the tissues.[3]**
- **A positive inotropic effect is exerted on the heart further increasing the flow of blood.[4]**
- **Pancreatic lysosomes are stabilized halting the production of MDF.[5]**
- **Capillary membranes in the brain are stabilized, and capillary permeability reduced.[6]**

Through its network of protective actions, Solu-Medrol strengthens the chain of organ systems and increases the shock patient's chances of survival.

Solu-Medrol

Upjohn CE 1376.1



CORRESPONDENCE

Surgery for Morbid Obesity

To the editors.—The "editorial" of Freeman and Burchett on page 247 of this issue requires comment. It does not actually tell "where we are going" in the surgery for morbid obesity. Indeed our follow-up is comprehensive, with charts of patients who undergo jejunoileal bypass and gastric partitioning each having separate drawers in our filing-cabinets, and summarized in loose-leaf books. Preoperatively we explain to the patient that lifelong medical surveillance is mandatory. If patients do not keep appointments, a letter is sent, followed by a telephone call; some of the patients who had to be thus retrieved had the best results.

One patient who had a jejunoileal bypass has moved and is now under the care of another surgeon well known in this field. One underwent reversal elsewhere as stated in our paper and the remaining 62 (not 61) jejunoileal bypass patients are well and being followed up by us. One patient who had gastric partitioning will not return for follow-up, and we are only aware of her state through her friends who have undergone the procedure.

Our graphs are intended as a simple means of displaying the data: weight loss is shown on the left, mean percent loss in body weight on the right, with the important standard deviation from the mean and number of patients at each point of follow-up being included. The follow-up (in months), to the time the manuscript was submitted, is shown on the bottom of the graphs, and is obviously not long term in the gastric partitioning group. In the jejunoileal bypass groups, with the measurements and technique outlined in the paper, more than 80% of patients maintain a weight loss greater than 25% of body weight, but 100% have weight loss of more than 15% of body weight.

Surgeons who have never performed jejunoileal bypass tend to decry this procedure, and we have emphasized its hazards, but Payne of Los Angeles with more than 20 years' experience,[1] Gourlay of Vancouver with 535 intestinal bypass patients over 9 years,[1] and Buchwald[2] after operating on 800 patients have all found this to be an acceptable procedure. Because the post-bypass course demands the surgeon's availability, many have switched to gastric procedures. Serious liver problems have been rare in non-alcoholics. One of us (M.A.), together with the two pathologists acknowledged at the end of our paper, has continued to follow-up the condition of the livers in these patients and this is the topic of another paper. We have found the decrease in stool frequency to agree with that noted by MacLean in his review;[3] diarrhea is rare in our patients after 18

continued on page 351

Fate of Omental Graft after Revascularization of the Heart

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Three patients who had had epicardiectomy, and internal mammary artery and omental grafts implanted as part of myocardial revascularization, recently underwent a second operation for coronary artery bypass grafting. All internal mammary artery implants were patent and appeared to revascularize the myocardium distal to the occluded segment of the coronary artery, as determined by angiography. Although adhesions were present at operation, there was no evidence of the free omental graft in two patients. The pedicled omental graft was present and viable in the remaining patient, but did not appear to have vascular communication with the epicardiectomized myocardium.

Trois patients qui avaient eu une épicardiectomie, avec une greffe d'artère thoracique interne et greffe épiploïque comme moyen de revascularisation du myocarde, ont dû subir récemment une seconde opération, soit un pontage aorto-coronarien. Les greffes d'artères thoraciques internes étaient toutes perméables et paraissaient, à l'angiographie, avoir revascularisé la partie du myocarde distale au segment oblitéré de l'artère coronaire. Bien que des adhérences aient été visibles à l'opération, chez deux patients il n'y avait aucun indice de perméabilité des greffes épiploïques. Chez le troisième patient le pédicule de la greffe épiploïque était présent et viable, mais ne semblait pas avoir de communication vasculaire avec le myocarde épicardiectomisé.

The objective of this paper is to report the findings at reoperation of the fate of the omental graft placed in patients who had had internal mammary artery implantation, epicardiectomy and omental grafting and who underwent coronary artery bypass grafting because of recurrent angina.

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Case Reports

Case 1

A 60-year-old man had undergone left internal mammary artery implantation, epicardiectomy and free omental grafting 8 years before admission. He presented with incapacitating angina. Coronary angiography revealed severe triple-vessel coronary artery disease and a patent internal mammary artery implant without the formation of collateral vessels via the free omental graft (Fig. 1). At operation, adhesions were found between the pericardium and the epicardium without visual and microscopic evidence of the presence of the omental graft. Triple-vessel coronary artery bypass grafting was performed; recovery was smooth.

Case 2

A 64-year-old man had had right and left internal mammary artery implants, epicardiectomy and free omental grafting 9½ years previously. On admission he was suffering from unstable angina. Cardiac catheterization revealed triple-vessel coronary artery disease. Both internal mammary artery implants were patent but there was no evidence of collateral circulation through the free omental graft (Fig. 2). The operative findings were similar to those in case 1.

Case 3

Ten years before admission, a 63-year-old farmer had undergone left internal mammary artery implantation, epicardiectomy and pedicled omental grafting. He presented with angina refractory to medical therapy. Coronary angiographic examination revealed triple-vessel coronary artery disease and a patent internal mammary artery implant. There was no evidence of collateral circulation by way of the omental graft (Fig. 3). At operation, the pedicled omental graft was found to be viable. It occupied a large space in the posterolateral and inferior pericardial sac. The graft was easily peeled away from the epicardium. Through-and-through left ventricular myocardial biopsy was performed in an area where the omental

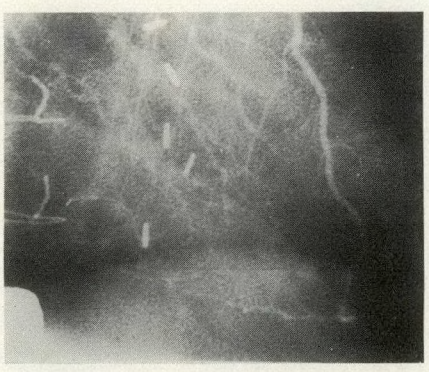

FIG. 1—Case 1. Coronary angiogram gives no evidence of collateral circulation between heart and omental grafts.

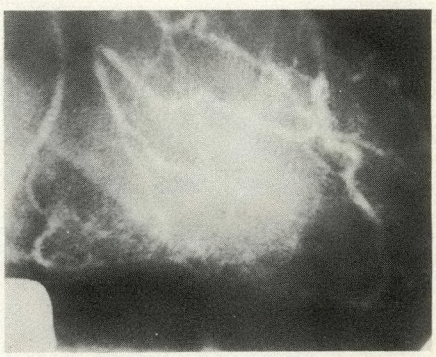

FIG. 2—Case 2. Coronary angiogram shows no evidence of collateral circulation between heart and omental grafts.

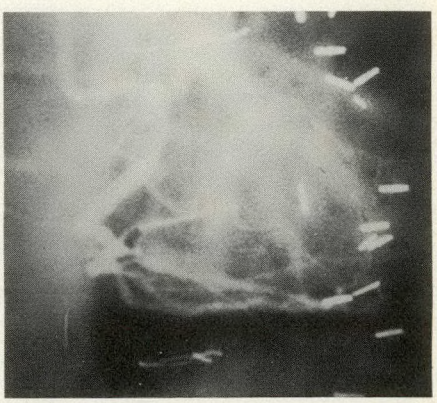

FIG. 3—Case 3. Coronary angiogram gives no evidence of collateral circulation between heart and omental grafts.

graft was intact and appeared to communicate with the epicardium. Microscopic examination revealed no evidence of increased collateral circulation or communication with the omental graft. After triple-vessel coronary artery bypass grafting the patient made an uncomplicated recovery.

Discussion

O'Shaugnessy[1] in 1936 first used the greater omentum attached to its abdominal blood supply to revascularize the ischemic myocardium. This was followed by extensive work by Vineberg and associates[2-4] on the free omental graft as an adjunct to myocardial revascularization with the internal mammary artery implant. Using the dog as the model, Vineberg and his coworkers[5,6] demonstrated that in the ameroid constricted coronary artery, collaterals developed between the ischemic epicardiectomized heart, the free omental graft and the surrounding tissues. Free omental strips were successfully implanted into the walls of both ventricles in animals.[7] Vineberg and associates,[5] in an addendum to their manuscript, reported that the implanted omentum would fill with dye when either coronary artery was injected in a patient who died 18 months after operation.

For whatever reasons, coronary artery bypass grafting has recently gained popularity in the treatment of ischemic heart disease, whereas the free and pedicled omental grafting, epicardiectomy and the internal mammary artery implantation are infrequently used. Yet several clinical reports[8-10] attest to the efficacy of internal mammary artery implantation in revascularizing the human heart. Similarly, epicardiectomy and free omental grafting have been shown to increase collateral flow in the ischemic hearts of dogs; collateral channels and viable omental grafts have been demonstrated at 6 months after operation.[11]

Little is known about the fate of the omental graft in the human heart, the degree of vascularity created by epicardiectomy and omental grafting, and the technical difficulties that might be encountered if coronary artery bypass grafting is contemplated in such patients. The omentum was not apparent in both patients who had received a free omental graft, as determined by lack of collateral flow at angiography, lack of bleeding during operation and by light microscopy of the myocardial biopsy specimen. The pedicled omental graft, on the other hand, was found to be present and viable at time of operation, occupying a large space in the opened pericardium. It was peeled away from the epicardium without difficulty and the myocardial biopsy specimen did not show invasion of the epicardium by omental vessels, nor was there evidence of increased vascularity. By light microscopic examination the epicardial layer of these three hearts could not be distinguished from another heart that did not have epicardiectomy or omental grafting. In all three patients the internal mammary artery implants were carefully preserved.[12] The bypass surgery was performed without technical difficulties.

One should be cautious in drawing conclusions based on three patients, when animal work would appear to support the concept of omental grafting in addition to internal mammary artery implantation and epicardiectomy. The difference between our patients and the animal model could be due to species differences or techniques of surgery, or both. That these patients had internal mammary artery implantation, epicardiectomy and omental grafting did not preclude subsequent coronary artery bypass grafting. The technical aspects of the procedure did not differ from routine bypass grafting reoperations, except that the internal mammary artery had to be snared during cardioplegic arrest[12] in order to prevent noncoronary collateral flow from washing off the cardioplegic solution. Also, the internal mammary artery is a valuable source of blood in these ischemic hearts and care must be taken to preserve it.

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months; then it occurs only after excessively greasy meals. Freeman and Burchett do not give the reference for diarrhea persisting after 1 year in 20% and being the main cause for reversal. They give no references for any of their statements.

For gastric partitioning we did not use the technique of Pace, but chose our outlet on the greater curvature side, where future procedures would be more accessible. The first 56 patients who did not have reinforcement of the outlet are not all failures; the data and standard deviations indicate that adequate weight loss occurred in a proportion. The "non-reinforced" group of 56 patients continue to show weight loss that is greater than 25% in 18 patients, with a maximum loss in body weight at 36 months of 36%; weight loss is greater than 20% in 25 of the 56 patients and greater than 15% in 28, which we regard as unacceptable. As noted in the paper, four patients returned to their preoperative weight. However, we are suspicious that many of the "non-reinforced" patients who have lost significant amounts of weight have been able to maintain this through their own effort after an initial substantial weight loss. The reoperation to reinforce the outlet is usually not a major undertaking.

We agree that the circumferential imbricating suture "does not guarantee perfect results". However, in the recent report by Gomez,[4] published 2 months after we submitted our manuscript, using the identical technique, there was a mean weight loss of 32% at 12 and 18 months, and 33% at 24 months. He had only one postoperative death in approximately 200 patients (0.5%), from cardiac cause.[4] The patients with outlet reinforcement continue to vomit if they attempt to overeat. Patient compliance is directly related to the time in explanation that anyone who undertakes this surgery must spend with the patient.

Freeman and Burchett do not give a reference for the 4% mortality with reoperation, and do not indicate if this is a personal experience.

A properly reinforced stoma should not enlarge, unless the suture is broken. We do not concur that a patient spitting up saliva and feeding via a jejunostomy for 6 months or longer is acceptable; moreover, a stricture may become tighter. We suggest that dilatation by a Gruntzig balloon catheter inserted through a pediatric gastroscope be attempted. Reoperation in our experience may be simple or arduous. We disagree that failures are those who have lost more than 15% of their initial weight. We believe that loss of less than 25% of body weight should be regarded as a failure. If Freeman and Burchett's 15% is used, 50% of our patients with "unreinforced" partitions and 100% of those with "reinforced" partitions have lost adequate weight.

In markedly obese patients who present at weight-loss clinics, sustained weight loss has been reported in 2% at 2 years,[5] and there is a high rate of recidivism on long-term follow-up, with an ultimate weight loss of less than 1%.[6]

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Claforan® IM/IV
(cefotaxime sodium)

ANTIBIOTIC

ACTION
In vitro studies indicate that the bacterial action of CLAFORAN (cefotaxime sodium), a semisynthetic cephalosporin antibiotic, results from inhibition of cell wall synthesis.

INDICATIONS AND CLINICAL USES
CLAFORAN (cefotaxime sodium) may be indicated for the treatment of infections caused by susceptible strains of the designated micro-organisms in the diseases listed below:

Lower respiratory tract infections: pneumonia and lung abscess caused by *Streptococcus pneumoniae* (formerly *Diplococcus pneumoniae),* other *Streptococci* (excluding *Enterococci,* e.g., *S. faecalis), Staphylococcus aureus* (penicillinase and non-penicillinase producing), *Escherichia coli, Hemophilus influenzae,* (including ampicillin-resistant strains), and unspecified *Klebsiella* species.

Urinary tract infections: caused by *Escherichia coli,* unspecified *Klebsiella* species (including *K. pneumoniae), Proteus mirabilis,* indole-positive *Proteus, Serratia marcescens* and *Staphylococcus epidermidis.* Also, uncomplicated gonorrhea caused by *N. gonorrhoeae* including penicillin-resistant strains.

Bacteremia/Septicemia: caused by *Escherichia coli,* unspecified *Klebsiella* strains and *Serratia marcescens.*

Skin infections: caused by *Staphylococcus aureus* (penicillinase and non-penicillinase producing), *S. epidermidis,* Group A *Streptococci, Escherichia coli, Proteus mirabilis* and indole-positive *Proteus.*

Intra-abdominal infections: caused by *Escherichia coli,* and unspecified *Klebsiella* species.

Gynecological infections: including pelvic inflammatory disease, endometritis and pelvic cellulitis caused by *E. coli,* Group A *Streptococci* and *Staphylococcus epidermidis,* anaerobic bacteria including unspecified *Peptococcus* and *Peptostreptococcus* strains and some strains of *Bacteroides fragilis.* In several cases, although clinical cures were achieved, bacteriological follow-up was not available.

Clinical experience with CLAFORAN in anaerobic infections is limited. CLAFORAN has been used with some success in wound and intra-abdominal infections against some strains of unidentified *Bacteroides* and anaerobic cocci.

CLAFORAN has been shown to be active against some strains of *Pseudomonas.*

In the treatment of infections encountered in immunosuppressed and granulocytopenic patients, results of therapy with cefotaxime have not been impressive.

CLAFORAN should not be considered in the treatment of enterococcal infections, i.e. *Strep. faecalis.*

Specimens for bacteriologic culture should be obtained prior to therapy in order to isolate and identify the causative organisms and to determine their susceptibilities to CLAFORAN. Therapy may be instituted before results of susceptibility studies are known; antibiotic treatment should be re-evaluated once these results become available.

CONTRAINDICATIONS
CLAFORAN is contraindicated in patients who have shown hypersensitivity to cefotaxime sodium, the cephalosporin, or the penicillin groups of antibiotics.

WARNINGS
Before therapy with CLAFORAN is instituted, it must be determined whether the patient has had previous hypersensitivity reactions to cefotaxime, cephalosporins, penicillins or other drugs. CLAFORAN should be given with caution to patients with Type 1 hypersensitivity reactions to penicillin. Antibiotics, including CLAFORAN should be administered with caution to patients who have demonstrated some form of allergy, particularly to drugs. If an allergic reaction to CLAFORAN occurs, the drug should be discontinued and the patient treated with the usual agents (e.g. epinephrine, antihistamine, pressor amines or corticosteroids).

PRECAUTIONS
Pregnancy: The safety of CLAFORAN (cefotaxime sodium) in pregnancy has not been established. Consequently, use of the drug in pregnant women requires that the likely benefit from the drug be weighed against the possible risk to the mother and fetus.

Nursing Mothers: CLAFORAN is excreted in human milk in low concentrations. Caution should be exercised when the drug is administered to nursing mothers.

Pediatric Use: The safety and effectiveness of CLAFORAN in infants and children have not yet been established. *It has, however, been used in children at a dosage range of 100-150 mg/kg/day in 2-4 divided doses.*

Kidney: Although CLAFORAN rarely produces alterations in kidney function, evaluation of renal status is recommended, especially in severely ill patients receiving high doses.

Patients with markedly impaired renal function should be placed on the special dosage schedule recommended under DOSAGE AND ADMINISTRATION, because normal doses in these individuals are likely to produce excessive serum concentration of the metabolites.

Prolonged use of CLAFORAN may result in the overgrowth of nonsusceptible organisms. Constant observation of the patient's condition is essential. If superinfection occurs, therapy should be discontinued and appropriate measures taken.

Positive direct Coomb's test is known to develop in individuals during treatment with the cephalosporin group of antibiotics, including cefotaxime sodium.

In laboratory tests a false positive reaction to glucose may occur with reducing substances but not with the use of specific glucose oxydase methods.

ADVERSE REACTIONS
The most frequent adverse reactions encountered are:
Local (5%) – injection site inflammation with intravenous administration. Pain, induration and tenderness after intramuscular injection.

Hypersensitivity (1.8%) – Rash, pruritus, fever.

Gastrointestinal (1.7%) – Colitis (not pseudomembranous), diarrhea, nausea, and vomiting. A few cases of pseudomembranous colitis have been reported.

Hemic and Lymphatic System – Mild reversible leukopenia, granulocytopenia and thrombocytopenia have been reported. Some patients developed positive direct Coomb's test during treatment with CLAFORAN (cefotaxime sodium).

Genitourinary System – Moniliasis, vaginitis.

Central Nervous System (0.2%) – Headache.

Liver – Transient elevations in SGOT, SGPT, serum LDH, and serum alkaline phosphatase levels have been reported.

Kidney – Increased serum creatinine and BUN have occasionally been observed.

SYMPTOMS AND TREATMENT OF OVERDOSAGE
Since no case of overdosage has been reported to date with CLAFORAN, no specific information on symptoms or treatment is available. Treatment of overdosage should be symptomatic.

DOSAGE AND ADMINISTRATION
CLAFORAN (cefotaxime sodium) may be administered intravenously or intramuscularly after reconstitution (see table with recommended mode of reconstitution according to route of administration). Dosage and route of administration should be determined by susceptibility of the causative organisms, severity of the infection and condition of the patient.

USUAL ADULT DOSAGE OF CLAFORAN (CEFOTAXIME SODIUM)

Type of Infection	Daily Dose (grams)	Frequency and Route
Uncomplicated Gonorrhea	1	1 gram IM (single dose)
Uncomplicated infections such as pneumococcal pneumonia or acute urinary tract infection	2	1 gram every 12 hours IM or IV
Moderately severe infections	3-4	1 gram every 6-8 hours IM or IV
Severe infections	4-6	1 gram every 4-6 hours IM or IV
Infections commonly needing antibiotics in higher dosage (e.g., septicemia)	6-8	2 grams every 6-8 hours IV
Life-threatening infections	up to 12	2 grams every 4 hours IV

THE MAXIMUM DAILY DOSAGE SHOULD NOT EXCEED 12 GRAMS.

Administration of CLAFORAN should be continued for a minimum of 48 to 72 hours after the patient defervesces or after evidence of bacterial eradication has been obtained; a minimum of 10 days of treatment is recommended for infections caused by Group A beta-hemolytic *Streptococci* in order to guard against the risk of rheumatic fever or glomerulonephritis; frequent bacteriologic and clinical appraisal is necessary during therapy of chronic urinary tract infections and may be required for several months after therapy has been completed; persistent infections may require prolonged treatment. *Doses less than those recommended should not be employed.*

DOSAGE FOR PATIENTS WITH IMPAIRED RENAL FUNCTION
In patients with estimated creatinine clearance of less than 20 ml/min/1.73m² the dose of CLAFORAN must be halved (see PRECAUTIONS). If serum creatinine values alone are available, the following formula (based on sex, weight and age of the patient) may be used to convert these values into creatinine clearance.

Males: $\frac{\text{Weight (kg)} \times (140 - \text{age})}{72 \times \text{serum creatinine}}$

Females: 0.85 x above value

RECONSTITUTION
The following table is provided as a guide for reconstituting CLAFORAN (cefotaxime sodium) for both intramuscular and intravenous administration.

STRENGTH	AMOUNT OF DILUENT TO BE ADDED (ml)*	APPROXIMATE WITHDRAWABLE VOLUME (ml)	APPROXIMATE AVERAGE CONCENTRATION (mg/ml)
Intramuscular			
500 mg vial	2	2.2	230
1 g vial	3	3.4	300
2 g vial	5	6.0	330
Intravenous			
500 mg vial	10	10.2	50
1 g vial	10	10.4	95
2 g vial	10	11.0	180

*Shake to dissolve.

Parenteral drug products should be inspected visually for particulate matter and discoloration prior to administration. Solutions of CLAFORAN range from light yellow to amber, depending on concentration and diluent used. The dry powder as well as solutions tend to darken, depending on storage conditions.

For intramuscular use: CLAFORAN should be reconstituted only with Sterile Water for Injection in accordance with the volumes recommended in the preceding table.

For intravenous bolus use: 500 mg, 1 gram and 2 gram vials should be reconstituted with at least 10 ml of any of the solutions recommended under COMPATIBILITY/STABILITY.

For intravenous infusion use: 1 or 2 grams of CLAFORAN may be reconstituted with 50 or 100 ml of any of the solutions recommended under COMPATIBILITY/STABILITY.

ADMINISTRATION
Intramuscular: CLAFORAN (cefotaxime sodium) should be injected well within the body of a relatively large muscle such as the upper outer quadrant of the buttock (i.e., gluteus maximus); aspiration is necessary to avoid inadvertent injection into a blood vessel.

Intravenous: The intravenous route is preferable for patients with bacteremia, bacterial septicemia, or other severe or life-threatening infections, or for patients who may be poor risks because of lowered resistance resulting from such debilitating conditions as malnutrition, trauma, surgery, diabetes, heart failure, or malignancy, particularly if shock is present or impending.

For bolus administration a solution containing 1 gram or 2 grams of CLAFORAN can be injected over a period of three to five minutes. Using an infusion system, it may also be given over a longer period of time through the tubing system by which the patient may be receiving other intravenous solutions. Butterfly® or scalp vein type needles are preferred for this type of infusion. However, during infusion of the solution containing CLAFORAN, it is advisable to discontinue temporarily the administration of other solutions at the same site.

COMPATIBILITY AND STABILITY
CLAFORAN (cefotaxime sodium) may be reconstituted with any of the following and should be used freshly prepared:
- Sterile Water for Injection
- Sodium Chloride Injection B.P.
- 5% Dextrose Injection B.P.
- Dextrose and Sodium Chloride Injection
- Compound Sodium Lactate Injection B.P.

If a solution must be stored, only the solution prepared with Sterile Water for Injection may be kept under refrigeration (below 5°C) for not longer than 24 hours. Any unused solutions should be discarded.

CLAFORAN is compatible with 1% lignocaine. Only freshly prepared solutions should be used. CLAFORAN solutions exhibit maximum stability in the pH 5-7 range.

A solution of 1 gram of CLAFORAN in 14 ml of Sterile Water for Injection is isotonic.

Solutions of CLAFORAN must not be admixed with aminoglycoside solutions. If CLAFORAN and aminoglycosides are to be administered to the same patient, they must be administered separately and not as a mixed injection.

DOSAGE FORMS
CLAFORAN (cefotaxime sodium) is supplied as a sterile, white to pale yellow powder, in vials containing 500 mg, 1.0 gram and 2.0 grams of cefotaxime sodium (expressed as acid on a dry basis).

CLAFORAN in the dry state should be stored at room temperature (below 30°C), protected from light and heat. Solutions of CLAFORAN range from light yellow to amber, depending on concentration and the diluent used. The solutions tend to darken depending on storage conditions and should be protected from elevated temperatures and excessive light.