7-1-1987

Volume 30, issue 4

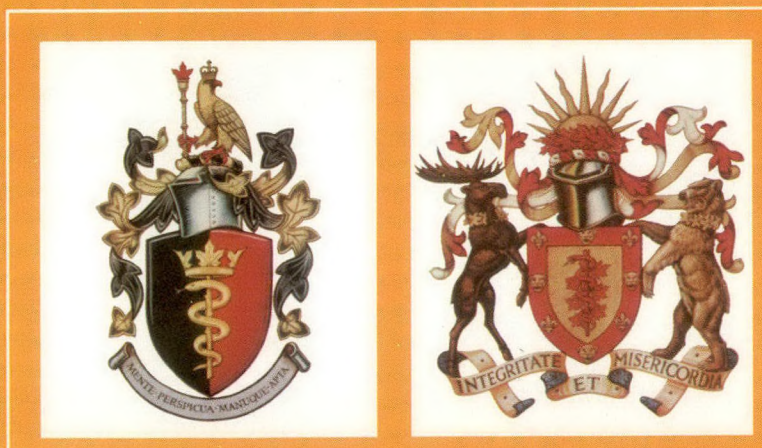
Canadian Medical Association

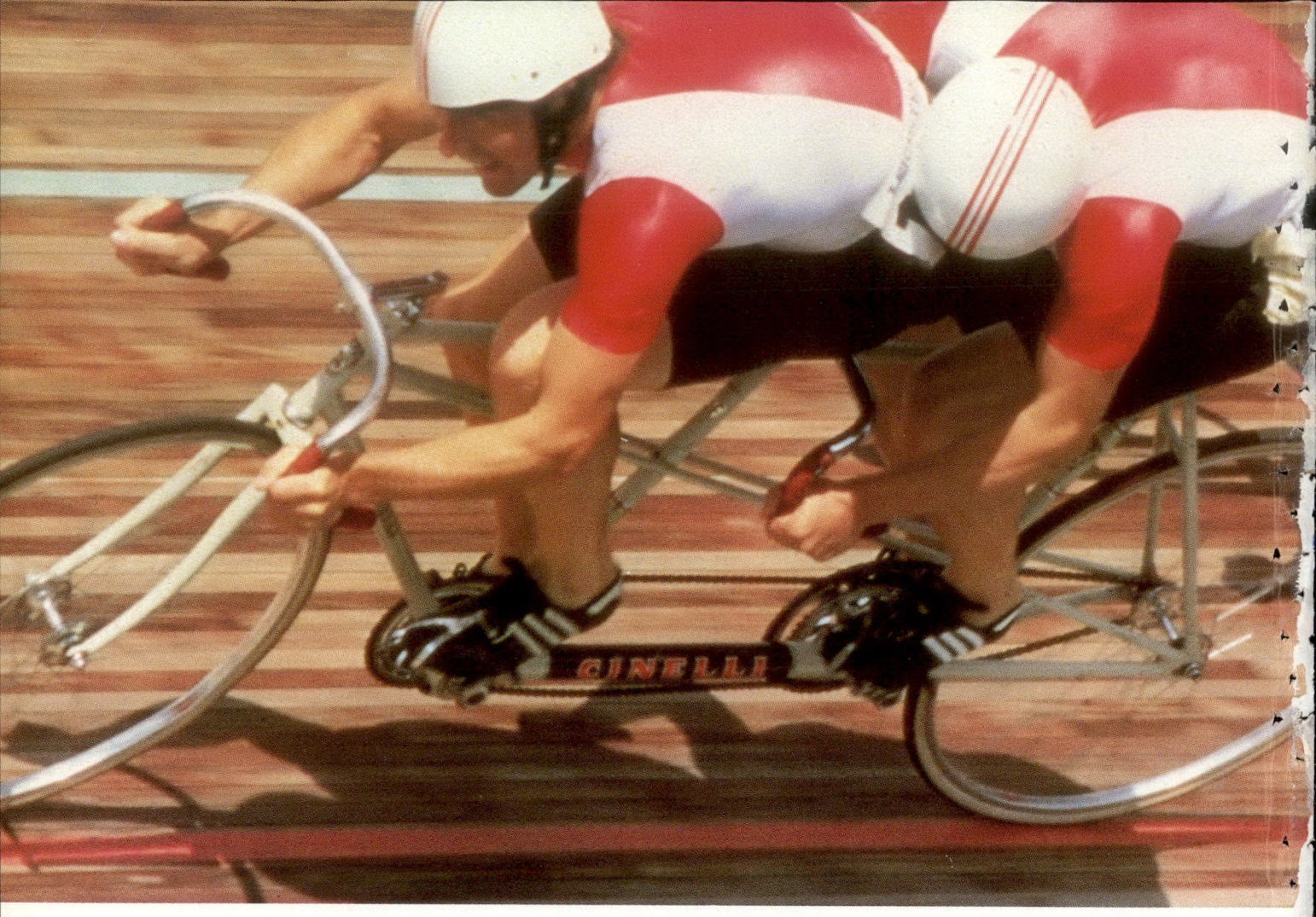
Volume 30, No. 4, July 1987

St. Anthony's Fire
Renal Lacerations
Anorectal Incontinence

The Canadian Journal of Surgery

Le journal canadien de chirurgie





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VOLUME 30, NUMBER 4

JULY 1987 JUILLET

ISSN 0008-428X

The Canadian Journal of Surgery / Le journal canadien de chirurgie

LIST OF CONTENTS

LIST OF CONTENTS Cont'd

ABSTRACTS—ROYAL COLLEGE MEETING 1987

QUILL ON SCALPEL

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

Survival After Hepatic Resection for Metastatic Disease

The article "Hepatic resection for metastatic disease" by Olak and colleagues (*Can J Surg* 1986; 29: 435-439) is important for several reasons: it deals with an issue that is of great interest to surgeons, it comes from a major academic institution whose credibility should not be in question, and the authors state that their data support the position that selected groups of patients with metastatic colorectal cancer will obtain long-term benefit from resection of liver metastases.

The core of the paper is a life-table analysis of the survival of 25 patients who had primary colorectal carcinoma and who underwent hepatic resection for metastases. For all patients surviving the hepatic resection, the 5-year survival projected by the analysis was 50.3%.

The life table is the best method for handling longitudinal data when patients enter a study at different times and at its conclusion have been observed for different lengths of time. Its efficiency lies in the fact that the survival experience of all patients under study is included in the analysis, not just of those who have died by the time the study is terminated. However, the authors do not seem to recognize the limitations of life-table analysis. As Peto and colleagues[1] pointed out, "if few patients are at risk for more than a certain time and after that time none of these few happens to die, there will be an apparent plateau in the life table. Such plateaux at the ends of life tables are very common and should never be taken as evidence that after a certain time most patients are cured unless there are large numbers of patients still at risk at the time of the plateau." In the Montreal study, there is a plateau after 30 months with fewer than five patients followed out to 5 years as shown in Fig. 1 of Olak's paper on page 437. The survival of 50.3% at 30 months should therefore not be projected to 5 years and any comment on long-term survival is invalid.

Another crucial point is that the probability values that make up the life table are determinations calculated from a sample of patients. As such they are subject to sampling variation that can be measured as standard error. By calculating the standard error of the probability of survival, we can answer the question, "With what precision does the survival of our sample of patients estimate the true survival of the population from which our sample is drawn?" The tabulated data allow us to carry out our own analysis to include the standard error of the probability of survival at each event (Table I). If we include the two operative deaths, which for some reason were omitted from the original analysis, the survival estimate at 36 months (the time of the last two deaths) is 43% with a 95% confidence interval of 17% to 69%. Thus, with a small sample of 25 patients, the estimate is imprecise and the true survival figure could be as bad as 17% or as good as 69%.

From the methodologic point of view, this is a weak study. There is no concurrent control group of untreated patients and reliance is placed upon comparison with historical control groups described

The Canadian Journal of Surgery
1867 Alta Vista Dr.
Ottawa, Ont. K1G 3Y6
Tel.: (613) 731-9331

The Canadian Journal of Surgery is published by the Canadian Medical Association and sponsored by the Royal College of Physicians and Surgeons of Canada. The establishment of editorial policy is the responsibility of the Royal College. The objectives of the Journal, endorsed by the Council of the College, are: (1) to contribute to the effective continuing education of Canadian surgical specialists, using innovative techniques when feasible and (2) to provide Canadian surgeons with an effective vehicle for the dissemination of their observations in the area of clinical research.

Published every 2 months by the Canadian Medical Association, PO Box 8650, Ottawa, Ont. K1G 0G8. Printed by Harpell's Press Cooperative, Gardenvale, PQ H0A 1B0. Second-class mail registration No. 5375. Return postage guaranteed. *Subscription rate for Canada and USA $30.00 per year ($15.00 per year for trainees in surgery in Canada only), for all other countries $35.00 per year. Single copies (current issue) available at $5.00 each, back issues at $6.00 each.*

Detailed instructions to contributors, in English and French, appear on page 41 of the January 1987 issue.

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by other authors. For instance, Adson and associates[2] reported that 20% of patients with solitary lesions that were not resected lived for 3 years or more. This figure falls within the 95% confidence interval of the estimate at 3 years for patients who underwent surgery in the Montreal study. Thus, from the purely statistical standpoint, the survival of the Montreal patients was not significantly different from that of Adson's control group.

What information, therefore, can we glean from this paper? Simply that the survival of patients who undergo hepatic resection for metastatic disease may be anywhere between 17% and 69% at 3 years. Whether or not patients will benefit from resection of liver metastases remains to be determined by larger studies that include the survival experience of a comparable group of untreated patients.

Table I—Survival Analysis for 25 Patients After Hepatic Resection for Metastatic Colorectal Cancer (Adapted From Olak and Associates, Table II)

Kaplan–Meier survival analysis*					
Time, mo	Died	Censored patients	Survival	Standard error	No. patients remaining
0	2	0	0.920	0.054	23
1	0	2	0.920	0.054	21
3	0	1	0.920	0.054	20
4	0	1	0.920	0.054	19
9	1	0	0.872	0.070	18
10	0	1	0.872	0.070	17
11	2	1	0.769	0.092	14
13	1	0	0.714	0.100	13
16	1	0	0.659	0.107	12
20	1	1	0.604	0.111	10
29	0	1	0.604	0.111	9
30	0	1	0.604	0.111	8
33	0	1	0.604	0.111	7
36	2	0	0.432	0.130	5
45	0	1	0.432	0.130	4
53	0	1	0.432	0.130	3
55	0	1	0.432	0.130	2
61	0	1	0.432	0.130	1
82	0	1	0.432	0.130	0

*All patients have been included (including the 2 operative deaths); 95% confidence intervals = observed estimate of survival ± 2 standard errors.

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1. PETO R, PIKE MC, ARMITAGE P, et al: Design and analysis of randomized clinical trials requiring prolonged observation of each patient. II. Analysis and examples. *Br J Cancer* 1977; 35: 1-39
2. ADSON MA, VAN HEERDEN JA, ADSON MH, et al: Resection of hepatic metastases from colorectal cancer. *Arch Surg* 1984; 119: 647-651

Relationship Between Improved Angina and Left Ventricular Function Change After Aortocoronary Bypass Grafting

Coronary artery bypass surgery has proven to be an effective treatment for the relief of intractable angina. Improvements in surgical technique and myocardial protection have made it a safe and acceptable approach even in poor-risk patients with severe left ventricular dysfunction.

In this issue (pages 269 to 271), Morton and colleagues from Ottawa address the question of whether a correlation exists between symptomatic improvement in angina and changes in left ventricular function following bypass surgery in patients with moderate to marked left ventricular dysfunction preoperatively. They conclude that no such relationship exists but that substantial relief of angina can be expected in these patients regardless of what happens to left ventricular function postoperatively.

Although this study has several deficiencies (it is retrospective, only a portion of the total group were restudied for varying reasons, and the assessment of symptomatic improvement is imprecise), it does makes some important points.

Angina is a result of an acute but transient imbalance between myocardial oxygen supply and demand. Fixed, significant coronary artery stenosis leads to exertional angina when coronary flow cannot increase to meet oxygen demand. The goal of bypass surgery is to ensure an adequate coronary flow reserve. A successful result is signalled subjectively by the elimination of exertional angina and objectively by the ability to achieve a higher external workload (heart rate multiplied by blood pressure product). Both goals are usually achieved after successful bypass surgery regardless of resting left ventricular function preoperatively.

Improved left ventricular function postoperatively has, in contrast, been more the exception than the rule. As Morton and his colleagues speculate, the obscurity of this association may relate in part to perioperative damage to previously normal myocardium. In the present era of improved myocardial protection, however, this association is more likely related to method of assessment. Utilizing serial radionuclide ventriculography, we were able to demonstrate a modest, but consistent, improvement in left ventricular function in patients with only mild to moderate impairment preoperatively.[1] Much of the reduction in left ventricular function seen in patients with more severe dysfunction, however, is likely due to myocardial necrosis and will not change with revascularization. The case for bypass surgery in these patients, therefore, depends upon whether or not there are substantial areas of myocardium that become ischemic upon exertion. This rationale is independent of left ventricular function and although the operative risk in such patients is undoubtedly greater, with proper selection the benefits of surgery are, as Morton's study confirms, very real, and the risk is quite acceptable.

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Reference

1. TCHERVENKOV CI, SYMES JF, SNIDERMAN AD, et al: Improvement in resting ventricular performance following coronary bypass surgery. *Ann Thorac Surg* 1985; 39: 340-345

CORRESPONDENCE

Contributions to the Correspondence section are welcomed. They should be typewritten and double spaced.

Fat Embolism and Total Knee Arthroplasty

To the editors.—I read with interest the article entitled "Fatal fat embolism during cemented total knee arthroplasty: a case report" by Orsini and colleagues in the September 1986 issue of the journal (pages 385 and 386). This type of complication, although previously described, certainly warrants reporting. However, I think it was somewhat misleading to attribute the complication simply to "cemented total knee arthroplasty" as the title implies. In fact, the fatal fat embolus was probably related to the introduction of a long stem into the medullary canal of the femur. For this reason, such a complication is more commonly observed after total hip arthroplasty. The prosthesis used in this patient, a Guepar II total knee arthroplasty, is generally regarded as obsolete. The indications for fully constrained hinged prostheses such as this are very limited. Moreover, modern resurfacing total knee arthroplasties rarely have intramedullary stems attached.

Notwithstanding this criticism, I thought the case was clearly and concisely reported. It is the title that was misleading, suggesting fat embolism was a complication of total knee arthroplasty rather than the result of introducing a prosthetic stem into the medullary canal.

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To the editors.—Dr. Vince correctly states that the total knee prosthesis associated with fatal fat embolism in our case report had a long intramedullary stem and that most contemporary total knee prostheses do not utilize intramedullary stems. However, research in our laboratory has clearly demonstrated that it is the combination of the introduction of a long intramedullary stem *and* bone cement that produces the high intramedullary pressures associated with fat embolism.[1,2] This evidence, together with the infrequent association of the fat embolism syndrome with total knee arthroplasty, led to our choice of title. Perhaps a more accurate title would have been "Fatal fat embolism during cemented *long-stem* total knee arthroplasty". Nevertheless, we wish to emphasize that this complication was associated with the use of bone cement rather than, as Dr. Vince suggests, simply the introduction of an intramedullary stem. We thank Dr. Vince for his comments.

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2. ORSINI EC, BYRICK RJ, MULLEN JMB, et al: Cardiopulmonary function and pulmonary microemboli during cemented and noncemented arthroplasty: the role of intramedullary pressure. *J Bone Joint Surg* [*Am*] (in press)

Surgical Management of Crohn's Disease

To the editors.—The article "New options in the surgical management of Crohn's disease" (*Can J Surg* 1987; 30: 133–136) is very helpful and timely.

It is not uncommon to find at operation relatively short and often multiple strictures, but deciding which strictures to include and how long an excision should be has been very difficult in the past. Strictureplasty makes management much simpler.

I would recommend a variant of the technique described in the article. With relatively short strictures, the Finney technique with a GIA stapler is quick and simple. The technique is similar to that used for a Finney pyloroplasty, if one envisages the stricture as the pylorus. I have used this technique, without any problems.

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Determining and Rationalizing the Supply of Physicians

To the editors.—I was surprised to read in your March 1987 issue (pages 77 and 78) that there is a move afoot to increase the number of training slots for general surgeons in Canada. The reasons given for this philosophy are twofold — that there will be an increasing number of general surgeons retiring in the future and that "training takes a minimum of 4 years so there's no turn on, turn off mechanism".

Before embarking on a project with such long-term ramifications, more than those two factors should be taken into consideration. Surgeons have been retiring ever since the specialty was first instituted, so that is nothing new. Even though it may take 4 years to train a surgeon, he will stay in the pool for up to 30 years making the "turn on, turn off" mechanism even more difficult to manage. McPhedran talks of the difficulties encountered in trying to increase the number of training slots. That is mere child's play when compared to the difficulties encountered when trying to reduce the number of training slots because of many vested interests.

The figures quoted are based on simple historical population statistics. These do not take into account the fact that the number of surgical procedures is slowly being reduced. For example, until the 1970s, peptic ulcer surgery was so common that even junior residents were given an opportunity to perform gastric and duodenal surgery. Then came H2 blocking agents. During that same era, rheumatic heart disease provided cardiac surgeons with an endless supply of patients

requiring valve replacements. Today, they have to content themselves with doing coronary artery bypass surgery, for rheumatic heart disease has disappeared. In our hospital last year, the introduction and aggressive use of indomethacin intravenously in neonates with patent ductus arteriosus has made duct ligation a rare event. Over the last three decades the incidence of appendicitis has dropped to half what it was in the pre-antibiotic era. Since medical research continues at an ever-increasing pace, one can anticipate that the number of diseases requiring surgery for their cure will continue to decline.

In the United States there is now an oversupply of surgeons. In 1974, the average number of surgical procedures performed per individual surgeon was 217. It had decreased by 1979 to 176 and by 1984 to 161.[1] This is an average of one procedure every other day. What an incredible waste of manpower. How are junior surgeons going to gain the experience and proficiency that are such a necessary part of their profession? It is stimulating and exhilarating to be busy and most surgeons are quite capable of handling a much greater caseload without difficulty. Concurrently, because of increased experience and practice, they may even perform better surgery.

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Reference

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Value of Trauma Centres

To the editors.—The recently published article by Roy (*Can J Surg* 1987; 30: 17–22), regarding the value of trauma centres, points out clearly the difficulties faced by health-care planners, surgeons and politicians in assessing the impact of regional trauma centres on the care of the acutely injured patient. Unfortunately, as Roy has pointed out, a great deal of the often-quoted data is anecdotal or publications of case series, with few well-designed prospective trials. Moreover, an overwhelming number of these reports come from the United States where injury patterns are known to differ from those in Canada. To a certain extent, similar disparities already exist in Canada with respect to injury patterns and outcomes (e.g., quoted population differences between Sunnybrook Medical Centre and St. Michael's Hospital in Toronto).

Several other factors relevant to the trauma-centre argument should be explored before any system of regional trauma care can be established in any region of Canada:

- An analysis of the morbidity of all trauma patients treated in trauma centres versus non-trauma centres.
- Mortality and morbidity of patients who are not identified as having major trauma (injury severity score greater than or equal to 16, trauma score less than or equal to 12).
- The incremental costs of trauma care in trauma versus non-trauma centres.

Such factors as these necessarily point to well-designed before-and-after studies of trauma patterns, which would then be followed by the development of regional trauma units (i.e., a true establishment of need in a given region).

I wholeheartedly support Dr. Roy's argument that accurate well-designed provincial trauma registries (independent of hospital medical records) must be firmly in place to answer all of these questions.

However, I take some exception to Dr. Roy's concluding remarks in this otherwise excellent article. Just as there is inconclusive evidence in the medical literature regarding trauma centre designation and acutely injured outcomes, so I must ask the question, "Is there evidence that the Advanced Trauma Life Support Course makes any difference to trauma care in these situations?" In addition, in my experience, emergency physicians no longer need further education in early trauma care. This needs to be directed to family physicians who infrequently take emergency calls in their community hospitals, and to general and orthopedic surgeons who think that their FRCSC automatically qualifies them as experts in resuscitation of traumatized patients.

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To the editors.—All of Dr. Girotti's points are well taken. As a group, emergency department physicians in *university* teaching hospitals are probably the best providers of advanced trauma life support in Canada. By "emergency physicians" I meant simply the providers of primary care, particularly at the periphery. This would, of course, include many family physicians who only do occasional emergency department duty. This should definitely be the target group in any such educational program. That is not to say that an FRCSC should be interpreted as a licence to resuscitate. Many residency programs, including our own, offer the Advanced Trauma Life Support Course as part of the program, and the McGill University Medical School offers it to all medical students at the clinical clerkship level.

I have no proof that the Advanced Trauma Life Support Course per se makes a detectable difference. It is merely a framework, a cookbook if you like, to be followed. However, I know of no other method that will efficiently teach a group of physicians in a short period the essentials that I feel are necessary for optimal resuscitation and treatment in the "golden hour".

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Knee Disassembly

To the editors.—The paper entitled "Role of total knee replacement in failed knee fusions" in the January 1987 issue of the journal (pages 25 to 27) is pioneering. However, the statement that "nothing has yet appeared in the literature on disassembly" is not correct.

A case[1] treated by Wright was described as follows: a 26-year-old woman with previous knee fusion for tuberculosis at age 13 had a simple arthroplasty done, followed by spontaneous ankylosis, then a Walldius total knee replacement. One year later, loosening required revision with cementing. At 5 years, the patient passively flexed (by gravity) to 80°, had no active extension, but by hand lifted her knee straight when getting up from a chair. The gait was acceptable and she was happy with the result to that point.

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Reference

1. SILLER TN: Arthrodesis in treatment of degenerative arthritis in the knee. In CRUESS RL, MITCHELL NS (eds): *Surgical Management of Degenerative Arthritis of the Lower Limb*, Lea & Febiger, Philadelphia, 1975: 203–208

To the editors.—I should like to apologize to Dr. Siller for omitting mention of the case he describes in Cruess and Mitchell's book on degenerative arthritis of the lower limb.

What is of interest in the case described by Siller is the lack of active extension. In the most recent fusion takedown I have

done, I found the patellar tendon exquisitely thin and was very concerned about the possibility of rupture. I therefore reinforced it with a couple of wire loops extending from above the patella to drill holes in the tibia. This man has powerful extension, but is very, very slow in gaining flexion.

The finding of a very thin patellar tendon suggests that the patient reported by Dr. Siller had in fact a patellar tendon rupture, which would explain her loss of active extension.

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Resection of the Liver for Metastatic Disease

To the editors.—We are grateful for the opportunity to respond to Dr. Gately's editorial.

We all recognize that, ultimately, the ideal study to decide the relative merit of alternative forms of therapy, is a prospectively randomized double-blind controlled trial. Unfortunately, this is not a perfect world!

Since less than 15% of patients with colorectal cancer and liver metastasis will be candidates for resection, the experience of any one institution is going to be extremely small. Indeed, it was only by creating a registry and personally reviewing the records and experience of a great many institutions across the country that Foster[1] was able to suggest this was a viable and perhaps superior treatment option in the hands of experienced hepatic surgeons. The number of subsets to be analysed is also great. They include: stage of the primary tumour; age and sex of the patient; synchronous versus metachronous hepatic metastasis; size and number of liver metastases; extent of hepatic resection required, etc. A large multicentre (if not multinational) trial with all its inherent difficulties and tremendous expense would be necessary. In addition, the difficulty in generating a concurrent control group of untreated patients is real. There is still a place, and indeed a necessity at this stage, for major institutions and hospitals to report their experiences to the surgical community at large.

Gately's suggestion that "the core of the paper is a life-table analysis of the survival of 25 patients who...underwent hepatic resection for metastases" is incorrect and myopic. Our article was entitled "Hepatic resection for metastatic disease" and not "Survival after hepatic resection..." as implied in Dr. Gately's editorial comments. It was intended to share and communicate our total experience with hepatic resection for metastatic disease including types of procedures, mortality and complications, and to relate outcome to the various subsets previously described, looking for apparent differences that might lead to better patient selection.

In reporting our observed survival, we have presented data in a form comparable to that reported by other authors who also use life-table methods. Our epidemiologists and statisticians at McGill were consulted before we collected our data and they agreed that this was the best method of presentation. We certainly do recognize the limitations of this method and have clearly indicated the number of patients under observation at each 6-month interval on every life table reported in the article. Furthermore, we deliberately presented, in Table IV, "Observed Survival (in Months)" and have clearly stated throughout that the life-table curves generated only a "projected survival figure". Since patients were being entered in the study up to 1985, it goes without saying that most will not have been observed for 5 years! Dr. Gately himself states that "the life table is the best method for handling longitudinal data when patients enter a study at different times and at its conclusion have been observed for different lengths of time". If the life-table method is not the "best-fit" method for representing these data, Dr. Gately has not told us which method is. We have been careful to avoid making statistical conclusions or analyses based on projected numbers at 5 years.

Dr. Gately correctly notes that the two operative deaths were omitted from the original life-table analysis. We are attempting to look at the effect of a surgical intervention on the biology and natural history of hepatic metastases. This is not a cost–benefit analysis and operative deaths, of necessity, must be excluded in this context. That is not to say that operative mortality is unimportant. It is incumbent upon any surgeon and institution to prove that this operation can be accomplished with low morbidity and mortality and was a greater impetus for us to review and discuss our own data than to analyse survival.

With small sample size, estimates of long-term survival will be imprecise as Dr. Gately correctly emphasizes. All institutions must continually update and report their survival figures with time and we are currently in the process of doing so with 18 additional months of follow-up. To our dismay, recurrences are being seen as an ongoing event; however, regardless of the ultimate *overall* survival data, there is no question that in some patients survival is being prolonged with a potential for "cure" in a few. At no time is "reliance...placed upon comparison with historical control groups described by other authors" as Gately states. The fact that Adson and associates[2] reported that 20% of patients who had unresected solitary lesions lived 3 years or more does not detract from our report that 13 of 23 patients at risk, taking all subgroups, were alive and free of disease a mean of 24 months after hepatic resection. How better to define and select the subgroup with greatest potential for gain is the present interest. This will come with newer and better methods of detecting subclinical extrahepatic metastasis prior to undertaking hepatic resection, not by more concurrent control groups of untreated patients as suggested by Dr. Gately.

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2. ADSON MA, VAN HEERDEN JA, ADSON MH, et al: Resection of hepatic metastases from colorectal cancer. *Arch Surg* 1984; 119: 647–651

BOOKS RECEIVED

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

Advances in Surgery. Volume 20. Edited by John A. Mannick. 352 pp. Illust. Year Book Medical Publishers, Inc., Chicago, 1987. $47.95 (US). ISBN 0-8151-5751-7.

Aesthetic and Reconstructive Otoplasty. Jack Davis. 581 pp. Illust. Springer-Verlag, New York, 1987. $199.00 (US). ISBN 0-387-96308-1.

Bladder Reconstruction and Continent Urinary Diversion. Edited by Lowell R. King, Anthony R. Stone and George D. Webster. 379 pp. Illust. Year Book Medical Publishers, Inc., Chicago, 1987. $65.00 (US). ISBN 0-8151-5070-9.

Case Presentations in General Surgery. T.V. Taylor, C.P. Armstrong, R.N.P. Carroll, 205 pp. Butterworth & Co. (Publishers) Ltd., London; Butterworth Publishers, Stoneham, Mass., 1987. $19.95 (US). ISBN 0-407-00545-5.

Ciclosporin in Renal Transplantation. Contributions to Nephrology, Volume 51. Edited by C. Ponticelli and A. De Vecchi. 167 pp. Illust. Karger, Basel, 1986. $74.50 (US). ISBN 3-8055-4357-3.

Essentials of Thoracic Surgery. Raymond Hurt and Michael Bates. 270 pp. Illust. Butterworth and Co. (Publishers) Ltd., London; Butterworth Publishers, Stoneham, Mass., 1986. $59.95 (US). ISBN 0-407-00397-5.

continued on page 236

If most ads this will do it for

There's method to our madness here. We're dramatizing a point about Marcaine.* The point is, Marcaine lasts a lot longer than other local anaesthetics. Up to twice as long, in fact. (See Covino, B.G. and Scott, D.B. Handbook of Epidural Anaesthesia and Analgesia. Grune & Stratton. In Orlando, 1985; 70.)

Since Marcaine lasts twice as long, our ad is twice as long. We understand that you may not have the time at one sitting to read and digest the amount of information shown here. But if you've read this far, you have the main point about Marcaine, the reason why it is so well respected and so well used, and later we'd like to table some discussion points about additional uses for Marcaine, and perhaps reacquaint you with its other advantages, such as the degree of participation it allows the mother in labour, and the fact it is 95% plasma protein bound which limits the amount of drug transfer across the placental barrier to the foetus.

Longer duration is the key benefit of Marcaine to the anaesthetist. It lasts up to twice as long as lidocaine which means that you can achieve optimum pain control during surgical procedures. It also saves the obstetric anaesthetist time because fewer top-ups are necessary with Marcaine (see Ostheimer, G.W., Regional Anaesthesia Techniques in Obstetrics, Breon Laboratories Inc., 1980, p.23). But another key point about Marcaine is that it separates sensory from motor block. Excellent sensory anaesthesia can be obtained with less motor block than any other local anaesthetic. This separation allows for complete pain relief during and after surgery, yet lessens immobility in the post-operative phase. In obstetrics, Marcaine can maintain the mother's ability to bear down in labour while providing relief of pain. Greater involvement and cooperation of the mother are considered desirable in obstetrics for the 80's. (The reference material in the above paragraph outlines data on this phenomenon as well as evidence for the duration of Marcaine.)

Indeed, the long duration of Marcaine (and in actual fact, Marcaine can last twice as long as other injectable local anaesthetics) not only reduces the time you spend because of fewer top ups during a procedure, it produces an extended pain free period in the mother after the birth when perineal repair has been required. Unlike general anaesthesia, the mother is also conscious during and immediately after the delivery so bonding and breast feeding can occur earlier.

What are the advantages of Marcaine and epidural anaesthesia relative to general anaesthesia? First and foremost is the avoidance of one of the principal hazards of general anaesthesia, i.e. vomiting and aspiration of stomach contents. Secondly, consider the area of endocrine stress response to anaesthesia and surgery (Lindall, S., et al: Endocrine Stress Response during General and Epidural Anaesthesia for Elective Caesarean Sections, Acta Anaesthesiol Scand. 1983; 27:50-55). With inhalation anaesthesia maternal plasma levels of cortisol and ACTH - endocrine stress markers - are consistently higher. The foetus may have a higher endocrine stress response.

And the argument can be effectively summarized by this statement: "Bupivacaine (Marcaine) is currently established as the safest and most effective amide-linked local anaesthetic for obstetric anesthesia." (Bromage P.R.: Choice of Local Anesthetics in Obstetrics. In Shnider, S.M. and Levinson, G. (eds.): Anesthesia for Obstetrics. The Williams & Wilkins Company, Baltimore, 1979, 113.) And this statement: "The clear advantages to the post-partum mother are sufficient grounds for encouraging the teaching and eventually the routine use of epidural anaesthesia for caesarean section on all suitable patients." (Morgan B.M., et al: Anaesthetic Morbidity Following Caesarean Section Under Epidural or General Anaesthesia. The Lancet. 1984: 328-330). We have already mentioned the longer duration of Marcaine and the additional benefit that Marcaine is 95% plasma protein bound which limits the amount of drug transfer across the placenta to the foetus. Marcaine also causes minimal foetal and neonatal depression of neuro behaviour and muscle tone.

At this point, please allow for a pertinent digression from the discussion on the longer duration of Marcaine and other benefits.

INDICATIONS: Peripheral nerve block, infiltration, sympathetic blockade, caudal, epidural, and pudendal blocks.

CONTRAINDICATIONS: Bupivacaine is contraindicated in persons with known sensitivity to local anesthetics of the amide type. The use of bupivacaine is contraindicated in the presence of sepsis near the site of proposed injection, in severe shock and in heart block.

WARNINGS: USAGE IN PREGNANCY: Decreased pup survival in rats and an embryocidal effect in rabbits have been observed when bupivacaine hydrochloride was administered to these species in doses comparable to nine and five times respectively the maximal recommended daily human dose (400 mg). There are no adequate and well-controlled studies in pregnant women of the effect of bupivacaine on the developing fetus. Bupivacaine hydrochloride should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus. This does not exclude the use of Marcaine 0.25% or 0.50% at term for obstetrical anesthesia or analgesia.

OBSTETRICS: The highest (0.75%) concentration is not recommended for obstetrical anesthesia. There have been reports of cardiac arrest with difficult resuscitation or death following its use for epidural anesthesia in obstetrical patients. Due to the high risk to the fetus, paracervical block is no longer recommended. The obstetrician is warned that severe persistent hypertension may occur after administration of certain oxytocic drugs, if vasopressors have already been used during labor (e.g. in the local anesthetic solution or to correct hypotension). Until further experience is gained in children younger than 12 years, administration of bupivacaine in this age group is not recommended.

PRECAUTIONS: RESUSCITATIVE EQUIPMENT AND DRUGS SHOULD BE READILY AVAILABLE WHEN ANY LOCAL ANESTHETIC IS USED. The safety and effectiveness of local anesthetics depend upon proper dosage, correct technique, adequate precautions, and readiness for emergencies. Marcaine (bupivacaine) should be used cautiously in persons with known drug allergies or sensitivities, particularly to the amide-type local anesthetics. Caution is advised in administration of repeat doses of bupivacaine to patients with severe liver disease. The following precautions apply to all local anesthetics: Select needles of proper length and bevel for the technique employed. Inject slowly with frequent aspirations and if blood is aspirated, relocate the needle. Inadvertent intravascular injection may cause serious complications. Absorption is more rapid when injections are made into highly vascular tissues. In caudal or epidural anesthesia, abandon the method if the subarachnoid space has been entered, as shown by aspiration of spinal fluid. The lowest dosage that gives effective anesthesia should be used, to avoid high plasma levels and serious systemic side effects. Injection of repeated doses of bupivacaine may cause a significant increase in blood levels due to accumulation of the drug or its metabolites or slow metabolic degradation. Tolerance varies with the status of the patient. Debilitated, elderly and acutely ill patients may require reduced doses commensurate with age and physical condition. It should be remembered that solutions containing a vasopressor agent, e.g. epinephrine, should be used with caution, if at all, in patients who are receiving monoamine oxidase inhibitors or anti-depressants of the triptyline or imipramine type, because severe, prolonged hypertension may result. Dose-related cardiac arrhythmias may occur if preparations containing epinephrine are employed in patients during or immediately following the administration of chloroform, halothane, cyclopropane, trichloroethylene or other related agents. In deciding whether to use these products concurrently in the same patient, the combined action of both agents upon the myocardium, the concentration and volume of vasoconstrictor used, and the time since injection, when applicable, should be taken into account. The decision to use a local anesthetic containing a vasoconstrictor in areas with a limited blood supply or in patients with peripheral vascular disease, will depend on the physician's appraisal of the relative advantages and risks. Local anesthetics which contain preservatives, i.e. those supplied in multiple dose vials, should not be used for caudal or epidural anesthesia.

EPIDURAL USE: It is advised that a test dose, generally 2-3 mL of 0.5% bupivacaine (or other amide anesthetic) containing 1:200,000 epinephrine (10-15 micrograms) be administered to check that the spinal canal or a blood vessel has not been entered while locating the epidural needle or catheter. In the event of spinal injection clinical signs of spinal block would become evident in a few minutes. In the event of intravascular injection a transient increase in pulse rate and possibly momentary increase in systolic blood pressure are usually detectable with a monitor. The other symptoms and signs of "epinephrine response" are less dependable. The effects of other medication the patient has received may modify this response. When reinforcing doses are required the test dose should be used again to check the catheter location.

ADVERSE REACTIONS: Reactions to bupivacaine are characteristic of those associated with amide-type local anesthetics. A major cause of adverse reactions to this group of drugs is excessive plasma levels, which may be due to over-dosage, inadvertent intravascular injection, or slow metabolic degradation. Other causes of reactions to these local anesthetics may be hypersensitivity, idiosyncrasy, or diminished tolerance. Excessive plasma levels cause systemic reactions involving the central nervous system and the cardiovascular system. The CENTRAL NERVOUS SYSTEM EFFECTS are characterized by excitation or depression. The first manifestation may be nervousness, dizziness, blurred vision, or tremors, followed by drowsiness, convulsions, unconsciousness, and possibly respiratory arrest. Since excitement may be transient or absent, the first manifestation may be drowsiness, sometimes merging into unconsciousness and respiratory arrest. Other central nervous system effects may be nausea, vomiting, chills, constriction of the pupils, or tinnitus. The CARDIOVASCULAR MANIFESTATIONS of excessive plasma levels may

put you to sleep, twice as long.

include depression of the myocardium, blood pressure changes (usually hypotension), and cardiac arrest. Recent clinical reports and animal studies suggest this may be more likely to occur with the long acting amide local anesthetics such as bupivacaine.

ALLERGIC REACTIONS are characterized by cutaneous lesions (e.g. urticaria, edema) and other manifestations of allergy. Reactions following epidural or caudal anesthesia may include: high or total spinal block, urinary retention; fecal incontinence, loss of perineal sensation and sexual function; persistent analgesia, paresthesia, and paralysis of the lower extremities; headache and backache; and slowing of labor and increased incidence of forceps delivery. It should be noted that reactions due to systemic absorption may be slow or rapid in onset. Those of rapid onset include respiratory depression, cardiovascular collapse and cardiac arrest. This type of reaction necessitates a high degree of preparedness since it can occur with little warning.

In co-ordinated studies of 3200 procedures carried out by 15 investigators, there were 2 severe systemic reactions. Both patients experienced convulsions as a result of inadvertent vascular injection. Fetal bradycardia has been observed with the use of bupivacaine. Most cases, including a few fatalities, occurred when the paracervical route was used (see "Warnings").

In some subjects bupivacaine may produce marked peripheral vasoconstriction in unanesthetized areas which may last for several hours.

TREATMENT OF OVERDOSE AND SEVERE REACTIONS: Toxic effects of local anesthetics require symptomatic treatment; there is no specific cure. The physician should be prepared to maintain an airway and to support ventilation with oxygen and assisted or controlled respiration as required. Supportive treatment of the cardiovascular system includes intravenous fluids and, when appropriate, vasopressors (preferably those that stimulate the myocardium). Convulsions may be controlled with oxygen and intravenous administration, in small increments, of a barbiturate or muscle relaxant, as follows: preferably, an ultra short-acting barbiturate such as thiopental or thiamylal, if this is not available, a short-acting barbiturate (e.g. secobarbital or pentobarbital) or a short-acting muscle relaxant (succinylcholine). Intravenous muscle relaxants and barbiturates should only be administered by those familiar with their use.

DOSAGE AND ADMINISTRATION: As with all local anesthetics, the dosage varies and depends upon the area to be anesthetized, the vascularity of the tissues, the number of neuronal segments to be blocked, individual tolerance, and the technique of anesthesia. The lowest dosage needed to provide effective anesthesia should be administered.

In recommended doses, bupivacaine produces complete sensory block, but the effect on motor function differs between the three concentrations.

0.25% when used for caudal, epidural, or peripheral nerve block, produces incomplete motor block. Should be used for operations in which muscle relaxation is not important, or when another means of providing muscle relaxation is used concurrently.

0.5% provides motor blockade for caudal, epidural, or nerve block, but muscle relaxation may be inadequate for operations in which complete muscle relaxation is essential.

0.75% produces complete motor block. This concentration is recommended only for epidural block (single dose) in abdominal operations requiring complete muscle relaxation without the aid of other medication. It is not recommended for epidural block in obstetrical patients.

The duration of anesthesia with bupivacaine is such that, for most procedures, a single dose is sufficient. Maximum dosage limit must be individualized in each case after evaluating the size and physical status of the patient, as well as the usual rate of systemic absorption from a particular injection site. Most experience to date is with single doses of bupivacaine up to 225 mg with epinephrine 1:200,000 and 175 mg without epinephrine; more or less drug may be used depending on individualization of each case. At present there is insufficient clinical evidence with multiple dosage or intermittent dose techniques to permit precise recommendations for such procedures to be given. However, limited clinical experience in this area of use indicates that bupivacaine may be repeated in 3 to 6 hours up to a maximum dose of 400 mg in 24 hours. In most cases the duration of anesthetic effect is prolonged by the addition of epinephrine. The following table is presented as a guide to the use of bupivacaine. The doses shown have generally proved satisfactory for the average adult. They may require adjustment in relation to age and the physical condition of the patient.

RECOMMENDED CONCENTRATIONS AND DOSAGE FOR ADULTS

Type of Block	Concentration	mL	Each Dose	mg	Motor Block[1]
Local Infiltration	0.25%	up to max		up to max	———
Epidural[3]	0.75%[2]	10-20		75-150	Complete
	0.50%	10-20		50-100	Moderate to Complete
	0.25%	10-20		25- 50	Partial to Moderate
Epidural[3]	0.50%	2-3		10- 15	———
Test Dose	w/epi			(10- 15μg epinephrine)	
Caudal	0.50%	15-30		75-150	Moderate to Complete
	0.25%	15-30		37.5- 75	Moderate
Peripheral Nerves	0.50%	5-30		25-150	Moderate to Complete
	0.25%	5-60		12.5-150	Moderate to Complete
Sympathetic	0.25%	20-50		50-125	———

1. With continuous (intermittent) techniques in caudal and epidural block using 0.25 and 0.5% solutions, repeat doses increase the degree of motor block. The first dose of 0.5% may produce complete motor block in most intercostal nerve blocks for intra-abdominal surgery, the 0.25% concentration has produced satisfactory motor blockade.
2. For single dose use not for intermittent technique.
3. Use of an appropriate test dose is recommended prior to injecting the full epidural dose (see "Precautions")

AVAILABILITY: ISOTONIC SOLUTIONS:

0.25% – 10 mL single dose vials (without preservative), (boxes of 5).
- 20 mL single dose vials (without preservative) with and without epinephrine (boxes of 5).
- 50 mL multiple dose vials (containing methylparaben as preservative) without epinephrine (boxes of 1).

0.50% – 3 mL single dose ampuls (without preservative) with epinephrine (boxes of 10).
- 10 mL single dose vials (without preservative), (boxes of 5).
- 20 mL single dose vials (without preservative) with and without epinephrine (boxes of 5).
- 50 mL multiple dose vials (containing methylparaben as preservative) without epinephrine (boxes of 1).

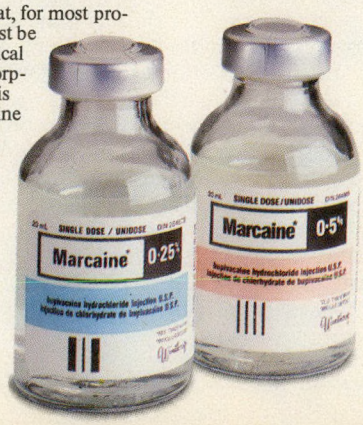
0.75% – 3 mL single dose vials (without preservative) without epinephrine (boxes of 5).

The solutions are made isotonic with NaCl and the pH is adjusted with NaOH or HCl. The pH range for solutions without epinephrine is 4.0-6.5 and for solutions with epinephrine is 3.4-4.5. Plain solutions may be autoclaved but those containing epinephrine may not. Each mL of solution with epinephrine contains 0.0091 mg epinephrine bitartrate with 0.5 mg sodium metabisulfite. 0.001 mL monothioglycerol, and 2 mg ascorbic acid as antioxidants, 0.002 mL 60% sodium lactate buffer and 0.1 mg edetate calcium disodium as stabilizer.

Product Monograph is available on request.

Marcaine allows better participation of the mother in labour. Marcaine is 95% plasma protein bound which limits the amount of drug transfer across the placenta to the foetus.

But first and foremost, Marcaine offers longer duration of pain control needed for surgical and obstetrical procedures.



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SURGEONS' UPDATE

What's new in surgery is the subject of this column. The short items are designed to let readers know who's doing what and why. Surgeons are interested in what other surgeons are doing in research, education, practice and administration. Surgery is a vibrant specialty, and, as its practitioners, you must be the source as well as the readers of this column.

Fees and Benefits: a Challenge to Universality of Medicare

The schedules of benefits paid for by provincial health insurance plans across Canada are likely to change markedly in the next few years, as Alberta joins BC as provinces that have disallowed some payments previously supported.

In Alberta in mid-May, the government cut back its list of "basic health services" in an effort to save $40 million and asked the public to help save another $25 million. When the Alberta government announced that it would no longer automatically pay for, among others, gastroplasty, eye examinations, circumcision, sterilization (and reversal operations) or blepharoplasty, the country turned toward Ottawa to hear the federal response. Health and Welfare Minister Jake Epp replied a few days later that such cutbacks would not endanger transfer payments as long as the services were not medically necessary.

In an open letter to Albertans, the Minister of Hospitals and Medical Care said, "Since 1980 costs have more than doubled...we have seen the price tag for health-related services rise from $1.1 billion in 1980 to approximately $3 billion in 1986, or from under $600 for every Albertan...to $1300...." The letter, signed "Marv Moore", solicited public help: "In order to meet our budgetary target of having no increase in the present expenditure of $694 million for the Alberta Health Care Insurance Plan, it will be necessary to save a further $25 million. If we all help by using our health care system in a responsible manner and perhaps using the system one time less, then I believe we can save even more and succeed in preserving the quality of our services for years to come."

The news release accompanying the announcement said, "...a new restructured Utilization Committee is being formed and will be working particularly in the area of reducing unnecessary medical tests and x-rays which have escalated dramatically in recent years".

At present, the concern about rising costs may be the only universal element in the provinces' health systems despite the Canada Health Act. Efforts to increase the funds available to pay the bill included New Brunswick's official ban of extra-billing at the end of March, thus making that province the last to become eligible for transfer payments withheld by the federal government.

Earlier this year the Manitoba government refused a medical fee increase that was recommended by a three-lawyer arbitration team. The government later changed its position, citing ambiguity in the arbitrators' report as the reason for its reticence in accepting the 5.6% increase in fees and the 0.9% increase anticipated for growth in utilization. The refusal came during the first test of an agreement between the province and the medical association to put an end to extra-billing.

The recent move by Alberta, now given the federal nod, is likely to become popular.

A look at the different schedules for fees and benefits shows that some provinces have more freedom to maneuver than others. For example, Ontario and Alberta have agreed to pay for one complete annual health examination for each resident, whereas this is not a benefit in many other provinces; Newfoundland has a clause permitting payment for consultations required by law or statute "presumably in the best interest of patients and hospitals", but other provinces specifically forbid such payments.

The differences in benefits are likely to be closely scrutinized in future, as is the lack of parity in payments to practitioners. Theoretically, accessibility is at risk for patients who come from a province where practitioners have fallen far behind in their fee increases.

A sampling of surgical procedures (Table I) for which payment is allowed in all the provinces indicates the great variation in amounts paid; the preambles for the schedules in some instances offset the differences, in others increase them. Regular "business" hours for physicians vary from 9 hours in Alberta, Newfoundland and Saskatchewan to a high of 13 for GPs in Quebec, where Saturday is considered part of the work week.

The fee for most surgical procedures in Quebec includes 90 days of preoperative care and 15 days of postoperative care. Although all the other provinces also include some preoperative and postoperative care in the price of the procedure, most allow surgeons to charge a fee for the major consultation, include some premium for performing the procedure outside normal business hours or add a surcharge for special visits.

QMA Continues Bid to Negotiate Fees

The discrepancies in Quebec physicians' reimbursements when compared with practitioners in the rest of Canada prompted the Quebec Medical Association to draft a new schedule for GPs in an attempt to gain negotiating power for the province's physicians. Last year, the QMA's bid for negotiating power for specialists was rejected by government primarily because it was presented during fee negotiations with the Federation des médecins spécialistes du Québec. The QMA is appealing the decision, maintaining that negotiations are continuous and

text continued on page 307

Contributions to this column are welcome. Please send your material to: Mrs. Amy Chouinard, *Canadian Journal of Surgery*, PO Box 8650, Ottawa, Ont. K1G 0G8.

Table I—Selected Differences in the Preambles and Fees Listed in Provincial Schedules

	Quebec		Ontario	New Brunswick	Newfoundland	Prince Edward Island	Nova Scotia	Saskatchewan	Manitoba	Alberta	British Columbia
	GP	Specialist									
PREAMBLE											
Working hours	08:00–21:00	07:00–19:00	07:00–17:00	08:00–18:00	08:00–17:00	08:00–19:00	08:00–19:00	08:00–17:00	nm	08:00–16:59	08:00–18:00
Special trip charge								$15	$23.35	$28.25	–
Evenings	–	19:00–24:00	17:00–24:00	–	17:00–24:00	19:00–24:00	17:00–24:00	17:00–24:00	nm	17:00–22:59	18:00–23:00
Premium		+33%	+30%		+30%	+25%	–	–	–	–	–
Special visit fee			–		$36.20	$8.45	$32.22	$22	$23.25	$39.75	$29.40
Night premium	–	+50%	+50%	+25% (minimum $25.80)	+50%	+25%	–	–	–	–	–
Special visit fee	$31.85				$54.30	–	$32.22	$22	$23.35	$79.50	$41.30
Work week	Mon–Sat	Mon–Fri	Mon–Fri	Mon–Fri	Mon–Fri	Mon–Fri	Mon–Fri	Mon–Fri	Mon–Fri	Mon–Fri	Mon–Fri
Surgical assistant's fee	21% of major fee + 10% of others	25% of major fee + 10% of others	$8.47/15 min to 2 h; $16.94/15 min thereafter + units for procedure	35% of surgeon fee	$9.67/15 min to 2 h; $19.34/thereafter + units for procedure	20% (minimum $27.45) 25% if surgeon/referring GP	25% (minimum $15)	$62.80/1 h; $18/15 min thereafter	Procedures of ≤ $132.75 = $52.90 (+ $17.90 for every $88.05)	$66.25 for 1 h; $16.50/15 min thereafter	Procedures ≤ $97.50 = $41.10; $97.50–291 = $76.50; $291 – 485 = $105
Asst. special trip	–	–	$28.50	–	–	–	$32.22	$15	$23.35	–	–
Evening			$42.80		$36.20		$32.22	$22	$23.35	$39.75	$29.40
Night			$42.80		$54.30		$32.22	$30	$23.35	$79.50	$41.30
Premium, evening	–	–	+40%	–	+40%	–	–	–	–	–	–
night			+50%		+50%						
Preoperative care included in procedural fee		All: 90 d	All except major consultation	All: 30 d	All except major consultation	All except major consultation	All: 30 d	All except major consultation	All unless documented	All: 30 d	All: 30 d
Postoperative care included in fee		All: 15 d	All: 42 d	All: 42 d	All: 8 wk	All: 30 d	All: 30 d	All: 42 d	All: 42 d	All: 42 d	All: 42 d
SUBSEQUENT PROCEDURES, % of normal fee											
Same anesthesia, same incision	50	50	85	50	85	50	50	75	50	75	50
Same anesthesia, new incision	50	50	85	75	85	50	65	75	75	75	75
Same anesthesia, different surgeon	nm	100	nm	nm	nm	100	nm	nm	100	100	100
New condition, within 14 d	nm	nm	85	nm	nm	100	nm	nm	nm	nm	nm
PROCEDURES, $											
Arthroscopy, hip	106.10	150	82.50	133.44	104.75	133.55	179	105	99.75	121	93
With biopsy	+26.50	175	+9.25								
With foreign body removal		200									
Arthrotomy, hip	238.70	225	256	258.24	325	356.15	313.25	265	293	362	351
Reduction, hip											
Closed	103.95	98	227.25	147.84	288.50	133.55	134.25	133	83	157.50	256
Open	206.85	195	344.60	295.68	437.50	311.65	313.25	292	293	518	428
Bladder, cystectomy											
Partial	265.20	250	313.50	295.68	398	356.15	358	299	427	249	341
With reimplant of ureter	371.30	350	453.70	398.40	576	445.20	483.30	362	514	405	499
With reimplant of ureters	636.55	600	602.60	nm	765	nm	nm	nm	514	572	nm
Urethroscopy	30.95	50	29.10	44.16	37	71.20	53.30	53.60	29.85	129.50	96.50
With biopsy	+10.05		64.20	44.16	81.50	71.20	53.30	nm	nm	nm	nm
With foreign body removal	77.40	75	140.20	110.40	178	133.55	133.25	92	119.50	nm	nm
Gastroscopy	51.60	50	74.80	88.32	95	133.55	134.25	83	97.25	103.50	96.50
With biopsy, photo	+15.50	+15	74.80	88.32	95	133.55	134.25	83	97.25	nm	nm
With foreign body removal	+32	+35	81.10	147.84	103	nm	nm	128	nm	nm	nm
Wedge resection for ulcer	230	230	330.80	292.80	420	320.45	331.15	542	493	474	558
Total gastrectomy	620	620	773.50	650.88	982	712.25	626.50	669	618	921	826
Gastroduodenostomy/ gastrojejunostomy	254.60	260	330.80	292.80	420	320.45	322.20	327	370	542	315
With vagotomy	350.10	370	+130	407.04	+165	445.20	429.60	467	+88.50	758	461
Pericardiectomy											
One side open	297.05	350	519.90	IC	660	356.15	358	542	688	610	582
Both sides open	488.05	460	819.20	IC	1040	534.15	537	nm	nm	nm	nm
Cardiotomy											
With exploration	265.20	250	430.10	IC	546	445.20	537	440	558	469	428
With foreign body removal	328.90	310	519.90	IC	660	445.20	447.50	444	nm	836	nm

nm = not mentioned; IC = independent consideration.

MARK E. BOYD, MD, FRCSC, FRCOG

Care of the Ureter in Pelvic Surgery

Maintaining the integrity of the ureter is crucial in pelvic surgery. The ureter is best safeguarded by routine intraoperative exposure, which will also allow immediate recognition of injury to it. If doubts over possible injury persist, it is best to open the bladder. The flux of urine from the ureteric orifices or the retrograde passage of catheters will then confirm or deny clinical suspicions. If specialist help is unavailable, the pelvic surgeon must be able to perform simple ureteric repairs or temporize in a way that allows the safe delay of definitive surgery. End-to-end ureteric anastomosis and ureteroneocystostomy are straightforward procedures that all pelvic surgeons should be familiar with. If they cannot be performed safely, the situation may be salvaged by draining the proximal ureter through the lateral abdominal wall; later, definitive surgery can be performed.

En chirurgie pelvienne, il est crucial de maintenir l'intégrité de l'uretère. Le meilleur moyen de sauvegarder l'uretère consiste à l'exposer de routine durant l'opération, ce qui permet également de reconnaître les lésions qui pourraient l'affecter. Si le doute persiste quant à des lésions possibles, mieux vaut ouvrir la vessie. L'écoulement d'urine à partir des orifices urétéraux ou le passage rétrograde de cathéters permettront alors de confirmer ou d'infirmer les soupçons cliniques. Si on ne peut compter sur l'aide d'un spécialiste, le chirurgien pelvien doit être capable d'effectuer des réparations simples des uretères ou de temporiser de façon à permettre de retarder l'opération définitive en toute sécurité. Les anastomoses urétérales bout-à-bout et les urétéronéocystostomies sont des interventions simples que tout chirurgien du bassin devrait connaître. Si elles ne peuvent être pratiquées sans risque, on peut sauver la situation en drainant l'uretère proximale via le côté de la paroi abdominale; la chirurgie définitive pouvant être remise à plus tard.

Pelvic surgeons are concerned with maintaining the integrity of the ureter, for, should injury occur, the postoperative course will often be complicated and frequently secondary surgery will be needed. This review will detail the circumstances surrounding the occurrence of ureteric injury and its management. The discussion of repair will be confined to that undertaken at the time of surgery; treatment of ureteric injuries that are first recognized in the postoperative period need not be immediate, and there will be time to obtain specialist help.[1-3]

The Injury

Two-thirds of all iatrogenic ureteric trauma occurs during gynecologic surgery, most often for abdominal hysterectomy or the excision of uterine adnexa.[1,4] The hysterectomy is frequently described as "straightforward" and "uncomplicated".[5] Pelvic inflammatory disease, endometriosis, uterine or ovarian tumours may displace the ureter and contribute to injury but not to the extent generally believed. Less common sources of injury are abdominoperineal or anterior resection of the rectum; the ureters are at risk near the point where the inferior mesenteric artery is ligated.[6,7] Vascular surgery also accounts for a number of ureteric injuries.[1] Hyperangulation and obstruction of the ureter may result from placing vaginal sutures more laterally than recommended during a Burch urethropexy.

The ureter is frequently injured while hemostasis is being obtained. Therefore, the more common sites of injury are deep in the pelvis, close to the point where the uterine artery crosses over the ureter and at the brim of the pelvis, where the ureter and ovarian vessels converge. The usual mechanism of injury is a crush or cut of a small portion (less than 1.0 cm) of an otherwise normal ureter. In gynecologic surgery for benign disease, the ureter is never "stripped" and thus its blood supply is rarely interrupted.[8,9]

Prevention

The traditional means of avoiding operative injury to the ureter is to stay well clear of it. For example, at hysterectomy, dissection is confined to the area in close proximity to the uterus. There are, however, difficulties with this approach. At times, pelvic lesions may so distort the ureter's pathway that a dissection, believed to be far from the ureter, may place it in jeopardy, or a pelvic mass may limit the surgeon to dissection in areas where direct contact with the ureter is unavoidable. At other times, the ureter may be in its usual position, but landmarks are lost and the ureter is inadvertently clamped, incised or ligated.

The best way to avoid ureteral injury is to expose it through a retroperitoneal dissection.[5] This dissection is not difficult and it allows an easier approach to pelvic surgery. A further advantage is the immediate discovery of any ureteric damage.[10,11]

Retroperitoneal dissection is initiated by cutting the round ligament at its midpoint. The peritoneum is incised in a caudal direction for 4.0 cm and the incision

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Accepted for publication Jan. 15, 1987

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extended cranially well above any pelvic disease. This cranial-directed incision is placed lateral to the ovarian vessels (infundibulopelvic ligament).

The medial edge of the cut peritoneum is grasped with two or three small curved forceps and elevated medially. Immediately beneath the peritoneum a condensation of the endopelvic fascia will be seen; the small blood vessels in this layer are best cauterized on sight. This prevents staining of tissues and enhances the dissection. The endopelvic fascia is then incised for 3 to 4 cm, giving access to loose retroperitoneal tissue that is characteristically free of adhesions. The retroperitoneum in this location can easily be divided with the forefingers.

It is emphasized that dissection must be directed both medially and posteriorly to avoid the common and internal iliac blood vessels. As the surgeon proceeds along the medial leaf of the peritoneum, the ovarian vessels are first encountered; just deep to them, the ureter will be found attached to the peritoneum. The ureter is usually easy to identify by the peristaltic movements, its "macaroni-like" appearance and the fine blood vessels that cover it. The ureter runs from the brim of the pelvis (which it enters by crossing above the bifurcation of the common iliac blood vessels) downward to the medial aspect of the lateral cervical ligament. It then enters the ureteric canal and passes beneath the uterine artery, advancing through the vesicouterine ligament into the bladder.

The whole course of the ureter (from the pelvic brim to the ureteric canal) can be traced, with little bleeding, in a few moments. A further advantage is that the location of the ovarian and uterine vessels has been established.

Recognition

It should be emphasized that most ureteral injuries occur after uncomplicated surgery when injury to the ureter has not been considered.[5] Less than one-third of injuries are recognized intraoperatively; delay compounds the potential complications. Immediate repair is usually straightforward and the postoperative result is generally good.[1,7,12]

During surgery injury may be suspected if an unusual collection of fluid is noted in the pelvis or if dissection was especially difficult in the vicinity of the ureter.

When the surgeon suspects injury but is not willing to expose the ureters, fearing dissection may in turn cause injury, the use of indigo carmine or methylene blue dye is called for. After the administration of 40.0 mg of furosemide (Lasix), a 5-ml intravenous injection of the dye is given. Discolouration of the fluid in the pelvis indicates ureteric injury. If the concern is one of ureteric occlusion, patency can be established by opening the bladder through a vertical cystotomy and noting the escape of jets of blue urine from the ureteric orifices.

If uncertainty persists, the suspect ureters should be catheterized; a no. 8 French infant feeding tube serves this purpose admirably. The catheterization may not be easy, for a mucosal flap can prevent passage of the catheter. In such an event, tension applied to a suture, temporarily placed in the middle of the interureteric bar, will straighten the intramural ureter and permit the catheter's passage.

Repair of the Injury

Usually only a small portion of a normal ureter is damaged, but there is a regrettable tendency to underestimate such injuries.[13,14] Segments crushed by suture or forceps should be excised and partial transections should be treated as though they were complete. Another mistake is failure to ensure that the repair performed is well clear of any pelvic disease.

End-to-End Anastomosis

This method of repair is an uncomplicated procedure that restores anatomy and preserves the normal vesicoureteric junction. Some years ago it was discouraged because stenosis frequently occurred at the site of repair. This problem can be minimized by enlarging the anastomotic site and preventing the pooling of urine in the retroperitoneal space. Urine in this location excites a fibrotic response; this fibrosis outside the ureter and the scarring of the ureter itself at point of injury may result in obstruction of the ureter.

To reduce pooling of urine, the anastomosis is made as watertight as possible and the area is drained with a Jackson–Pratt drain. Years ago it was taught that urine must be diverted away from the anastomotic site by a vertical relieving incision.[15] It is now argued that in the absence of radiation injury or infection diversion is unnecessary.[16]

A recent introduction is the internal splint that not only provides a conduit for the urine and thus diminishes leakage but also gives support to the anastomosis itself. The malleable double-J ureteral catheter is particularly attractive because it is inert and retains its place in both the renal pelvis and the bladder. The use of such a stent can be seen as an added safety measure and its use is encouraged.

The following technique of anastomosis is suggested. The ureter is first debrided to ensure viability and mobilized to prevent excessive tension on the suture line. The opposing ends are spatulated to enlarge the circumference of the anastomosis, and a double-J ureteral catheter is inserted. A watertight anastomosis is then performed using interrupted 5-0 polyglycolic acid sutures through the full thickness of the ureteral wall. A Jackson–Pratt drain is placed close to the anastomotic site and brought out through a separate incision.

Ureteroneocystostomy

The ureteral injuries that occur at hysterectomy are most often deep in the pelvis where limited access makes end-to-end anastomosis technically difficult and increases the risk of ischemic damage to the distal segment of the ureter. In these circumstances ureteroneocystostomy, using an antireflux technique, is advised. Urologists have become increasingly confident with this technique as a result of their renal transplant experience.

The principles of repair are similar to those already mentioned. The ureter must be viable and the anastomosis with the bladder must be tension-free and tunnelled, but need not be in the region of the trigone. Tension-free anastomosis is best achieved by mobilizing the bladder, as in the Burch procedure, but dissection of the proximal portion of the ureter should be limited. After viability of the ureter is ensured by resection of the damaged end, it is brought through the muscle wall of the bladder by means of a submucous tunnel — the length of the tunnel should be five times the diameter of the ureter. A mucosa-to-mucosa anastomosis is performed between the ureter and bladder. A double-J ureteral catheter is inserted and a Jackson–Pratt drain placed retroperitoneally, close to the anastomotic site.

Anastomotic disruption is usually caused by tension, which can be avoided by mobilizing the bladder, but if adequate mobilization is not possible, a psoas hitch or the Boari–Ockerblad flap should be used.[17] Both procedures permit upward extension of the bladder so it comfortably reaches the injured ureter. Because of its simplicity and better results, the psoas hitch has generally replaced the Boari–Ockerblad flap.[14,18] The hitch is performed by first mobilizing the bladder, and with two fingers in its interior, a horn is created which is sutured to the iliopsoas fascia. The site chosen is lateral to and above the external iliac artery; care is taken not to injure the genitofemoral nerve which overlies the psoas muscle in that area. Anastomosis with the ureter is carried out as described.

Other Maneuvers

There are circumstances in which the suggested repairs are not possible; for example, a large segment of ureter may be missing and a urologist is not available. A procedure that allows renal

drainage but does not place the other kidney in jeopardy or compromise later definitive repair, is needed. In these cases, a double J ureteral catheter or an infant feeding tube is inserted into a linear ureterostomy well above the site of injury. The catheter or tube is secured and brought out through the abdominal wall. The ureterostomy is closed in a watertight manner and the retroperitoneum drained.[14]

Experienced urologists have other options, including transureteroureterostomy, ureteral replacement by a segment of ileum, mobilization of the kidney, renal autotransplantation and nephrectomy. These procedures should not be attempted by the less-experienced surgeon, as further damage to the urinary tract may result.[19] Simple ligation of the ureter for autonephrectomy should rarely be done, for infection and fistula formation frequently result.[20]

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continued from page 229

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continued on page 251

Symposium on the Perioperative Management of Abdominal Aortic Surgery

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1. Preoperative Management of the Patient With Coronary Artery Disease Before Abdominal Aortic Surgery

Coronary artery disease accounts for more than half of the morbidity and mortality associated with abdominal aortic surgery. To improve the results of vascular surgery, the risk of perioperative cardiac ischemia should be evaluated in each patient. Routine coronary angiography demonstrated severe correctable coronary artery disease in 14% of patients who had no history or electrocardiographic evidence of coronary artery disease. Exercise testing before abdominal aortic aneurysm repair will identify patients at high risk of cardiac ischemia. Dipyridamole–thallium imaging will identify high-risk patients before surgery for aortoiliac occlusive disease. Some patients with symptomatic coronary disease who are at extremely high risk should undergo preoperative coronary revascularization. Others should have their vascular surgery deferred, because their cardiac risk may exceed the anticipated benefit of the vascular surgery. Patients at moderate risk may need more intensive intraoperative monitoring. Patients without evidence of cardiac ischemia with stress may undergo vascular surgery with a low risk of perioperative cardiac ischemia. Finally, patients who have evidence of ischemic heart disease should be considered for coronary revascularization following successful vascular repair in order to prolong their survival.

La maladie artérielle coronarienne représente plus de la moitié de la morbidité et de la mortalité associées à la chirurgie aortique abdominale. Pour améliorer les résultats de la chirurgie vasculaire, le risque d'ischémie cardiaque périopératoire doit être évalué chez chaque patient. L'angiographie coronarienne de routine démontre une sévère et corrigible maladie artérielle coronarienne chez 14% de patients sans antécédents ni évidence électrocardiographique de maladie artérielle coronarienne. Des tests d'exercice avant correction de l'anévrisme aortique abdominal permettent d'identifier les patients présentant un risque élevé d'ischémie cardiaque. La visualisation au dipyridamole thallium permet d'identifier les patients à hauts risques avant chirurgie lors de maladie occlusive aortoiliaque. Certains patients atteints de maladie coronarienne symptomatique et présentant un risque extrêmement élevé doivent subir une revascularisation coronarienne préopératoire. D'autres doivent avoir leur chirurgie vasculaire retardée car le risque cardiaque peut excéder le bénéfice escompté après la chirurgie vasculaire. Les patients à risque modéré nécessitent une surveillance intraopératoire plus intensive. Les patients sans évidence d'ischémie cardiaque lors de stress peuvent subir la chirurgie vasculaire avec un risque minime d'ischémie cardiaque périopératoire. Finalement, les patients présentant des évidences de maladie cardiaque ischémique doivent être retenus pour une revascularisation coronarienne, suite à une chirurgie vasculaire réussie, afin de prolonger leur survie.

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Presented as part of a symposium on the perioperative management of abdominal aortic surgery at the 8th annual meeting of the Canadian Society for Vascular Surgery, held in conjunction with the 55th annual meeting of the Royal College of Physicians and Surgeons of Canada, Toronto, Ont., Sept. 24, 1986

Supported by the Heart and Stroke Foundation of Ontario

Accepted for publication Dec. 18, 1986

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More than half the perioperative morbidity and mortality associated with abdominal aortic surgery results from coronary artery disease.[1–5] In addition, the poor long-term survival after successful abdominal aortic surgery usually results from coronary artery disease.[6–11] The vascular surgeon must carefully assess the risks of perioperative cardiac ischemia in each patient before elective vascular surgery is carried out.

Improved techniques of intra- and postoperative management have decreased the risks of abdominal aortic surgery and have reduced the frequency of perioperative deaths from cardiac ischemia. However, the incidence of myocardial infarction postoperatively remains substantial,[5] and a perioperative myocardial infarction may limit long-term survival after vascular surgery.[6,7] Perioperative cardiac injury may also induce persistent cardiac symptoms postoperatively. Vascular surgeons therefore should attempt to minimize perioperative myocardial ischemic injury.

Extent of Coronary Artery Disease

Hertzer and colleages[12] performed routine preoperative coronary angiography in 1000 patients who presented at the Cleveland Clinic between 1978 and 1983 for consideration of elective vascular reconstruction. In patients with an abdominal aortic aneurysm, 6% had normal coronary arteries, 29% had mild to moderate coronary artery disease, 29% had compensated coronary artery disease, 31% had severe correctable coronary disease and 5% had severe inoperable disease. The Cleveland Clinic group carefully reviewed the coronary angiograms and the ventriculograms to develop these complex categories. They classified coronary artery disease as mild when no lesion in a major coronary artery exceeded 70% stenosis. Coronary artery disease was

advanced but compensated when a major coronary artery was stenosed but there was no immediate indication for myocardial revascularization either because the region at risk was adequately perfused by collateral vessels, or because it had already been replaced by a scar from a previous myocardial infarction. Coronary artery disease was considered severe but correctable when a severe coronary artery stenosis supplied a region of functioning myocardium which was at risk for a myocardial infarction. They classified disease as severe and inoperable when there was diffuse distal disease or inadequate ventricular function.

The Cleveland Clinic group compared these results of coronary angiography to their suspicion of coronary artery disease from clinical examination (Table I). Among the 1000 patients (263 with abdominal aortic aneurysms, 381 with lower limb ischemia, 295 with cerebrovascular disease and 61 with other vascular problems), 554 were suspected of having coronary artery disease before angiography because of a history of myocardial infarction or angina pectoris and electrocardiographic evidence of a previous infarction, ST- and T-wave changes or a left bundle-branch block. Severe correctable coronary artery disease was found in 34% of those suspected of having coronary artery disease before angiography (Table I) compared with 14% of the patients who had no clinical evidence of coronary artery disease. Therefore, the patient's history and an electrocardiogram may not provide a reliable guide to those at high-risk for perioperative ischemic injury.

At the Cleveland Clinic, 250 patients underwent coronary angiography before abdominal aortic aneurysm repair. Eighty patients (32%) had severe correctable coronary artery disease. Of the 250 patients, 76 underwent coronary artery bypass grafting and 4 (5.3%) died; 206 patients then underwent abdominal aortic aneurysm repair and 7 (3.4%) died. Overall there were 282 operations, resulting in 11 deaths (3.9%). In the entire series only one of the patients who had undergone coronary bypass grafting preoperatively died during or after peripheral vascular surgery.

From their experience, Hertzer and colleagues[12] recommended a modified approach to identify patients with severe but correctable coronary artery disease. They recommended preoperative coronary angiography for those with clinical indications of coronary artery disease. If a major cardiac region was jeopardized by a critical stenosis, inadequately perfused by collaterals and not damaged by a previous infarction, they recommended preoperative coronary revascularization. In patients who had no clinical indication of coronary artery disease, they recommended extensive noninvasive testing to identify the 14% of patients who will have severe correctable coronary artery disease.

Noninvasive Testing

Two-dimensional echocardiography will identify wall-motion abnormalities from a previous myocardial infarction but will not detect regions at risk of ischemia or infarction with stress. Patients with an abdominal aortic aneurysm can undergo exercise testing to evaluate their risk of perioperative ischemic injury. Exercise testing with or without thallium imaging[13] may identify the extent of inadequately perfused cardiac regions during exercise. Rest and exercise nuclear ventriculography will identify global or regional ventricular dysfunction with exercise suggesting severe correctable coronary artery disease.

Patients with aortoiliac occlusive disease usually cannot undergo standard exercise testing. Thallium imaging before and after intravenous administration of dipyridamole may identify patients at high risk of perioperative ischemic injury. Boucher and colleagues[14] at the Massachusetts General Hospital evaluated 54 patients with suspected coronary artery disease before they underwent peripheral vascular surgery. Six had abnormal thallium test results and underwent coronary bypass surgery before the abdominal aortic procedure, with no cardiac complications. Twenty patients had normal thallium scans and 12 had persistent thallium defects (suggestive of a previous myocardial infarction); all 32 underwent vascular surgery without cardiac ischemic complications. Sixteen patients had evidence of ischemia (thallium redistribution) with stress (dipyridamole) and 8 suffered cardiac ischemia perioperatively. Four patients had a myocardial infarction perioperatively and three of them died. Four had angina and transient electrocardiographic changes and two underwent successful coronary bypass surgery.

Therefore, exercise testing or dipyridamole–thallium imaging may identify patients who are at high risk for cardiac ischemia perioperatively, but the management of these high-risk patients remains controversial.

Preoperative Management of Coronary Artery Disease

Figure 1 illustrates the preoperative management protocol that we have used at the Toronto General Hospital to evaluate patients before performing abdominal aortic surgery. After a complete history, a physical examination and an electrocardiogram, most patients can be divided into one of three groups: (a) a history of previous cardiac events, (b) likely to have a cardiac condition or (c) unlikely to have a cardiac condition. Preoperative evaluation should attempt to identify patients who are at high risk of ischemic injury perioperatively (severe stenosis in a vessel supplying a large segment of functioning myocardium) rather than identify patients with a cardiac condition. Those who have severe ventricular dysfunction but do not show evidence of ischemia with stress will require intensive monitoring intraoperatively, but they may not be at risk of ischemic events perioperatively and may not benefit from preoperative coronary revascularization. Patients who have angina that is poorly controlled with full medical therapy should probably undergo coronary angiography preoperatively. The remaining patients should probably undergo noninvasive testing that includes an exercise test (with electrocardiographic monitoring or nuclear ventriculography) or a

Table I—Angiographic Classification of Coronary Artery Disease (CAD) Before Vascular Surgery at the Cleveland Clinic

Angiographic classification	Suspected CAD,* %	No indication of CAD, %
Normal	4	14
Mild	18	49
Compensated	34	22
Severe, correctable	34	14
Severe, inoperable	10	1

*Suspected CAD = cardiac history or an abnormal electrocardiogram.

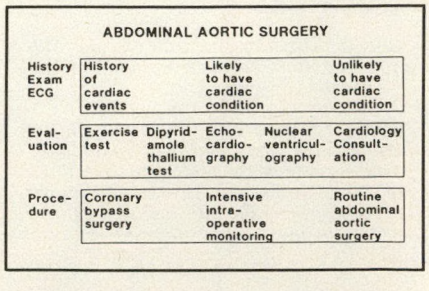

FIG. 1—Preoperative management protocol. Careful history, physical examination and electrocardiogram will determine likelihood of cardiac disease, but noninvasive evaluation may be required preoperatively to identify those patients who have evidence of cardiac ischemia with stress and severe, correctable coronary artery disease. Management of these patients requires careful assessment of risks and benefits of abdominal aortic surgery.

dipyridamole–thallium test (for those who cannot exercise). An echocardiogram may identify regional abnormalities of wall motion or dysfunction of the cardiac valve. A cardiology consultation may help in selecting the appropriate tests. Noninvasive testing may identify the 14% who have severe correctable coronary artery disease without a history of a cardiac condition and the 34% with a history of a cardiac condition who have severe correctable coronary artery disease.

After preoperative noninvasive evaluation, most patients can be classified into one of three groups: low, moderate and high probability of suffering cardiac ischemia perioperatively. Patients with a low probability should undergo routine abdominal aortic surgery. Those with a moderate probability may require extensive intra- and postoperative cardiac monitoring. Experienced anesthetists employing intensive monitoring can provide excellent hemodynamic stability during operation that may reduce possibility of cardiac ischemia. Patients who have evidence of myocardial ischemia with stress should have the risks and benefits of the vascular procedure carefully reviewed. Some should undergo vascular surgery with intensive monitoring. Some should be considered for coronary angiography and preoperative coronary bypass surgery. For some patients with severe coronary artery disease, the risks of the vascular procedure may outweigh the potential benefits. In these patients vascular surgery should be delayed.

Conclusions

Cardiac asssessment before abdominal aortic surgery may reduce the incidence of cardiac ischemic events perioperatively. Preoperative coronary angiography and bypass surgery should be reserved for patients with documented, severe ischemia in response to stress. Patients with compensated coronary artery disease or ventricular dysfunction can usually undergo vascular surgery with extensive intraoperative monitoring.

Finally, the long-term survival of patients after successful abdominal aortic surgery may depend on the extent of their coronary artery disease.[6–11] Therefore, some patients who had evidence of myocardial ischemia on preoperative noninvasive testing should be referred for coronary angiography postoperatively. Patients with critical anatomy (stenosis of the left main coronary artery) and those whose ischemia cannot be controlled with medical therapy should be referred for late postoperative coronary revascularization. An increased awareness of the risks of coronary artery disease among vascular surgeons may improve the long-term results of abdominal aortic surgery.

This report was prepared with the assistance of my vascular surgical colleagues at the Toronto General Hospital, and I would particularly like to thank Dr. K.W. Johnston for his innovative ideas. I thank Ms Eslyn Kesper for preparing the manuscript and Ms M. Mindy Madonik for preparing the figure.

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2. Anesthetic Considerations in Abdominal Aortic Surgery

Contributions of the anesthetist to the management of patients requiring abdominal aortic surgery must complement the roles of the cardiologist and vascular surgeon. Identification of risk factors and assessment of the degree of functional impairment of vital organs allows the anesthetist the best opportunity to provide the vascular patient with as stress-free a surgical experience as possible.

Patient profiles, monitoring, induction and maintenance phases in the administration of the anesthetic, and concerns of the early postoperative period are discussed from the anesthetist's perspective. General principles are emphasized. Specific details of assessment and management must be guided by local experience and resources.

Les contributions de l'anesthésiste à la surveillance des patients nécessitant une chirurgie aortique abdominale doivent compléter les rôles du cardiologue et du chirurgien vasculaire. L'identification des facteurs de risques et l'établissement du degré de détérioration fonctionnelle des organes vitaux permettent à l'anesthésiste de fournir au patient de meilleures garanties d'absence de complications lors de la chirurgie vasculaire.

Les profils des patients, le monitoring, les phases d'induction et de maintien lors de l'administration de l'anesthésique, ainsi que la surveillance du stade précoce postopératoire sont discutés sous la perspective d'un anesthésiste. Les principes généraux sont dégagés. Les détails spécifiques d'évaluation et de contrôle doivent être guidés par l'expérience locale et les ressources disponibles.

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Presented as part of a symposium on the perioperative management of abdominal aortic surgery at the 8th annual meeting of the Canadian Society for Vascular Surgery, held in conjunction with the 55th annual meeting of the Royal College of Physicians and Surgeons of Canada, Toronto, Ont., Sept. 24, 1986

Accepted for publication Dec. 18, 1986

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Mutual understanding of the respective contributions of medicine, surgery and

anesthesia can benefit the patient who presents with abdominal aortic disease. Advances in technique and aggressive monitoring have become the benchmarks of anesthesia's contribution.

This discussion will outline the extent of anesthesia's role. I shall begin with some comments on patient profile and monitoring, then consider induction, maintenance and the early postoperative period.

Patient Profile

The medical profile of a patient scheduled to undergo abdominal aortic surgery has been well documented. Given its generalized nature and the host of predisposing factors contributing to its onset, patients with atherosclerosis who require abdominal aortic surgery may present with disease and functional impairment of the heart, lungs, kidneys and brain.

By interviewing the patient preoperatively and reviewing the investigative data on the chart, the anesthetist can assess the functional reserve of the vital organs.

Myocardial function has repeatedly been demonstrated to be the most important factor in determining patient outcome after aortic surgery. Through a history of congestive failure or limited physical activity, the patient with functional impairment of the left ventricle will be relatively easy to identify. The patient without an abnormal cardiac history presents a more difficult problem. Here, the extent of coronary artery disease is unknown, as is the likely response of the left ventricle to the critical events of the perioperative period. A normal resting electrocardiogram and an exercise electrocardiogram that is limited by the physical constraints of peripheral vascular disease leave the question of functional reserve and myocardial ischemic risk unanswered. The expense and risks of coronary angiography preclude its routine use in patients scheduled for vascular surgery. Dipyridamole–thallium scanning as reported by Boucher and associates[1] appears to offer a solution to this major gap in our knowledge. Patients may be separated, preoperatively, into low- and high-risk categories for myocardial ischemia and dysfunction. Their management would differ, but the objective of vascular surgery completed successfully with the least risk of perioperative myocardial ischemia and its sequelae could be achieved in a larger percentage of patients. If this imaging technique proves reliable, it may become a routine investigation before major vascular surgery.

With the high rate of smoking in this group of patients, pulmonary dysfunction is common and may complicate the perioperative period. Altered response to fluctuations in hydrogen-ion concentration and arterial oxygen content coupled with the depressant effects of anesthetic agents and narcotics can place these patients at greater risk than others. Increased pulmonary secretions and disturbances in chest-wall mechanics contribute to morbidity.

To cope with the stresses imposed during the perioperative period, the kidneys must maintain three functions — a glomerular filtration rate adequate to maintain nonuremic blood chemistry, the ability to autoregulate intrarenal blood flow over a range of blood pressures and have tubules capable of controlling water and electrolyte fluxes. Consequently, the anesthetist will review the creatinine index, a range of the patient's blood-pressure determinations and the specific gravity on a urine specimen taken before surgery.

Preinduction

Application of currently available monitoring techniques to patients who undergo abdominal aortic surgery allows the anesthetist to follow closely physiological functioning intraoperatively. Limitations of these monitoring techniques require extrapolation from actual information provided and is accepted clinical practice in the management of such patients.

During electrocardiographic monitoring, lead II will be adequate to interpret simple dysrhythmias but is considered inadequate for ischemia as only inferior wall events are chronicled. At a minimum, leads II and V5 must be displayed. Addition of an esophageal lead would allow the posterior myocardium to be monitored for ischemia. One must realize that ST depression is a late indicator of myocardial ischemia, and such changes on the oscilloscope are preceded by the following: regional myocardial dysfunction, decreased ventricular compliance with the appearance of new V waves on the wedge- or occlusion-pressure tracing, myocardial lactate production and angina pectoris in the awake patient.

Catheterization of the radial artery is universally accepted for monitoring, but its limitations must be appreciated in that both false high and false low pressures may be presented. For example, reverberation in an inadequately damped system will give false high systolic pressures and, similarly, low pressures may be artefactual. The electronically derived mean arterial pressure is more consistent in representing true events. Devices are now available to adjust damping in the catheter-transducer system without altering natural resonating frequencies.

Right atrial catheterization should be performed to determine right ventricular filling pressures. It also allows access to the central venous circulation for the infusion of vasoactive drugs.

Pulmonary artery catheterization is now common in monitoring patients who undergo major vascular procedures, and multifunction catheters are available. The catheter should be placed in the awake but sedated patient, preferably several hours before the scheduled surgery. This allows time to observe the response of the left ventricle to volume challenging as well as the degree of relationship between the pulmonary artery diastolic pressure and the pulmonary artery occlusion pressure. Convincing arguments have been made to justify the use of the pulmonary artery catheter in patients with good left ventricular function. Waller and associates[2] have shown that many hemodynamic episodes were missed or misinterpreted without the use of the catheter. Conners and colleagues[3] showed that the hemodynamic status of critically ill patients could not be predicted on the basis of clinical signs. The occlusion pressure was predicted correctly in only 42% of cases and the cardiac index in only 44%. Further, in 48% of cases correct information obtained from the pulmonary artery catheter monitoring prompted a change in drug therapy. Preoperative knowledge of the left ventricular ejection fraction appears not to predict a relationship between central venous pressure and pulmonary artery occlusion pressure in patients who undergo abdominal aortic surgery. Ansley and colleagues[4] suggested that both pressures must be independently measured to assess the ventricular filling pressures.

Ongoing assessment of renal function by observation of urine output is generally followed on an hourly or half-hourly basis. By convention, renal function is assumed to be normal if hourly flows greater than 0.5 ml/kg are maintained. During infrarenal cross-clamping, a reversible, functional oliguria commonly develops. Frequently observed and inadequately explained, this oliguria is self-correcting if adequate intravascular volume and systemic pressures are maintained throughout the procedure and into the recovery period.

Induction

Induction of anesthesia leading up to surgery is the first critical event in intraoperative management of the patient. It is a time requiring the closest attention to any alteration in functions. With careful titration of selected induction agents and with airway management, the patient is prepared for intubation. By observing individual responses to a series of graded stimuli, ranging from loss of sympathetic tone with lapse into uncons-

ciousness to the degree of pressor response to laryngoscopy without intubation, the anesthetist can judge both the level of anesthesia and degree of hemodynamic control. Dysrhythmias and severe hemodynamic changes are most likely to occur at this stage. Once intubation is complete, stimulation is minimal until the surgical incision is made. Throughout the induction sequence it is essential that the anesthetist have available the drugs necessary to treat detrimental alterations in hemodynamics (e.g., nitrates, inotropes, β-blockers and calcium antagonists).

Maintenance

The maintenance phase brings concerns in addition to preserving myocardial oxygen balance, such as fluid replacement, rate of decline in body temperature, degree of accumulation of anesthetic agents and alterations of ventilation–perfusion ratios in the lungs. It is during the maintenance period that two other critical events take place — cross-clamping and declamping of the aorta.

Fluid replacement for the majority of these patients requires a rigorous response to marked alterations in extracellular fluid and blood volume. Rate of replacement is guided by serial central venous and occlusion pressure readings, changes in the morphology of arterial waveform, urine output and ongoing estimates of blood loss. Crystalloid in the form of Ringer's lactate solution or normal saline is employed initially and replaced with a more osmotically active solution, such as hetastarch or stored plasma, later in the procedure. It is common to administer more than 1 L/h of crystalloid. Oxygen-carrying capacity is supported with packed cells as indicated, on the basis of initial hematocrit value and blood loss.

A second problem, hypothermia, has numerous adverse effects that include alterations in functioning of the following: anesthetic agents, the coagulation system, the oxyhemoglobin dissociation curve, performance of the myocardium and the ability to buffer or metabolize lactic acid. Maintenance of normal body temperature is difficult in the intraoperative period. The rate of decline of body temperature can be slowed by active warming of inhaled gases and intravenous fluids and must be considered part of the standard intraoperative care of these patients.

A third concern is the accumulation of anesthetic agents. Tissue uptake of fixed and volatile drugs, especially lipid-soluble drugs is a well-characterized component of anesthetic pharmacokinetics. The two main determinants of the rate and degree of tissue uptake are tissue blood flow and drug storage capacity. Constant doses of fentanyl citrate or isoflurane throughout the operation will result in a large tissue store requiring metabolization and excretion in the early postoperative period.

Problems accompany mechanical ventilation of the lungs. Monotonous fixed-volume ventilation will allow a progressive deterioration in ventilation–perfusion ratios, resulting in a progressive intrapulmonary shunt and subsequent impaired oxygenation. High peak airway pressures may result in a reduction in venous return to the heart. Positive end-expiratory pressure may alter pulmonary artery pressures and position of the intraventricular septum, thereby disturbing left ventricular stroke volume, but ongoing attention by the anesthetist will minimize these hazards. The anesthetist can manipulate ventilator settings to provide optimum gas exchange with a minimum of deleterious effects, aided by either a mass spectrometer or stand-alone oximeter and capnometer in conjunction with serial arterial blood-gas measurements.

Two critical events — cross-clamping of the aorta and subsequent declamping — require close cooperation between the anesthetist and the surgeon. Anticipation of these events allows for appropriate action to be taken to minimize the physiologic sequelae to sudden drastic alterations in peripheral vascular resistance and venous return. It is impossible to predict precisely the degree of response of either the myocardium or the myocardial oxygen supply–demand ratio to aortic cross-clamping. With information supplied by systemic and pulmonary pressure lines and close attention at the time of clamping, intervention with vasodilators or volume replacement, or both, will effectively counter adverse hemodynamic conditions. Declamping will again result in marked alterations in hemodynamic function. Vigorous volume replacement preceding declamping will counteract the hypotension commonly observed.

Early Postoperative Period

The hours immediately after abdominal aortic surgery are viewed by the anesthetist as a continuation of intraoperative management. Requirements for volume replacement, warming and the administration of analgesia and sedation continue.

Problems such as oliguria, coagulopathy or "breakthrough" hypertension may occur in the later stages of the maintenance period or *de novo* in the early postoperative period.

Renal failure developing perioperatively can seriously complicate the outcome in these patients. Prompt aggressive dialysis may improve results, although the death rate remains exceptionally high. Prevention seems the best course, so the anesthetist must constantly be concerned about renal function during the operation. Mechanisms available to prevent renal failure are similar, regardless of the etiology. These preventive measures include maintenance of renal blood flow, glomerular filtration rate and tubular flow. Urine volume remains the standard monitor of renal function intraoperatively. The quality of this urine is not routinely measured (e.g., specific gravity, sodium concentration, urine–plasma ratios). In addition, a mental checklist of the major determinants of urine volume (mean systemic arterial pressure, antidiuretic hormone, sympathetic tone, tubular osmotic load and plasma oncotic pressure) should be reviewed at intervals by the anesthetist. In response to a fall in urine output this checklist may suggest appropriate therapy (e.g., increase mean arterial pressure, crystalloid infusion or "renal dose" dopamine).

As with other major problems associated with this type of surgery, emergence of a bleeding disorder is often predictable. Rarely is intraoperative coagulopathy due to an isolated defect in the clotting system; it is usually multifactorial. Early signs of trouble such as persistent oozing from previously dry raw areas or failure to see clot formation in pooled blood will prompt the anesthetist to alert the blood bank to an anticipated increased need for blood and blood components. Tests to be performed might include prothrombin, partial thromboplastin and activated coagulation times, platelet count and measurement of fibrinogen and fibrinogen-split products. Early consultation with the hematology service can facilitate a prompt response from laboratory and blood bank. More importantly, expertise will be provided should the problem persist or worsen, although the mainstay of therapy for acquired coagulopathies in the perioperative period remains infusions of fresh-frozen plasma and platelets.

The appearance of an exaggerated hyperdynamic phase with hypertension and tachycardia early in the recovery period indicates that the patient has "broken through" the sedation and analgesia. Prompt appropriate use of vasodilators, narcotics, β-blockers and sedative hypnotics will prevent the potentially deleterious effects of increased myocardial work and oxygen consumption.

Conclusions

Postoperative care of the patient who has undergone abdominal aortic surgery will frequently pass from the anesthetist's hands upon transfer to the intensive care unit. By virtue of the preoperative evaluation and, more importantly, through

several hours of direct observation and care, the anesthetist has accumulated considerable information about each patient's response to various physiologic stresses. A great deal of information on the intraoperative course of the patient is contained in the anesthetic record. Unfortunately, interpretation of this record in the postoperative period is often difficult and sometimes impossible to those unfamiliar with the symbols and shorthand it contains. The result is either a potentially hazardous misinterpretation of intraoperative events or a disregard for information that may have a positive bearing on a postoperative problem. I would emphasize the need for direct communication among all specialties involved in care of the patient in the perioperative period.

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ALLAN D. CALLOW, MD, PH D, FACS

3. Postoperative Management of Patients Who Undergo Aortic Surgery

Because patients with occlusive or aneurysmal disease of the aorta frequently have coexisting disease in other important organ systems, complications after aortic surgery usually relate to the heart, lungs and kidneys. Postoperative care thus requires attention, in an intensive-care milieu, to respiratory, cardiac, renal and gastrointestinal systems, coagulation and, in rare instances, psychiatric problems.

Du fait que les patients souffrant d'occlusion ou d'anévrisme aortique sont fréquemment atteints de maladies au niveau d'autres organes vitaux, les complications après chirurgie aortique sont généralement reliées au coeur, poumons et reins. La surveillance postopératoire, en milieu de soins intensifs, nécessite une attention particulière aux systèmes respiratoire, cardiaque et gastro-intestinale, à la coagulation et, dans de rares cas, aux problèmes psychiatriques.

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Presented as part of a symposium on the perioperative management of abdominal aortic surgery at the 8th annual meeting of the Canadian Society for Vascular Surgery, held in conjunction with the 55th annual meeting of the Royal College of Physicians and Surgeons of Canada, Toronto, Ont., Sept. 24, 1986

Accepted for publication Dec. 18, 1986

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The patient with occlusive or aneurysmal disease of the aorta is generally in an age group in which coexisting disease in one of several organ systems is common and, hence, complications after aortic surgery usually relate to the heart, lungs and kidneys, rather than to the arterial reconstruction itself. Although the measurement of central venous pressure is a relatively reliable guide to cardiac function and volume status under normal circumstances, it is often inaccurate during stress and in the presence of cardiac and pulmonary disease. Swan–Ganz catheterization is therefore relied upon in these patients for more precise measurement of fluid and blood replacement.

Postoperative complications associated with intraoperative problems include the following: injury to the inferior vena cava and iliac veins, ureteral injury, intraoperative and postoperative thrombosis, intimal embolism, postoperative hemorrhage, stenosis or occlusion of an anastomosis, spinal cord ischemia and paraplegia and ischemia of the left colon. These are specific problems that are usually easily recognized and, except for spinal cord ischemia, amenable to correction.

Postoperative care requires attention to the following: intensive care monitoring, respiratory, cardiac, renal and gastrointestinal (stress ulceration) systems, bleeding and psychosis.

Monitoring

Initially the patient should be stabilized in an intensive care or recovery room where vital signs, urine output, cardiac rhythm and respiratory status can be continuously monitored for as long as necessary. Systemic blood pressure, heart rate and rhythm and arterial blood gases should be measured promptly and a chest x-ray film obtained to check endotracheal tube position and adequate lung expansion. Pre- and postoperative 12-lead electrocardiograms should be compared. If myocardial ischemia or infarction is suspected, blood samples should be submitted for measurement of cardiac isoenzymes, hematocrit, serum electrolyte values, blood glucose, prothrombin and partial thromboplastin times and platelet count. The most commonly mismanaged postoperative problem involves fluid administration. This must be meticulous, for these patients do not tolerate fluid overload. During aortic surgery, the patient often requires a large amount of fluid, which is retained postoperatively. Much of this is sequestered in the interstitial spaces where it remains until gradually mobilized and excreted in 48 to 72 hours. Inappropriate secretion of antidiuretic hormone results in sodium and water retention. Therefore, intravenous fluids for maintenance should be limited to approximately 80 ml/h or 1 ml/kg body weight of 5% dextrose and half-normal saline with 20 to 30 mmol/L of potassium chloride.

On arrival in the recovery room, these

patients are usually cold and vasoconstricted, and additional fluid may be necessary as rewarming and vasodilatation occur. A decreasing urine output, reduced blood pressure, tachycardia and lower cardiac filling pressures indicate a need for additional fluids together with packed red cells or whole blood, if available, if the hematocrit is less than 30%. A balanced salt solution, such as Ringer's lactate, is helpful.

We do not believe in early ambulation of patients after aortic or peripheral arterial reconstruction since many of them are hemodynamically unstable for several days. Incisional pain may be severe, causing tachycardia and hypertension, thus increasing the burden on the heart, and with the mobilization of excess fluid occurring 48 to 72 hours postoperatively, there may be expansion of the intravascular volume creating additional cardiac stress. It must be remembered that myocardial infarction occurs most commonly 3 days after major surgery.[1] Leg exercises against a foot board or other resistance-generating equipment improve venous emptying from calf muscles, increase blood flow to the legs and thus through the graft, maintain muscular tone and probably serve as prophylaxis against deep venous thrombosis.

Respiratory System

Support by a volume ventilator is generally necessary for several hours and sometimes several days. For the patient unable to breathe, controlled mechanical ventilation provides a preset tidal volume at a controlled frequency. For the patient who must remain intubated, an assist-control technique allows spontaneous breathing supplemented by assisted ventilations, thus preventing hypoventilation and respiratory acidosis. Intermittent mandatory ventilation provides good assistance and control. Between mandatory ventilations, the patient may breathe spontaneously, so that the mandatory ventilations can be progressively decreased as the patient's mechanics improve. Occasionally, those with reduced functional residual capacity will require continuous positive airway pressure or positive end-expiratory pressure as well as assisted ventilation. It must be remembered that high pressures (more than 15 cm H_2O) can decrease cardiac output.[2] Deep breathing and coughing are essential after extubation. Intermittent positive-pressure breathing is rarely necessary unless bronchodilators or mucolytic agents are to be given. If endotracheal stimulation and suction are necessary, adequate oxygenation should be assured and the pulse be monitored because of the occasional occurrence of arrhythmias.

Cardiac System

Incisional pain, increased sympathetic tone, and some as-yet-unaccounted-for elevation of the renin angiotensin activity may explain why hypertension is frequently associated with postoperative cardiac problems, producing excessive cardiac work. Cardiac work is the product of stroke volume, rate and systolic blood pressure. Thus, if hypertension is inadequately controlled, myocardial ischemia with failure may supervene. A useful guideline is to maintain systolic blood pressure within 15% above or below the preoperative level. Because arterial lines tend to record higher pressures than the sphygmomanometer, a comparison between the two should be made before an antihypertensive medication is administered. Often, the relief of pain by a small dose of morphine (2 to 5 mg) will moderate the hypertension and the tachycardia.

For more immediate action, sodium nitroprusside may be given — 50-mg doses in 250 ml of 5% dextrose in water at a rate of 0.5 to 1.0 $\mu g/kg \cdot min^{-1}$. This may be increased to a maximum of 10 $\mu g/kg \cdot min^{-1}$.[3] Blood pressure must be monitored continuously, for, although this drug has a short duration of action, possibly not more than 10 minutes, it is a potent vasodilator. It must be used cautiously in those with hepatic or renal insufficiency, either chronic or transient, inasmuch as it is metabolized to thiocyanate. Prolonged infusion or excessively high doses may lead to cyanide intoxication.

The combination of tachycardia and hypertension is particularly stressful on the myocardium. Hypovolemia may be detected by decreased urine output, falling blood pressure, especially with deep inspiration, and low heart filling pressures. If hypovolemia is not the cause, persistent tachycardia may be due to cardiac failure or increased sympathetic tone. A pulmonary capillary wedge pressure greater than 18 to 20 mm Hg suggests left ventricular failure for which digoxin may be administered. In the absence of hypovolemia and evidence of cardiac failure, tachycardia may be controlled by the administration of propranolol 0.1 to 1.0 mg. Again, continuous electrocardiographic and blood-pressure monitoring should be maintained, for rapid slowing of the heart rate may occur. An occasional side effect, particularly in patients with chronic obstructive airway disease, is bronchospasm; this is less likely to be produced when patients are given the β-blocker metoprolol (Lopressor), 50 mg orally twice daily. It should be used with care in the insulin-dependent diabetic for β-blockade may mask sympathetic signs of hypoglycemia.

Low cardiac output and hypotension must also be considered in association with hypovolemia, for pump failure or cardiogenic shock is a main cause of low cardiac output. Severe chronic cardiomyopathy or an intraoperative myocardial infarction makes it a likely possibility. So, too, must septic shock and cardiac arrhythmias be considered in determining the cause of low output. Cardiac output is a product of heart rate and stroke volume.[4,5] The principal determinants of stroke volume are preload, afterload and myocardial contractility.

Preload is the end-diastolic filling pressures of the right and left hearts. Stroke volume is directly related to preload. Afterload is the resistance against which the left ventricle contracts — the force-resisting fibre contraction. It is inversely related to stroke volume.

Contractility describes factors such as β-adrenergic agonists and antagonists that modify the force of contraction independent of pre- and afterload. Systemic blood pressure depends directly on cardiac output and peripheral vascular resistance.

In the low-perfusion state, both blood pressure and urine output drop, one frequently sooner or to a greater extent than the other. A 5- to 10-ml/kg bolus of fluid may improve perfusion. Preload is measured by ventricular filling pressure at end of diastole, which means central venous pressure for the right ventricle and pulmonary capillary wedge or left atrial pressure for the left. Filling pressures should be brought into the normal range before administering drugs or mechanical support. The best drugs to improve cardiac output are the inotropic and β-adrenergic group — isoproterenol or dopamine/dobutamine. Mechanical support may be necessary if these drugs fail. Cardiac output can also be increased by use of vasodilators to decrease afterload. A pulmonary capillary wedge pressure above 18 to 20 mm Hg may lead to pulmonary congestion and a rise above 30 mm Hg to frank pulmonary edema. It must be remembered that excessive positive end-expiratory pressure may falsely elevate central venous and pulmonary artery pressures, thus masking the true cause — hypovolemia. Nitroprusside or nitroglycerin, administered intravenously, may help to reduce afterload by vasodilatation. It may be maintained on a more sustained basis later in the postoperative period with hydralazine, captopril, nitrates and other parenteral vasodilators.[6] Myocardial contractility is most effectively enhanced by the inotropic drugs dobutamine, dopamine and digitalis. In acute cardiac failure, dobutamine is the agent of choice, acting rapidly to decrease both preload and afterload.

If bradycardia is the cause of the low output state, the use of atropine may accentuate the arrhythmias, making cardiac pacing necessary. Coronary perfusion may fall as a consequence of cardiac arrhythmias, and there may be associated diminished flow in other organ systems. There are a number of mechanisms that produce arrhythmias in the immediate postoperative course; hypothermia (quite common), hypoxia, hypo- or hyperkalemia, disturbances of acid-base metabolism and, of course, myocardial infarction. When premature ventricular contractions are common (often in the cold and acidotic patient) and occurring at a rate of more than 6/min, or associated with runs of ventricular tachycardia or bigeminy, treatment is required. Lidocaine (50 to 100 mg intravenously) administered as a bolus will often solve the problem. Rarely is a continuous infusion necessary.

Renal System

Renal failure in the absence of chronic renal insufficiency can usually be avoided by careful fluid management. The diabetic patient who has chronic renal insufficiency or is dehydrated is more likely to suffer failure postoperatively than the normal individual. An output of less than 15 ml/h in the presence of rising blood urea nitrogen and creatinine values makes for early recognition. In the high-risk patient, adequate preoperative hydration in the intensive-care unit, Swan–Ganz monitoring on the night before surgery and the administration of mannitol before infrarenal aortic clamping have been most effective in limiting this problem.[7] The ideal urine output in the normal patient is 30 to 60 ml/h. High-output renal failure (more than 60 ml/h) in an intravascular-volume-depleted patient is occasionally seen and can be confirmed by the finding of a urine plasma-creatinine ratio of less than 10. Because the normal kidney retains sodium (urine value 20 to 40 mmol/L), a urine sodium value of 80 to 100 mmol is a second indicator of renal failure. Finally, failure to concentrate urine (specific gravity of 1.010) or a urine–plasma osmolarity ratio of less than 1.3 is an additional sign. Prerenal failure is usually due to inadequate intravascular volume or low cardiac output. Actual renal causes are usually a consequence of toxic influences such as antibiotics, other medications or the dye used at arteriography. Obstruction of the urinary collection system is a cause of postrenal failure.

Gastrointestinal System

Postoperative problems in the gastrointestinal tract associated with reconstructive surgery revolve around peptic ulceration. Sepsis, prolonged hypotension, widespread tissue trauma, respiratory insufficiency, renal failure and advanced age are all risk factors.[8,9] Usually, the body of the stomach is involved, but the small bowel, including duodenum, as well as the colon have suffered such ulcerations. In most instances, nonoperative methods for controlling the bleeding are suitable but will not be effective if the underlying cause, as for example sepsis, continues. It should be remembered that the surgical death rate for stress hemorrhage in the stomach exceeds 50%.[9] Frequent administration of antacids through a nasogastric tube if necessary, with hourly check of gastric residuals to avoid the possibility of aspiration pneumonia, plus monitoring of the gastric juice pH, are all essential in prophylaxis and treatment.

Hemorrhage

Early reoperation is mandatory in the management of bleeding from an anastomosis or an area of dissection such as the bed of an aortic aneurysm. Complications of bleeding are the development of infection in a hematoma and the late formation of a pseudoaneurysm at the anastomotic line. If more than two units of blood are needed to maintain a hematocrit in the range of 30% ± 2% during the first 6 to 12 hours, intra-abdominal hemorrhage must be considered. The usual causes of persistent peri- or postoperative bleeding are inadequate surgical hemostasis, a failure to reverse or excessive amounts of anticoagulants and, rarely, defects in clotting factors such as disseminated intravascular coagulation and dilutional thrombocytopenia. Fresh-frozen plasma (10 ml/kg body weight) should be used to counteract the effect of heparin or Coumadin. The administration of large amounts of stored, packed, red blood cells may lead to dilutional thrombocytopenia, and fresh platelets should be available and administered with every six units of platelet concentrate. Spontaneous bleeding is rare with a platelet count of above 20.0 $\times$ 10^9/L.

Massive blood transfusions, major transfusion reaction, widespread tissue trauma, extensive ischemia and sepsis are the most common causes of consumptive coagulopathy. Rarely, it is seen as an idiosyncratic reaction to systemically administered heparin.[10] Disseminated intravascular coagulation involves consumption of platelets and coagulation Factors VIII and V as well as fibrinogen. Thrombocytopenia, prolonged prothrombin time and depressed fibrinogen levels are the criteria for the development of disseminated intravascular coagulation. The use of heparin should be reserved for when other efforts such as administration of platelets in fresh-frozen plasma fail to correct the problem.

Psychosis

The patient who undergoes reconstructive aortic surgery is generally elderly, in an age group susceptible to postoperative psychosis. Agitation, confusion, hallucinations, even combative behaviour are frequently seen. These changes are mild and generally resolve with no specific treatment. Sleep deprivation, unfamiliar surroundings, disturbances in physiology, such as hypoxia, hypoglycemia and hyponatremia, and the consequences of sepsis all play a role. A few may be suffering alcohol withdrawal. Narcotics may intensify the problem. Cimetidine has been implicated. Normal waking and sleeping patterns should be re-established if possible, normal sensory input provided and sedation, if used, must be carefully monitored. Haloperidol in doses of 0.5 to 2 mg orally or intramuscularly may be useful as is Librium.[11]

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Biologic Response to Uncemented Madreporic Canine Hip Arthroplasty

The Madreporic hip arthroplasty has provided encouraging clinical results. The surface of the prosthesis is unlike other uncemented prostheses since the rough surface is formed by multiple 1-mm diameter beads that are cast as an integral part of the prosthesis and increase its surface area four times. Seven Madreporic hip arthroplasties were implanted in adult male mongrel dogs. Specimens were harvested 5, 6, 9, 12, 20 and 52 weeks after insertion. One specimen became septic and loosened. The prosthesis was stabilized initially by friction fit between the prosthesis and the endosteal cortex of the proximal femur. Dense, uniformly organized, fibrous tissue surrounded the prosthesis at 5 weeks. The fibrous tissue appeared similar to Sharpey's fibres. Circumferential bone contact increased from 26% at 5 weeks to 60% at 52 weeks. Fluorescent labelling revealed active new bone formation within the interstices of the prosthesis without evidence of an intervening soft-tissue membrane. Firm mechanical anchorage of the Madreporic femoral prosthesis was demonstrated in this study.

L'arthroplastie Madréporique de la hanche a fourni des résultats cliniques encourageants. La surface de la prothèse diffère des autres prothèses non consolidés par le fait que la surface rude est formée de multiples grains de 1 mm de diamètre constituant une partie intégrante de la prothèse et augmentant de quatre fois sa superficie. Sept arthroplasties Madréporique de la hanche furent implantées chez des chiens mongrel adultes mâles. Des échantillons furent récoltés 5, 6, 9, 12, 20 et 52 semaines après insertion. Un échantillon devint septique et se décomposa. La prothèse fut stabilisée initialement par ajustement entre la prothèse et le cortex endostéal du fémur proximal. Du tissu fibreux, dense, uniformément organisé, entoura la prothèse au bout de 5 semaines. Ce tissu fibreux ressemblait aux fibres de Sharpey. Le contact osseux périphérique augmenta de 26% après 5 semaines, à 60% après 52 semaines. Le marquage fluorescent révéla une nouvelle formation osseuse active dans les interstices de la prothèse sans évidence d'apparition d'une membrane de tissu mou. Un assemblage mécanique ferme de la prothèse fémorale Madréporique a été démontré au cours de cette étude.

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Accepted for publication Dec. 1, 1986

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The long-term success of an arthroplasty depends on permanent anchorage of the prosthetic components within bone. Smooth-surfaced prostheses require bone cement for fixation. In spite of recent modifications in the use of methylmethacrylate to improve mechanical longevity,[1,2] cemented arthroplasties frequently loosen with time.[3,4] Uncemented prostheses have been developed as an alternative. Rough-surfaced components may allow biologic fixation that can respond to mechanical stresses over a long period without loosening.

There has been extensive clinical experience with the Madreporic (Benoist Girard, Paris; French manufacturing division of Howmedica Inc.) hip arthroplasty developed by Lord.[5-7] This prosthesis utilizes a rough surface of 1-mm diameter beads that are cast as an integral part of the femoral component. The Madreporic surface increases the surface area of the prosthesis by a factor of four. The beads are cast in a single layer with an interbead space of between 1 and 3 mm. This differs from other uncemented prostheses which generally have the rough surface secondarily attached to the smooth prosthetic core by sintering or a similar process.[8] This surface takes the form of wire mesh, atomized alloy metal or a composite structure. The interbead distance of the Madreporic prosthesis far exceeds the 50 to 400 μm pore size recommended for porous coated prostheses.[9] For these reasons, the Madreporic surface cannot be compared to porous coated prostheses which have received a great deal of experimental investigation.[10-15]

Prosthetic canine hip arthroplasties

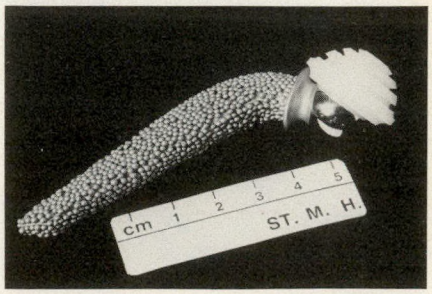

FIG. 1—Canine prosthetic hip arthroplasty components consisting of polyethylene acetabular component and femoral component with Madreporic surface.

were used to study the biologic response to the Madreporic surface. The prosthesis had an identical surface to that of the components used in human Madreporic hip arthroplasty. The purpose of the study was to determine the response of the proximal femur to the implantation of the prosthesis. Specifically, we sought to investigate the character of the interface between the prosthesis and the surrounding bone, and the presence of new bone formation within the proximal femur after insertion of the prosthesis.

Materials and Methods

Seven Madreporic hip arthroplasties were performed in adult mongrel dogs. The canine femoral component was triangular in cross-section with the base medially and the apex laterally (Fig. 1). This differs from the circular cross-section of the human prosthesis. This design difference gave immediate rotational stability without circumferential cortical contact.

The hip was exposed through an anterolateral incision. After capsulectomy, the joint was dislocated and the femoral head resected. The acetabulum was reamed and irrigated, and an acetabular prosthesis cemented in place using methylmethacrylate. The femur was reamed and the femoral component inserted, producing a snug fit to give initial mechanical stability (Fig. 2). Antibiotics were administered prophylactically for 48 hours postoperatively. The animals were not immobilized.

The dogs were given a single intravenous dose of xylenol orange,[16] 90 mg/kg, 1 week before sacrifice by Nembutal overdose. Both the acetabulum and the proximal femur were harvested, but only the femoral components were studied. Specimens were obtained 5, 6, 9, 12, 20 and 52 (two specimens) weeks following insertion.

The proximal femur was dehydrated, defatted and embedded in methylmethacrylate using a method similar to that of Jowsey and associates.[17] Modifications in the technique were necessary due to the large size of the specimens.[18] Cross-sections 1-mm thick were cut through the proximal, middle and distal portions of the prosthesis using a Buehler low-speed low-deformation saw. Fine-detail roentgenograms were obtained using Kodak XTL-2 mammography film and a Faxitron 805 (Hewlett Packard, Mississauga, Ont.). The sections were mounted on petrographic slides and ground with a rotary grinder to a thickness of 250 μm. The sections were analysed by light, polarized and fluorescence microscopy.

The extent of bone ingrowth was measured for the proximal, middle and distal cross-section of each prosthesis. A 20 × 25-cm photographic enlargement was made of a whole-mount polarized photomicrograph of each entire cross-section (original magnification × 4). An area analyser was then used to determine the percentage of the prosthetic perimeter that lay in direct contact with bone. Since the prosthesis has a rough surface rather than a porous coating, it was possible to make these measurements accurately.

Results

In general, the animals were ambulatory within 48 hours of surgery. One animal stopped walking on the operated leg 6 weeks postoperatively. Radiologic examination did not reveal any abnormality, but the specimen was loose when harvested 52 weeks after insertion and purulent material was present within the pseudocapsule of the hip joint. No other specimens could be manually extracted at the time of sacrifice. An undisplaced fracture of the proximal femur occurred intraoperatively in two dogs whose speci-

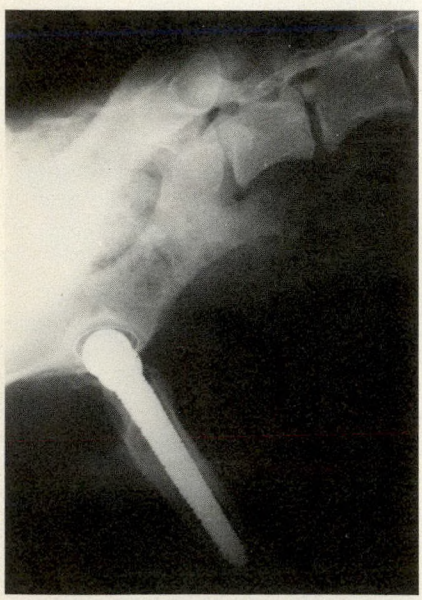

FIG. 2—Postoperative view of canine Madreporic hip arthroplasty. Acetabular component is cemented in place. Femoral component is inserted without cement and is initially stabilized by friction fit.

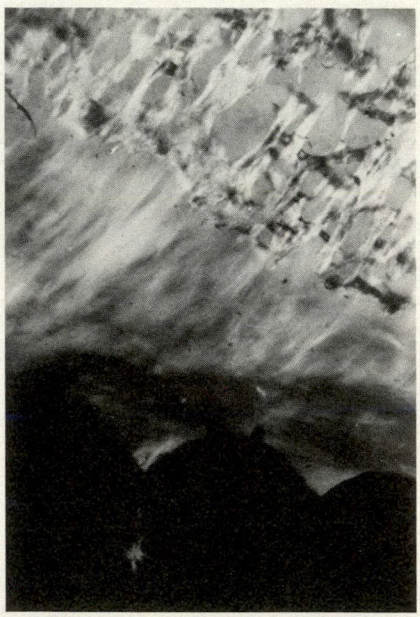

Fig. 3a

Fig. 3b

FIG. 3—(a) Polarized photomicrograph of proximal cross-section 5 weeks after insertion of prothesis. In this area there is no direct bone contact between prosthesis and endosteal cortex. Intervening space is filled with fibrous tissue. Immature woven bone is advancing toward prosthesis. Intramembranous ossification is occurring within fibrous tissue (original magnification × 20). (b) Polarized photomicrograph showing junction between woven bone and fibrous tissue surrounding prosthesis. Fibrous tissue is well organized and resembles Sharpey's fibres, suggesting that it may be important for load transference while bone ingrowth occurs (original magnification × 52).

Table I—Prosthetic Perimeter With Bone Contact (%)

Weeks	Proximal stem	Middle stem	Distal stem	Mean
5	18	20	40	26
6	48	27	54	43
9	13	78	26	39
12	43	29	41	38
20	58	48	46	50
52	64	57	61	60
52 (septic)	0	0	0	

mens were harvested 6 and 9 weeks after implantation; there was no difference in the recovery rate of these animals postoperatively.

Evaluation of the 5-week specimen demonstrated immature woven trabecular bone being formed along the endosteal cortical surface (Fig. 3a). Only a small portion of the prosthesis had direct endosteal bone contact (Table I). The trabeculae ranged from 50 to 200 μm in thickness (average 150 μm). The trabeculae were oriented at right angles to the prosthetic stem. Dense, fibrous tissue, oriented obliquely to the cross-section of the femoral component, filled the space between the prosthesis and the endosteal cortical bone. The fibrous tissue was well organized with parallel fibres that resembled Sharpey's fibres (Fig. 3b). A longitudinal section demonstrated that the fibrous-tissue fibres sloped downward from the endosteal bone to the prosthesis.

At 6 and 9 weeks the prosthetic

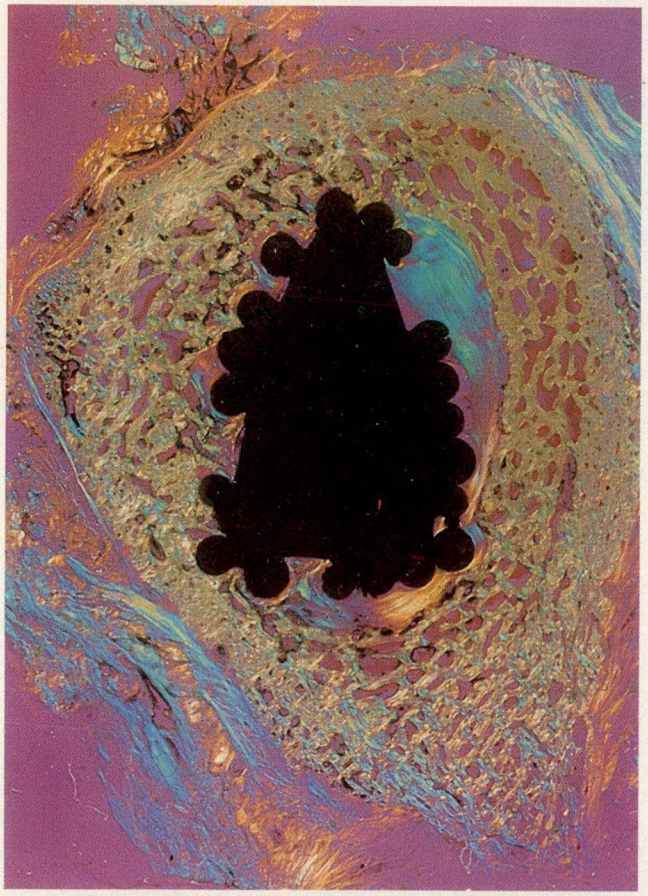

Fig. 4a

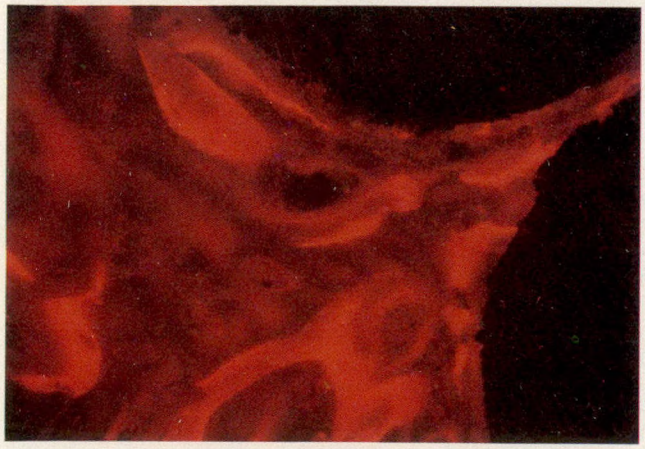

Fig. 4b

FIG. 4—(a) Polarized photomicrograph of cross-section of midstem of prosthesis at 20 weeks. Fifty percent of prosthetic perimeter has direct bone contact. Bone growth into interstices of prosthesis and trabecular hypertrophy are both advanced at apices of prosthesis (original magnification × 4). (b) High-power fluorescent photomicrograph of bone–prosthesis interface. New bone is forming within interstices of prosthesis and immediately adjacent to prosthesis without any evidence of intervening soft tissue (original magnification × 52).

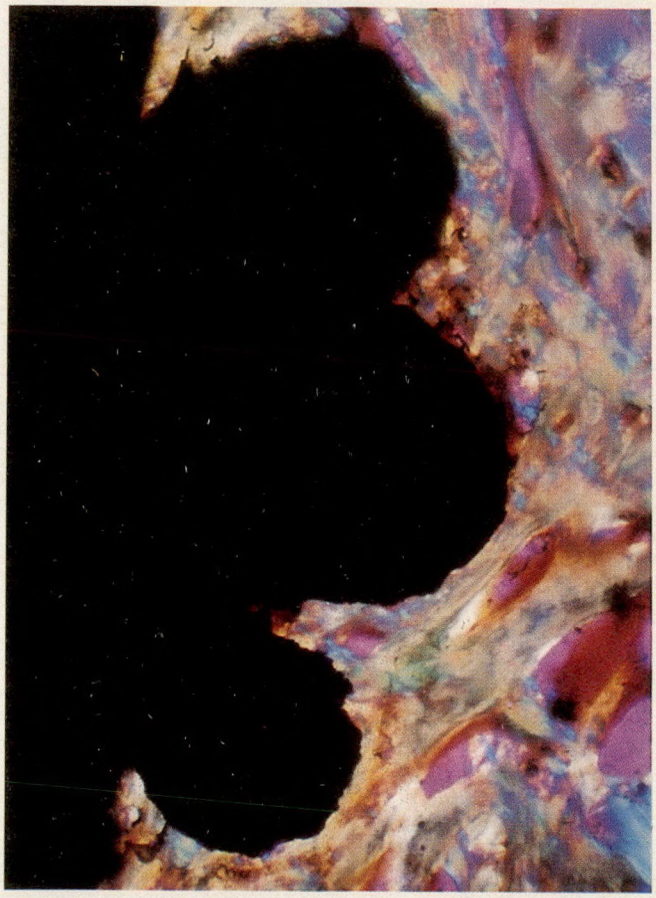

Fig. 5a

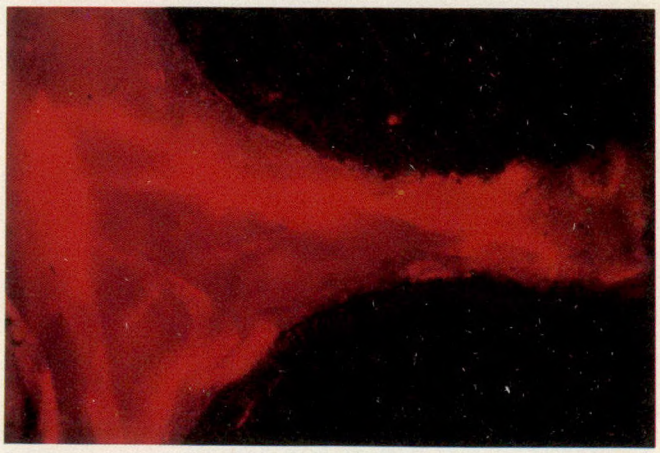

Fig. 5b

FIG. 5—(a) Polarized photomicrograph of cross-section of middle portion of specimen at 1 year. Mature trabecular bone lies in intimate contact with prosthesis (original magnification × 20). (b) High-power fluorescent photomicrograph of bone–prosthesis interface 1 year after implantation. Active new bone formation continues deep within interstices of prosthesis without any evidence of intervening soft tissue (original magnification × 52).

perimeter with direct bone contact had increased to 43% and 39% respectively. Predominantly woven bone was observed around the prosthesis. The intervening fibrous tissue remained well organized. The bone formation was concentrated at the proximal and distal ends of the prosthesis and also in cross-section at the apices of the triangular prosthesis. The split in the femur had healed in both specimens. At 12 weeks, 37% of the prosthetic perimeter had direct bone contact. The lateral apex of the prosthesis demonstrated the greatest bone ingrowth and hypertrophy.

Fifty percent of the prosthetic perimeter had direct bone contact at 20 weeks (Fig. 4a). Bone ingrowth and trabecular hypertrophy were maximal at the apices of the prosthesis. New bone formation was observed in many of the interstices of the prosthesis (Fig. 4b). In these areas, the new bone formation approximated the prosthesis without evidence of an intervening soft-tissue membrane.

At 52 weeks, 60% of the prosthetic perimeter had direct bone contact. Mature trabecular bone was in contact with the prosthesis in many areas without intervening soft tissue (Fig. 5a). Fluorescent labelling demonstrated continued new bone formation deep within the interstices of the prosthesis without intervening soft tissue (Fig. 5b).

The septic specimen demonstrated no fibrous or bone ingrowth (Table I). The prosthesis was encircled by a reactive 1-mm thick shell of endosteal new bone formation located approximately 2 mm from the edge of the prosthesis. The cortical bone of the proximal femur had undergone cancellous change.

Discussion

Our study demonstrates that the Madreporic surface provided firm fixation of the femoral component of the prosthesis both initially and throughout the 1 year of the study. Immediately postoperatively, the prosthesis was anchored by friction fit. With time, well-organized fibrous tissue and new bone surrounded the prosthesis to stabilize it. Bone formation around the prosthesis was progressive, and in many areas no soft tissue intervened between areas of new bone and the prosthesis. Others[11,19,20] have noted that initial friction fit is of paramount importance in allowing bone ingrowth. Micromotion is acceptable, but macromotion is not. This concerned us in our study since two undisplaced femoral fractures occurred at the time the prosthesis was inserted. The fractures healed without displacement, and bone ingrowth was not adversely affected. Longitudinal splitting of the femur at the time of prosthetic insertion did not appear to have a deleterious effect on the quality or quantity of bone ingrowth, providing the initial friction fit was maintained.

Since the prosthesis was triangular and the canine endosteal canal is circular, only a portion of the prosthesis had direct contact with the endosteal cortex at the time of insertion. In areas where the prosthesis was in direct contact with the endosteal cortex, new bone formation was observed, but fibrous tissue, fibrocartilage and hyaline cartilage were absent. Bone ingrowth was maximal at the apices of the prosthesis, which were often in direct contact with the endosteal cortex. Progressive trabecular maturation and hypertrophy were observed in the 20- and 52-week specimens.

Fibrous tissue, which was obliquely oriented to the longitudinal surface of the prosthesis, formed in areas where the prosthesis did not touch the endosteal cortex. This fibrous tissue attached to the endosteal bone and had the appearance of Sharpey's fibres. This tissue, also similar in appearance to periondontal ligament, has been observed by others[9,11,14,15,20,21] and was particularly prominent in the 5-, 6- and 9-week specimens. We speculate that this well-organized fibrous tissue plays a role in load transference initially, while bone ingrowth occurs. Its presence did not indicate loosening, since new bone formation was occurring within the interstices of the prosthesis in areas immediately adjacent to those with fibrous tissue.

Cameron and colleagues[22] demonstrated the inhibitory influence of early sepsis on bone ingrowth. Chen and colleagues[13] observed a lack of bone growth into a cementless canine hip arthroplasty when non-weight bearing occurred secondary to a dislocation. We observed a total absence of tissue ingrowth of any kind in the single animal with sepsis. We are uncertain whether the absence of tissue ingrowth was due to a direct effect of sepsis or was secondary to the dog's inability to bear weight. We think it was probably secondary to sepsis, because of the total absence of any type of tissue ingrowth.

The Madreporic prosthesis is not subject to damage by the shear forces that occur during insertion and that may disrupt porous sintered surfaces.[23] Sintered-particle migration does not occur and the large surface irregularities allow for the development of new bone between the endosteal bone and the prosthesis even in areas where there is no bone contact initially. Active new bone formation was observed at the bone–prosthesis junction throughout the study. Although only a small number of animals were studied and the important issue of stress shielding in the proximal femur was not addressed, this canine study demonstrates a favourable response to implantation of a femoral hip arthroplasty component with a Madreporic surface.

We acknowledge the assistance of Mrs. Isabella Urquhart, chief technologist in histology, Princess Margaret Hospital, Toronto and Mr. A.K. Collins, director of medical photography, St. Michael's Hospital, Toronto.

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Posterior Wall Disruption of the Left Ventricle After Mitral Valve Replacement: Management of Bleeding and Cardiac Enlargement

Two patients who underwent mitral valve replacement sustained posterior left ventricular disruption postoperatively and survived. Management consisted of repair from outside the heart without removal of the mitral prosthesis. This was achieved with a running Prolene suture in the atrioventricular junction in one patient and pledgeted sutures in the other who had transverse midventricular disruption. Biologic glue was crucial in controlling hemorrhage after the repair in both cases. The patient with transverse midventricular disruption required placement bilaterally of pectoralis muscle flaps to close the sternal wound because of swelling and dilatation of the heart.

Deux patients qui avaient eu un remplacement de la valvule mitrale ont subi en postopératoire une désunion ventriculaire gauche postérieure et ont survécu. Le traitement a consisté à réparer de l'extérieur du coeur sans enlever la prothèse mitrale. Ceci a été accompli chez une patiente à l'aide d'une suture courante de Prolène et, chez l'autre, qui avait une désunion méso-ventriculaire transverse, à l'aide de sutures en tampon. Dans les deux cas, l'utilisation d'une colle biologique fut cruciale pour juguler l'hémorragie après l'opération. La patiente qui avait subi une désunion méso-ventriculaire transverse a nécessité des greffes bilatérales de lambeaux de muscles pectoraux pour refermer la plaie sternale à cause d'une inflammation et d'une dilatation du myocarde.

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Supported by a grant from the Heart and Stroke Foundation of Ontario

Accepted for publication Jan. 29, 1987

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Ventricular disruption is a dreaded complication of mitral valve replacement and is associated with a high death rate.[1-4] Most deaths are due to bleeding[5-7] and heart failure.[5,8] We have encountered two such cases and achieved successful surgical repair without removing the prosthesis. Besides suturing the disruption from outside the heart, biologic glue was found to be crucial in controlling residual hemorrhage. Bilateral pectoralis muscle flaps[9] and bolstered skin traction of the sternal edges were used in one patient to accommodate the dilated heart in the pericardial cavity.

Case Reports

Case 1

A thin 75-year-old woman underwent mitral valve replacement with a 27-mm Ionescu–Shiley prosthesis for calcific mitral stenosis and insufficiency. Cardiac catheterization before surgery revealed normal coronary arteries, severe mitral stenosis with a gradient of 17 mm Hg across the mitral valve at rest, grade 3 mitral insufficiency and calcific mitral annulus with calcification extending posteriorly into the left ventricular myocardium. The annulus was decalcified but the posterior papillary muscles were calcified and were left adherent to the ventricular myocardium. Pledgeted sutures were used to secure the mitral prosthesis. A left ventricular vent was not used. The operation was uncomplicated. Three hours after the procedure she suffered a severe hemorrhage requiring immediate exploration. A site of profuse bleeding was identified at the back of the heart. Under cardiopulmonary bypass the disruption was pinpointed at the atrioventricular junction (type I disruption[10]). In view of the heavy calcification noted at the time of the initial operation, removal of the mitral prosthesis and repair from within the ventricle were considered unwise. After induction of cardioplegic arrest, a running 4-0 Prolene suture was applied to the atrioventricular region avoiding the circumflex coronary artery. Biologic glue, made up of thrombin, cryoprecipitate, plasma and calcium,[11] was applied to the area after suturing. The patient was weaned from bypass without difficulty. She made a smooth recovery and was well on follow-up 15 months later.

Case 2

An obese 65-year-old woman underwent replacement of a 29-mm Omniscience mitral valve prosthesis which had been implanted 8 months before for mitral stenosis and insufficiency. Malfunction of the prosthesis was evident; at catheterization the coronary arteries appeared normal. After opening the sternum, only the right side of the heart was dissected due to very dense adhesions over the left ventricle. There was tissue ingrowth into the prosthesis with pannus formation in the atrial and ventricular sides of the prosthetic valve. The valve was removed and replaced with a 29-mm Ionescu–Shiley prosthesis using pledgeted sutures. A left ventricular vent was not used. The operation was difficult but weaning from bypass posed no problems. Two hours after surgery, she had severe hemorrhage with shock and was immediately explored. Profuse bleeding was observed from behind the heart. By the time bypass was instituted she had been hypotensive for approximately 30 minutes. After induction of cardioplegic arrest and dissection of the remaining pericardial adhesions, a 3- to 4-cm longitudinal disruption was found on the posterior midventricular region of the left ventricle (type III[10]). Finger exploration of the ventricular cavity through the tear revealed that the struts of the prosthesis did not appear to be the cause of the tear, so we elected to repair the tear without removing the prosthesis. The defect was closed with pledgeted sutures of 3-0 Prolene placed longitudinally. Bleeding, which occurred after unclamping and heart ejection, ceased when biologic glue was applied to the posterior surface of the heart. She was weaned from bypass with the aid of the intra-aortic balloon pump. Due to profound hypotension when attempts were made to close the sternum or skin, both were left open. Temporary closure of the latter was achieved with a Vidrape. The morning after surgery she was returned to the operating room and several attempts were made to close the sternum and skin, again without success due to profound hypotension. Three days later

another attempt to close the sternum failed, but the skin was approximated. A final attempt at sternal closure was made 4 days later without success, so pectoralis muscle flaps were developed to fill the sternal wound. Stay sutures were needed to hold the sternal edges apart to prevent a tamponade-like effect while the muscle flaps were applied to the sternotomy incision. They were removed 7 days postoperatively. The intra-aortic balloon was removed a day later. She required prolonged ventilatory support and eventually a tracheostomy was performed 32 days after operation. The wounds healed without problems, but bacteremia occurred due to infection in a subclavian line used for hyperalimentation. She made a satisfactory recovery, with excellent cosmetic appearance of the sternotomy incision. The follow-up is short (9 months), but she remains well and has had the tracheostomy tube removed.

Discussion

Left ventricular disruption after mitral valve replacement is rare (0.5% to 2% of all mitral valve replacements) and carries a high death rate.[2–4] The condition was first reported by Roberts and Morrow in 1967.[12] Its cause is not completely understood.[10,12–15] Although spontaneous disruption of the left ventricle without antecedent trauma may occur,[15] most reports implicate a preceding traumatic event.[16–23] Several hypotheses have been advanced to explain this complication. The atrioventricular groove is a "weak" area in that the mitral annulus is only in continuity with the endocardium of the left atrium[4] without muscular continuity between atrium and ventricle. Thus, aggressive débridement or decalcification of this region may lead to atrioventricular separation. Similarly, insertion of sutures that penetrate the ventricular muscle and are tied vigorously may result in tearing of the ventricular muscle with resultant weakening and subsequent separation. This may explain type I injury. It has been suggested that type II injury is due to zealous excision of the papillary muscles with thinning of the ventricular wall followed by trauma from an adjacent prosthetic valve strut.[8,10] Cobbs and colleagues[15] and Spencer and associates[8] have hypothesized that type III injury is related to disruption of the posterior chordal attachments, predisposing the ventricle to a stretch injury, exacerbated by excessive ventricular relaxation during cardioplegic arrest. They suggest that preservation of the posterior mitral valve apparatus may eliminate this complication. In fact, the patient in our case 2 with a type III separation, did have excision of the posterior leaflets and chordae during the original operation, thus predisposing the ventricle to this type of separation. It must be noted, however, that confirmation of the mechanism of injury usually eludes the surgeon and, in view of this, particular care should be taken in the patient with a small ventricle, in the elderly and in the patient with previous mitral valve replacement who has had excision of the mural leaflet. Treatment includes removal of the prosthesis and suture repair with or without a patch[5] or repair of the tear without removal of the prosthesis.[8] Bleeding[5–8] and heart failure[5,8] appear to be the most common complications leading to death after repair.

In our patients, repair was achieved from outside the heart either by running sutures in the atrioventricular groove avoiding the circumflex coronary artery (case 1) or with pledgeted sutures on the ventricular myocardium (case 2). In both cases, profuse bleeding in inaccessible areas occurred after repair. Biologic glue allowed complete hemostasis after the repair and was life-saving. This type of physiologic glue[11] is readily available in any operating room.

Bleeding, edema and dilatation of the heart in the patient with type III disruption posed a considerable problem. Although bleeding was eventually controlled with biologic glue, cardiac swelling and dilatation precluded approximation of the sternum and skin. This problem was solved by the use of bilateral pectoralis muscle flaps to accommodate the heart in the pericardial cavity.[9] The sternal edges had to be held apart with bolster sutures to prevent cardiac compression when the flaps were laid over the sternal defect.

Conclusions

Bleeding and cardiac edema are major

problems when posterior left ventricular disruption occurs after mitral valve replacement. The surgeon should consider repair without removal of the prosthesis and the use of biologic glue to achieve hemostasis subsequently. Mobilization of muscle flaps to accommodate an edematous dilated heart should also be considered if sternal closure is impossible. It may become necessary to hold the sternal edges apart with stents or suture bolsters to avoid compressing the heart until swelling decreases with time.

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continued on page 285

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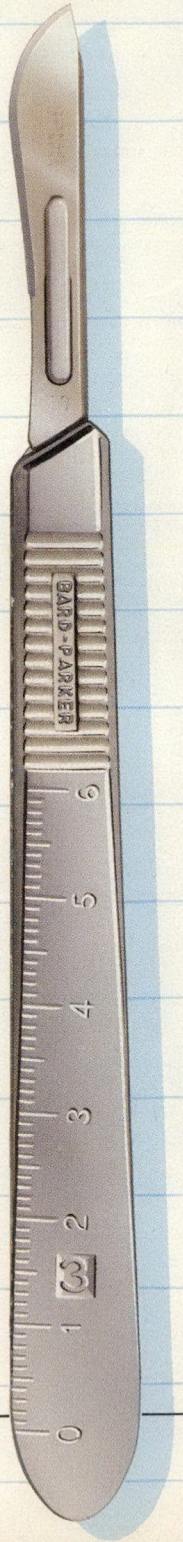

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Conservative Management of Renal Lacerations in Blunt Trauma

Controversy in the treatment of blunt renal trauma is largely focused on immediate surgery versus conservative management for parenchymal lacerations. A retrospective analysis of 133 cases of blunt renal trauma at the Victoria General Hospital in Halifax over a 10-year period revealed 26 cases of renal laceration. The conservative approach to radiologic diagnosis and treatment options is discussed. Our experience confirms a low rate of both nephrectomy and secondary complications using conservative management.

Le débat entourant le traitement des traumatismes rénaux fermés oppose surtout les tenants d'une chirurgie immédiate à ceux qui préconisent un traitement conservateur des lacérations du parenchyme. L'analyse rétrospective de 133 cas de contusions rénales vus au Victoria General Hospital d'Halifax au cours d'une période de 10 ans a révélé 26 cas de lacérations rénales. On commente l'approche conservatrice avec le diagnostic radiologique et les diverses options thérapeutiques. Selon notre expérience, on constate que le traitement conservateur est accompagné d'un faible taux de néphrectomie et de complications secondaires.

Despite increased experience over the last 30 years with the treatment of renal lacerations from blunt trauma, the issue of immediate surgery versus conservative management remains controversial. Advocates of immediate surgical débridement maintain that primary repair and drainage allow for primary healing and early rehabilitation of the patient.[1-4] Advocates of a conservative approach think that a high percentage of renal injuries will heal spontaneously, precluding the need for any renal surgery.[5-10]

At our institution, clinically stable patients with severe renal lacerations have been managed conservatively and a review of our experience appeared germane to the controversy of immediate versus expectant surgery.

Patients and Methods

A retrospective analysis at the Victoria General Hospital in Halifax of 133 patients who had suffered blunt renal trauma between 1975 and 1984 revealed 26 cases of severe renal lacerations. Routine intravenous pyleography was done on admission for all patients with suspected renal injury, except those requiring emergency surgery. Renal arteriography was performed when no renal lesion or a poorly defined one was seen on intravenous pyelography or, occasionally, as part of aortography to delineate intra-abdominal bleeding. Nuclear scanning and intravenous pyelography were used on follow-up to assess renal function.

Renal lesions were classified in two groups — parenchymatous and hilar. Under parenchymatous lesions, contusion was diagnosed when there was minimal functional or anatomical change on intravenous pyelography; laceration was defined as a deep parenchymal tear extending at least to the corticomedullary junction, with or without calyceal disruption (Fig. 1); a shattered kidney implied multiple lacerations. Hilar injuries were subdivided into pedicle injury, referring to renal arterial occlusion or laceration resulting in nonvisualization and failure of kidney perfusion, and pelvic injury, referring to a disrupted renal pelvis.

The clinical state of the patient decided the initial management of a renal laceration. If the patient was hemodynamically unstable because of severe renal hemorrhage at the time of admission, despite resuscitation, laparotomy was performed immediately. The clinically stable patient was treated conservatively with bed rest, frequent abdominal examinations and monitoring of vital signs, hydration, analgesics, serial complete blood count and follow-up renal scanning or intravenous pyelography. Renal exploration was done if there was persistent severe hemorrhage, massive or increasing urinary extravasation or renal sepsis.

Findings

Renal lacerations accounted for 18% of blunt renal injuries in our institution and 80% of them resulted from traffic accidents (Table I). In this adult population, 75% of the patients were under 30 years of age and 90% were men (Fig. 2). Emergency intravenous pyleography was performed on 25 patients; emergency

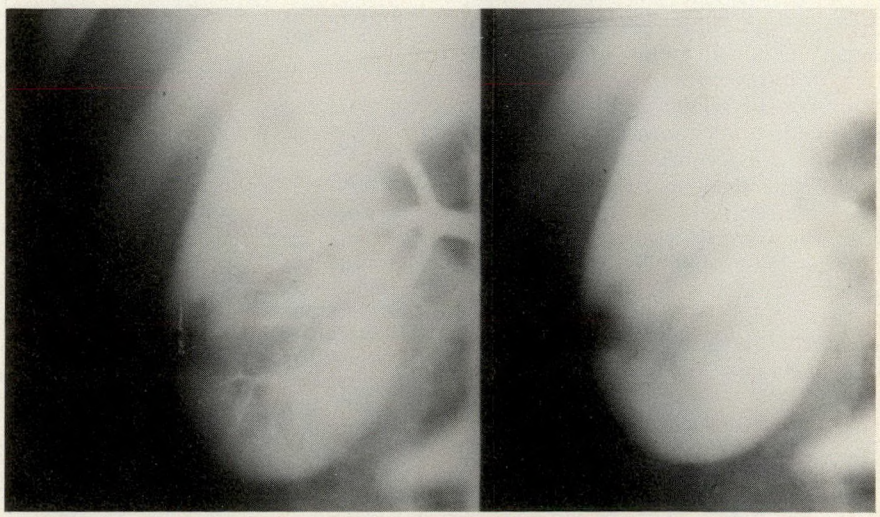

FIG. 1—Intravenous pyelogram and corresponding arteriogram of renal laceration.

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Presented at the annual meeting of the Canadian Urological Association, Montreal, PQ, June 20, 1985

Accepted for publication Jan. 16, 1987

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laparotomy precluded one patient from undergoing intravenous pyelography and the laceration was diagnosed at surgery. Fifteen patients had renal arteriography for more accurate definition of the renal laceration. There were three additional cases in which arteriography, used to define an associated abdominal injury, outlined the renal laceration. The intravenous pyelogram and arteriogram were congruous except in three patients in whom the intravenous pyelogram was suggestive of a contusion but the arteriogram showed a renal laceration.

Three patients required immediate renal exploration and nephrectomy for severe hemorrhage. Surgery (nephrectomy) was indicated in four other patients initially treated conservatively, three for persistent severe bleeding and one for renal sepsis. All kidneys removed immediately or after expectant treatment showed shattering.

Two (8% of the group of 26 with lacerations) patients requiring nephrectomy had associated renal anomalies. One had a pelvic kidney and required immediate nephrectomy for severe hemorrhage. The other had a hydronephrosis secondary to an obstruction at the ureteropelvic junction; the obstruction initially was managed expectantly, but persistent severe bleeding necessitated nephrectomy.

There were no postoperative complications in the three patients requiring immediate nephrectomy. One patient with a delayed nephrectomy required reexploration of the renal bed 24 hours after operation because of hemorrhage, seemingly from a slipped ligature.

In addition to the renal laceration, major associated injuries (Table II) were present in 17 patients, an average of 1.8 injuries per patient. There were three deaths but none due to renal causes. Excluding the three patients who died of non-renal causes and three patients who required immediate nephrectomies, 20 patients were managed expectantly. Four (20%) later needed nephrectomy. Those managed successfully showed improved renal function on follow-up renal scanning or intravenous pyelography by the time of discharge. For patients with major injuries associated with the renal laceration, the average time in hospital was 20 days. When the renal laceration was the main injury responsible for hospitalization, the average stay was 10 days. Six patients underwent intravenous pyelography at 6 months and one at 3 years; all showed completely healed kidneys. None suffered hypertension during a follow-up of 2 to 6 years. The remaining 13 could not be traced because of the transient patient population.

Discussion

In blunt renal trauma, renal contusion accounts for 80% of the injuries; the contusions are treated conservatively as healing usually occurs without complications.[11] Immediate surgical intervention is advised for hilar pedicle and pelvic injuries and renal laceration in patients who are hemodynamically unstable.[12] The controversy in the management of blunt renal trauma is thus focused on the less than 20% of patients with renal trauma who present with parenchymal lacerations.[1-10,13]

In accurately delineating the renal injury, infusion intravenous pyelography with tomography has an excellent (90%) correlation with both selective renal arteriography and the lesion seen at exploration.[14-16] Unfortunately, tomography is not available to every emergency department, and intravenous pyelography alone is said to have a correlation of 50% to 70%.[14,15,17] In this series, the intravenous pyelogram provided adequate information about the renal laceration in 22 of 25 cases for a false-negative result of 12%. On the other hand, arteriography confirmed the diagnosis of renal laceration whenever it was suspected on intravenous pyelography.

Renal arteriography is the most accurate study for defining the nature of

Table I—Cause of Renal Injuries

Cause	Type of injury: Contusions	Lacerations	Pedicle	Hilar	Totals, no. (%)
Automobile accidents	52	16	1	1	70 (53)
Motorbike accidents	18	5	2	0	25 (19)
Falls	14	3	1	0	18 (14)
Sports	10	1	0	0	11 (8)
Physical assault (blows)	8	1	0	0	9 (7)
Totals	102	26	4	1	133

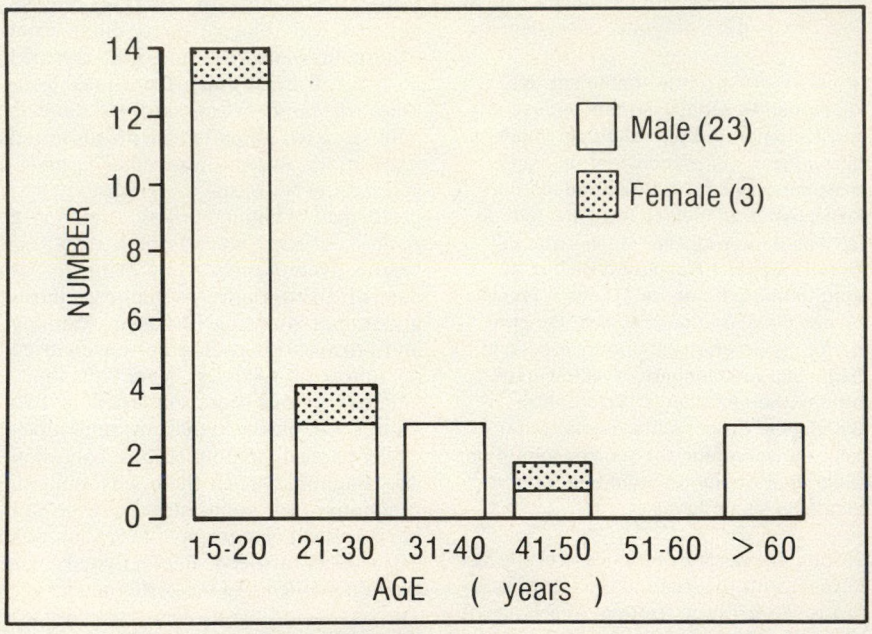

FIG. 2—Age and sex distribution of patients with renal lacerations.

Table II—Associated Major Nonrenal Injuries

Associated injury	Blunt injury: Contusion (n = 102)	Laceration (n = 26)	Pedicle (n = 4)	Hilar (n = 1)	Totals
Fractured ribs	23	3	3	1	30
Lacerated spleen	10	4	3	0	17
Lacerated liver	9	2	3	0	14
Lacerated bowel	3	2	3	1	9
Other lacerations (vessels, pancreas)	6	3	0	0	9
Severe head injury	13	2	0	0	15
Fractured skull	3	1	0	0	4
Fractured spine	14	5	0	0	19
Fractured pelvis	9	2	0	0	11
Fractured extremity	9	3	3	1	16
Severe lung injury	10	3	2	1	16
Totals	109	30	17	4	160

the renal injury. Lang[18] recommended its use in all patients with hematuria and renal trauma, citing its low morbidity, accuracy and assessment of associated intra-abdominal injury. However, it can be argued that if a conservative approach to managing renal laceration is the policy and the indication to explore is based on clinical criteria, establishing a functioning kidney on the intravenous pyelogram is all that is necessary, since knowing the extent of laceration will not influence the course of management. Arteriography should, therefore, be reserved for possible surgical intervention when there is clinical deterioration. Advocates of a more liberal use of renal arteriography could argue that the procedure may be unsafe or not feasible by the time clinical deterioration has occurred. It should be emphasized that the arteriographically defined renal injury may be more extensive than the patient's clinical condition indicates and the severity of the anatomical injury per se should not be an indication for surgery.

Recent reports[19,20] suggest that computerized tomography provides precise anatomical details of renal injuries and has been used in preference to renal arteriography. Computerized tomography has been advocated as the primary diagnostic modality in patients suspected of sustaining major renal and other organ injuries.[21] Although there is a possibility that a laceration may be missed because the slice taken by the scanner may not go through the site of the renal injury, enhancement of the renal parenchyma has the added advantage of ruling out a major vascular injury. Routine intravenous pyelography and selective use of arteriography still have their merits, particularly if renal surgery is contemplated.

In reviewing various series (Table III[2-4,9,10,13,14,22]) in which the policy of immediate surgical repair for all renal lacerations is applied, even with the technique of controlling the vascular pedicle before renal exploration, the nephrectomy rate is equivalent to that of conservative management. The merits of either approach thus seem to centre on immediate and long-term patient morbidity. Our experience and that of others indicate spontaneous healing and resolution without side effects in a large proportion of cases even when the laceration seems severe on arteriography. Immediate surgical management has been recommended for shattered kidneys with multiple lacerations in order to avoid morbidity, but invariably a nephrectomy is necessary.[4,23] Even in these most severe parenchymal injuries, a conservative approach has been used successfully with minor complications[7] and is recommended for the patient who can be stabilized clinically.

Classification of renal injuries has varied from a simple categorization into mild and severe to one of 15 subdivisions based on anatomic and topographic criteria.[11] We prefer the classification that separates the parenchymatous from the hilar lesions and attempts to categorize the extent of the parenchymatous lesion. In our opinion such classification is practical and has therapeutic implications.

There is a good correlation between the extent of trauma and the severity of the associated injuries. These extrarenal injuries frequently dictate the immediate management and account for the morbidity and mortality. The incidence of associated injuries in this study is comparable to those of previously reported series.[4,13,24] Mendez[12] reported that shock was present in 14% to 30% of trauma patients with renal injuries compared with 20% in our series. The frequency of associated major injuries could make the conservative approach more desirable.

Advocates of immediate surgical intervention have suggested that the expectant approach prolongs the hospital stay.[1,13] The length of stay in our series was found to be more a reflection of the associated injuries. For patients whose renal laceration was the main injury, we believe an average length of stay of 10 days is an acceptable figure when compared with that from surgical series.

Table III—Frequency of Nephrectomy in Series of Renal Lacerations Caused by Blunt Trauma

Series	No. of patients	%
Immediate surgery		
Cass and Ireland, 1973[3]	22	68
Peters and Bright, 1977[13]	?	18
Cass, 1979[2]	17	15
Cass and Luxenberg, 1983[4]		
lacerations	31	6.5
shattered	14	100
Expectant treatment		
Morrow and Mendez, 1970[14]	21	22
Cass and Ireland, 1973[3]	16	6
Thompson and associates, 1977[9]	44	2
Wein and associates, 1977[10]	42	2
Gibson and associates, 1982[22]	28	19.6
Present series	20	20

Conclusions

Emergency intravenous pyelography with renal arteriography in selected cases continues to be the method of choice in the initial evaluation of patients with renal injury. Conservative management of renal lacerations carries a low morbidity, an acceptable nephrectomy rate and we believe may obviate the need for surgery in many such cases. Major associated injuries frequently play an important role in the management of patients with renal lacerations.

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Simultaneous Occurrence of Chylothorax and Subarachnoid Pleural Fistula After Thoracotomy

A left chylothorax complicated elective left posterolateral thoracotomy and emergency decompressive laminectomy carried out on a 32-year-old woman with a benign neurofibroma. Failure of conservative therapy necessitated ligation of the thoracic duct and this in turn unmasked the concomitant subarachnoid–pleural fistula. Surgical repair and lumbar subarachnoid drainage were required to close the second fistula. The clinical setting, diagnosis and management of this uncommon condition are reviewed.

Un chylothorax gauche est venu compliquer une thoracotomie postérolatérale gauche non urgente et une laminectomie de décompression d'urgence pratiquées chez une femme de 32 ans souffrant d'un neurofibrome bénin. L'échec d'un traitement conservateur a nécessité une ligature du canal thoracique qui, à son tour, mit en évidence une fistule pleuro-sous-arachnoïdien. La réparation chirurgicale et le drainage sous-arachnoïdien lombaire ont dû être pratiqués pour refermer la seconde fistule. Le tableau clinique, le diagnostic et le traitement de cette rare affection sont passés en revue.

Chylothorax and subarachnoid–pleural fistula are uncommon complications of thoracotomy. Their simultaneous occurrence, a rare event,[1] must be considered in certain settings when treatment of a chylothorax is unsuccessful.

Case Report

A previously healthy 32-year-old woman was found to have an asymptomatic left posterior mediastinal mass on a routine chest film. Computerized tomography demonstrated its origin from the left T6 nerve root, erosion of the outer lip of the interverteral foramen and no dumb-bell extension into the spinal canal (Fig. 1). At another hospital a benign plexiform neurofibroma was electively excised through a left posterolateral thoracotomy. The patient awoke with a left Brown–Sequard syndrome, which progressed rapidly and required emergency decompressive laminectomy late that night. Excessive serous pleural drainage was noted postoperatively. The chest tubes were eventually removed and the patient transferred to our hospital 2 weeks after operation for treatment and rehabilitation of a left lower extremity paralysis.

On admission, the clinical diagnosis of a large left pleural effusion was confirmed radiologically. Thoracentesis was done, and examination of the milky fluid revealed a triglyceride content eight times that of plasma. A low-lying left intercostal drainage tube was inserted. The patient was placed on an elemental diet, but dietary intolerance required the institution of total parenteral nutrition (TPN). In spite of re-expansion of her left lung, adequate dependent drainage and "thoracic duct rest", the fistulous drainage persisted at a rate of 600 to 1200 ml/d. The left chest tube was repositioned and then replaced, with no effect.

A contrast lymphangiogram (Fig. 2) and a nuclear lymphangiogram (Fig. 3) demonstrated disruption of the thoracic duct with extravasation into the left pleural space. A left subclavian venogram confirmed a patent subclavian vein, thus ruling out venous occlusion as a cause for the persistent chylothorax. Twenty-seven days after admission, after the patient was given 200 ml of cream 6 hours preoperatively by nasogastric tube, supradiaphragmatic ligation of the thoracic duct was performed through a right posterolateral thoracotomy.

Postoperatively, the chest tube drainage decreased to 200 to 600 ml/d. A repeat nuclear lymphangiogram showed successful treatment of the chylothorax. However, persistent left-chest-tube drainage necessitated a left posterolateral thoracotomy 3 weeks later. A subarachnoid–pleural fistula was identified at

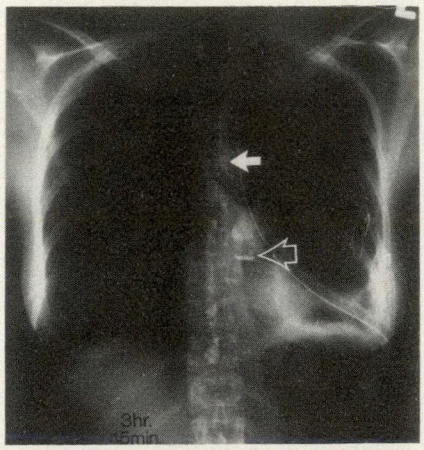

FIG. 2—Contrast lymphangiogram demonstrates chylous leak in vicinity of T6 vertebra (closed white arrow). Contrast material then flows freely into left pleural space after traversing paramediastinal cavity containing air-fluid level (open white arrow).

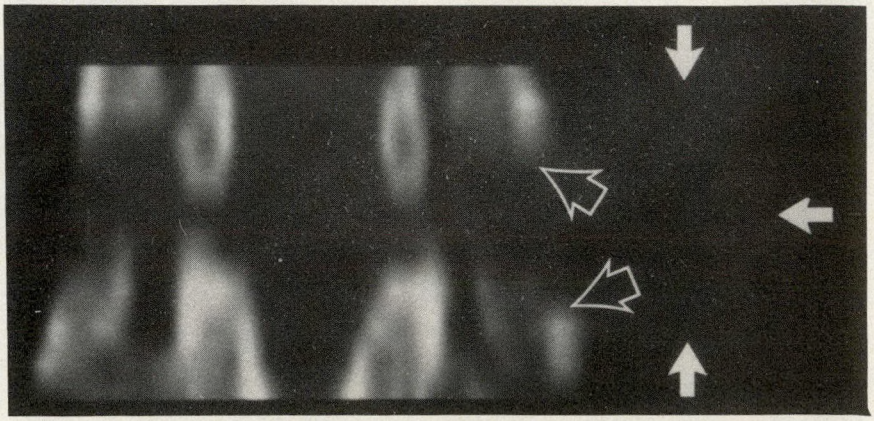

FIG. 1—Computerized tomogram (coronal view) of T6 vertebra showed neurofibroma arising from left T6 nerve root (closed white arrows). Erosion of superior and inferior lips of intervertebral foramen can be seen (open white arrows), but no dumb-bell extension into spinal canal.

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Accepted for publication Jan. 12, 1987

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the site of the previous excision. Dural closure could not be obtained, so a pedicled pleural patch was sutured to the tissue edges about the intervertebral foramen, using interrupted 3-0 silk sutures. The left lung was decorticated, the parietal pleura abraded and the subarachnoid space decompressed by continuous closed lumbar subarachnoid drainage. Postoperatively, the patient remained on bed rest with limited elevation of the head of the bed. The left-chest-tube drainage diminished considerably. Four days postoperatively the lumbar drain was removed and the chest tube drainage promptly increased to 200 to 400 ml/d. A nuclear myelogram (Fig. 4) demonstrated a persistent left subarachnoid–pleural fistula. Continuous closed lumbar subarachnoid drainage was reinstituted and after 1 week the fistula had closed. When the lumbar drain was removed the patient became febrile and had the signs and symptoms of meningitis. Culture of the cerebral spinal fluid grew *Pseudomonas aeruginosa*. The infection promptly responded to ceftazidime, which was continued in a dosage of 2 g every 8 hours for 2 weeks. The patient was transferred to our rehabilitation unit and was discharged home 14 weeks after transfer to our hospital, walking unaided with a minimal left foot drop. Her discharge chest x-ray film showed left pleural thickening (Fig. 5).

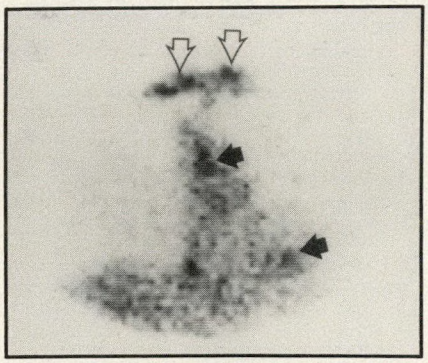

FIG. 3—Nuclear lymphangiogram demonstrates left chylothorax (closed black arrows). Some contrast material reaches supraclavicular nodes (open black arrows). Infradiaphragmatic uptake is in liver and spleen.

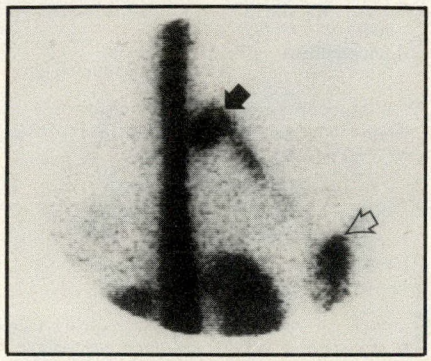

FIG. 4—Nuclear myelogram demonstrates left subarachnoid–pleural fistula (closed black arrow). Contrast material drains through chest tube into reservoir on patient's chest (open black arrow).

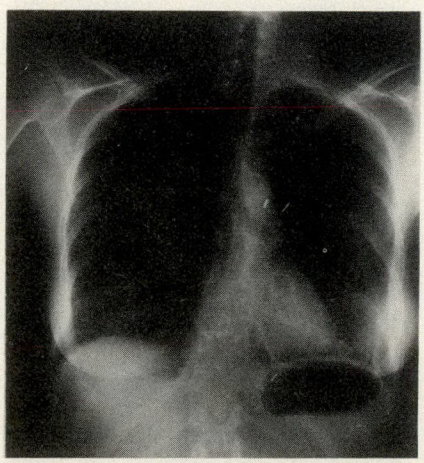

FIG. 5—Chest film at time of discharge from hospital shows minimal left pleural thickening, blunting of right costophrenic angle and surgical clips at site of fistulas.

Comments

Chylothorax

The anatomy of the thoracic duct is extremely variable. Its "normal" course is rarely seen (Fig. 6). The thoracic duct originates as the efferent vessel of the cisterna chyli, adjacent to the L3–T10 vertebral bodies. It lies to the right of the aorta, passing into the posterior mediastinum through the aortic hiatus. Here, in possibly the most constant location in the thorax, it is situated anterior to the vertebral bodies, posterior to the esophagus, to the left of the azygos vein and to the right of the aorta. At approximately the level of the T5 vertebral body, the duct passes behind the aorta, to lie on its left. At the apex of the thorax (root of the neck) the duct passes behind the carotid sheath and then arches anteriorly to enter the venous system at the junction of the left internal jugular and left subclavian veins. Along this course there are rich collaterals and anastomoses. This allows for safe ligation without sequelae.

Injury to the thoracic duct occurs most frequently during surgery about the thoracic aorta or esophagus but can complicate any thoracic procedure. Awareness of the variable anatomy of the duct is most important in preventing a chylothorax. Careful dissection about the probable course of the duct and meticulous control of suspected chylous leaks at operation are essential. If damaged, the duct may be safely ligated[2] or repair attempted.[3] In our hands ligation has been simple, safe and effective.

The diagnosis of a chylothorax is well documented.[2] Fluid analysis measuring total lymphocyte count, chylomicrons and triglyceride and cholesterol concentrations is helpful. Contrast studies are the mainstay of diagnosis. We have found nuclear lymphangiography, using technetium-99m antimony colloid to be a new, but useful, diagnostic tool. It is easy to administer, well tolerated and eliminates the potential pulmonary complications of standard contrast lymphangiography. In this case, nuclear lymphangiograms verified the presence of the chylous fistula and recorded its closure. Precise localization of the fistulous site can be difficult but if required, as in our case, standard contrast lymphangiography should be done.

Nonoperative management of a chylothorax includes an elemental diet or total parenteral nutrition. Administration of total parenteral nutrition through the left subclavian vein can be complicated

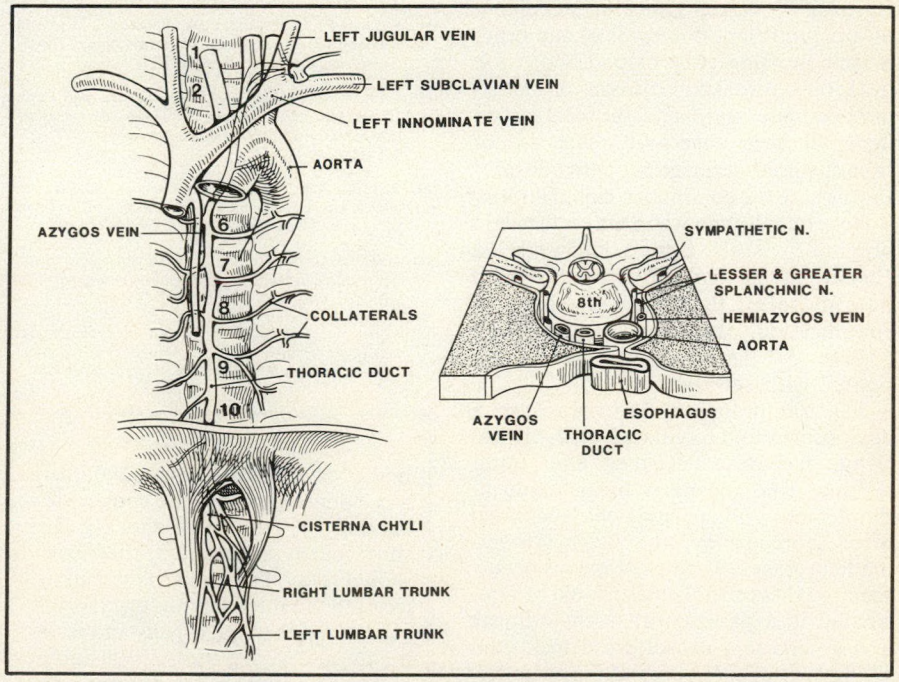

FIG. 6—Typical course of thoracic duct. Its most constant portion within thorax is depicted on right.

by venous thrombosis and paradoxically delayed fistula closure. For this reason we try not to use the left subclavian vein when treating a chylothorax and perform subclavian venography when conservative therapy fails.

We prefer the supradiaphragmatic approach for ligation of the thoracic duct.[2] Generally, a right thoracotomy is used; however, concomitant or persistent disease in the left pleural space may necessitate a left thoracotomy. In our case, an unsuspected subarachnoid–pleural fistula was masked by the chylous fistula, which was managed by a right-sided approach. Later, a left thoracotomy was needed to manage the subarachnoid–pleural fistula.

Subarachnoid–Pleural Fistula

This fistula is an uncommon entity. It occurs most frequently after blunt or penetrating trauma[1,4–9] but is rarely seen after thoracotomy.[10–12] The fistula may be suspected on plain films of the skull,[12] thoracic spine or chest. It may be demonstrated by myelography,[4,5,7,8,10] but if high-viscosity material is used there may not be free flow throughout the subarachnoid space, thus precluding the demonstration of a small fistula.[13] Radionuclide myelography is useful.[4,9,10,12,13] Analysis of the cerebrospinal fluid collected from the pleural space may be misleading, for a "dialysis effect" may alter its typical composition.

Treatment of a subarachnoid–pleural fistula by intercostal tube drainage alone may be successful.[6,7,11] Operative management may be conducted through a posterior laminectomy[4,5,8,10] or thoracotomy, or both.[1,4,9,12] Direct suture closure,[4,8,10] fascial grafting,[5] placement of a pleural flap,[1,4] intercostal and other muscle grafting (free or pedicled),[4] use of thrombin-soaked Gelfoam[4] or methyl methacrylate seal,[9] and thoracoplasty[12] have all been successful. The use of subarachnoid drainage is controversial.[14] In this case the continuous closed lumbar subarachnoid drain was a major factor in obtaining fistula closure. However, a *Pseudomonas* meningitis was uncovered on removal of the drain. This dreaded complication, which previously carried a 55% to 85% death rate, was successfully treated with ceftazidime.[15]

The simultaneous occurrence of a chylothorax and a subarachnoid–pleural fistula has been described after blunt trauma,[1] the diagnosis being made at thoracotomy. In our case, which occurred after thoracotomy, the diagnosis was made in stages. The simultaneous occurrence of these two fistulas should be considered in a patient with a chylothorax and a persistent neurologic defect, who presents after suffering trauma or undergoing thoracic neurosurgery. When the chylothorax fails to respond to standard therapy, in this setting, a diagnosis of a simultaneous subarachnoid–pleural fistula should be considered. Diagnosis by analysis of thoracentesis fluid is difficult because of contamination of the cerebrospinal fluid by chyle. Contrast and nuclear studies of each individual fistula are most useful. Staged surgical management was successful in our case; however, a single procedure is desirable.

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Correction

In the May 1987 issue, page 220, there was a printing error in the book review entitled "Treatment of shock". The short fourth paragraph of this review should read "The text is in three parts: treating the microcirculatory lesions, supporting the vital organs and treating the underlying cause." We apologize to Dr. Downs, the reviewer, for this error.

24 hours following surgery increases the possibility of adverse reactions but, in the majority of surgical procedures, does not reduce the incidence of subsequent infection.

If signs of postsurgical infection should appear, specimens for culture should be obtained for identification of the causative organism(s) so that appropriate therapy may be instituted.

CONTRAINDICATIONS

MEFOXIN* is contraindicated in persons who have shown hypersensitivity to cefoxitin or to the cephalosporin group of antibiotics.

WARNINGS

Before therapy with MEFOXIN* is instituted, careful inquiry should be made to determine whether the patient has had previous hypersensitivity reactions to MEFOXIN*, cephalosporins, penicillins or other drugs. MEFOXIN* should be given with caution to penicillin-sensitive patients.

There is some clinical and laboratory evidence of partial cross-allergenicity between cephamycins and the other beta-lactam antibiotics, penicillins and cephalosporins. Severe reactions (including anaphylaxis) have been reported with most beta-lactam antibiotics.

Pseudomembranous colitis has been reported with virtually all antibiotics. This colitis can range from mild to life threatening in severity. Antibiotics should therefore be prescribed with caution in individuals with a history of gastrointestinal disease, particularly colitis. It is important to consider a diagnosis of pseudomembranous colitis in patients who develop diarrhea in association with antibiotic use. While studies indicate that a toxin produced by *Clostridium difficile* is one primary cause of antibiotic-associated colitis, other causes should also be considered.

Any patient who has demonstrated some form of allergy, particularly to drugs, should receive antibiotics including MEFOXIN* with caution.

If an allergic reaction to MEFOXIN* occurs, administration of the drug should be discontinued. Serious hypersensitivity reactions may require treatment with epinephrine and other emergency measures.

PRECAUTIONS

The total daily dosage should be reduced when MEFOXIN* is administered to patients with transient or persistent reduction of urinary output due to renal insufficiency (see DOSAGE AND ADMINISTRATION) because high and prolonged serum antibiotic concentrations can occur from usual doses.

In patients treated with MEFOXIN* a false-positive reaction to glucose in the urine may occur with Benedict's or Fehling's solutions but not with the use of specific glucose oxidase methods.

Using the Jaffe Method, falsely high creatinine values in serum may occur if serum concentrations of cefoxitin exceed 100 μg/mL. Serum samples from patients treated with MEFOXIN* should not be analyzed for creatinine if withdrawn within two hours of drug administration.

Increased nephrotoxicity has been reported following concomitant administration of cephalosporins and aminoglycoside antibiotics.

The safety of MEFOXIN* in the treatment of infections during pregnancy has not been established. If the administration of MEFOXIN* to pregnant patients is considered necessary, its use requires that the anticipated benefits be weighed against possible hazards to the fetus. Reproductive and teratogenic studies have been performed in mice and rats and have revealed no evidence of impaired fertility or harm to the fetus due to MEFOXIN*.

Cefoxitin has been observed in the milk of nursing mothers receiving the drug.

Prolonged use of MEFOXIN* may result in the overgrowth of non-susceptible organisms. Repeated evaluation of the patient's condition is essential and if super-infection occurs during therapy, appropriate measures should be taken. Should an organism become resistant during antibiotic therapy, another antibiotic should be substituted.

In children 3 months of age or older, higher doses of MEFOXIN* (100 mg/kg/day and above) have been associated with an increased incidence of eosinophilia and elevated SGOT.

ADVERSE REACTIONS

MEFOXIN* is generally well tolerated. Adverse reactions rarely required cessation of treatment and usually have been mild and transient.

Local Reactions
Thrombophlebitis has occurred with intravenous administration. Some degree of pain and tenderness is usually experienced after intramuscular injections using water. Induration has occasionally been reported.

Allergic
Maculopapular rash, urticaria, pruritus, eosinophilia, fever and other allergic reactions have been noted.

Gastrointestinal
Symptoms of pseudomembranous colitis can appear during or after antibiotic treatment. Nausea and vomiting have been reported rarely.

Blood
Transient eosinophilia, leukopenia, neutropenia, hemolytic anemia, and thrombocytopenia have been reported. Some individuals, particularly those with azotemia, may develop positive direct Coombs tests during therapy with MEFOXIN*.

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

Kidney
Elevations in serum creatinine and/or blood urea nitrogen levels have been observed. As with the cephalosporins, acute renal failure has been reported rarely. The role of MEFOXIN* in changes in renal function tests is difficult to assess, since factors predisposing to prerenal azotemia or to impaired renal function have often been present.

TREATMENT OF OVERDOSE

Other than general supportive treatment, no specific antidote is known. MEFOXIN* can be eliminated by dialysis in patients with renal insufficiency.

DOSAGE AND ADMINISTRATION

MEFOXIN* may be administered intravenously or intramuscularly when required. (See complete monograph for full details on ADMINISTRATION and RECONSTITUTION.)

TREATMENT DOSAGE

Adults

The usual adult dosage is 1 g or 2 g of MEFOXIN* every 6 to 8 hours. Dosage and route of administration should be determined by severity of infection, susceptibility of the causative organisms, and condition of the patient. The usual adult dosages are shown in the Table below.

Usual Adult Dosage

Type of infection	Daily Dosage	Frequency and Route
Uncomplicated forms* of infections such as pneumonia, urinary tract infection, soft tissue infection	3-4 g	1 g every 6-8 h I.V. or I.M.
Moderately severe or severe infections	6-8 g	1 g every 4 h or 2 g every 6-8 h I.V.
Infections commonly needing antibiotics in higher dosage (e.g. gas gangrene)	12 g	2 g every 4 h or 3 g every 6 h I.V.

*Including patients in whom bacteremia is absent or unlikely

Therapy may be started while awaiting the results of susceptibility testing.

Antibiotic therapy for group A beta-hemolytic streptococcal infections should be maintained for at least 10 days to guard against the risk of rheumatic fever or glomerulonephritis. In staphylococcal and other infections involving a collection of pus, surgical drainage should be carried out where indicated.

Adults with Impaired Renal Function

MEFOXIN* may be used in patients with reduced renal function but a reduced dosage should be employed and it is advisable to monitor serum levels in patients with severe impairment.

In adults with renal insufficiency, an initial loading dose of 1 g to 2 g should be given. After a loading dose, the following recommendations for **maintenance dosage** may be used as a guide:

RENAL FUNCTION	CREATININE CLEARANCE mL/min	DOSE	FREQUENCY
Mild impairment	50-30	1-2 g	every 8-12 h
Moderate impairment	29-10	1-2 g	every 12-24 h
Severe impairment	9-5	0.5-1 g	every 12-24 h
Essentially no function	<5	0.5-1 g	every 24-48 h

In the patient undergoing hemodialysis, the loading dose of 1-2 g should be given after each hemodialysis, and the maintenance dose should be given as indicated in the Table above.

Neonates (Including Premature Infants), Infants and Children (See WARNINGS for Neonates under ADMINISTRATION in the complete monograph.)

Premature Infants with Body Weights Above 1500 g	20-40 mg/kg every 12 h I.V.
Neonates	
0-1 week of age	20-40 mg/kg every 12 h I.V.
1-4 weeks of age	20-40 mg/kg every 8 h I.V.
Infants	
1 month to 2 years of age	20-40 mg/kg every 6 h or every 8 h I.M. or I.V.
Children	20-40 mg/kg every 6 h or every 8 h I.M. or I.V.

In severe infections, the total daily dosage in infants and children may be increased to 200 mg/kg, but not to exceed 12 g per day.

MEFOXIN* is not recommended for the therapy of meningitis. If meningitis is suspected, an appropriate antibiotic should be used.

At present there is insufficient data to recommend a specific dosage for children with impaired renal function. However, if the administration of MEFOXIN* is deemed to be essential the dosage should be modified consistent with the recommendations for adults (see Table above).

PROPHYLACTIC USE

For prophylactic use, a three-dose regimen of MEFOXIN* is recommended as follows:

Vaginal or abdominal hysterectomy and abdominal surgery

2 g administered intramuscularly or intravenously just prior to surgery (approximately one-half to one hour before initial incision).

The second and third 2 g doses should be administered at 2-6 hour intervals after the initial dose.

Cesarean Section

The first dose of 2 g should be administered intravenously as soon as the umbilical cord has been clamped. The second and third 2 g doses should be given intravenously or intramuscularly four hours and eight hours after the first dose.

AVAILABILITY

MEFOXIN* (sterile cefoxitin sodium, Frosst Std.) is supplied as sterile powder in boxes of 10 vials:

No. 3356 1 g cefoxitin as sodium salt
No. 3357 2 g cefoxitin as sodium salt

Storage
MEFOXIN* in the dry state should be stored below 30° C.

PRODUCT MONOGRAPH AVAILABLE ON REQUEST

421-a,11.84

MEMBER
PMAC
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Frosst
P.O. BOX 1005, POINTE-CLAIRE
DORVAL, QUEBEC H9R 4P8

P.A. REILLY, PH D; M. THOMPSON, MD; A. BAYER, RN; F. KAZIM, MA, M SC; J. ROSENFELD, PH D; M.F.X. GLYNN, MD, D PHIL, MRCP(LOND), FRC PATH

Preservation of Platelets and Blood Products by Intravenously Administered Dipyridamole in Patients Who Undergo Coronary Artery Bypass Grafting

The combination of dipyridamole and acetylsalicylic acid has been proven effective in preventing coronary artery bypass graft occlusion, but the benefits of dipyridamole alone have not yet been evaluated. In order to assess the value of dipyridamole alone, the authors randomized 24 patients (age range from 47 to 76 years) who underwent coronary artery bypass grafting to treatment with either dipyridamole (120 mg/d) by constant intravenous infusion or isotonic dextrose solution. They recorded platelet counts and aggregates, hemoglobin levels, total blood loss, blood products and intravenous fluids given and dipyridamole plasma levels, starting 8 hours before operation and continuing for 3 days after.

The two groups were similar with respect to pump time, cross-clamp time and baseline demographic factors. Platelet counts during cross-clamping and 1 hour postoperatively were similar, but those on days 1, 2 and 3 postoperatively were significantly ($p = 0.01$ to 0.02) higher in the dipyridamole group. Mean blood losses in this group were 22% to 30% lower, but the difference was not significant. However, administration of erythrocytes and plasma was 49% to 58% less in the dipyridamole group ($p = 0.005$ to 0.048) over the same period. Dipyridamole plasma concentrations varied from 0.37 μg/ml before and during bypass to 1.5 μg/ml in the 3 days after.

The authors conclude that dipyridamole administered intravenously to patients who undergo coronary artery bypass grafting may preserve hemostatically effective platelets so that fewer blood products are required.

L'association dipyridamole/acide acétylsalicylique a démontré son efficacité dans la prévention de l'occlusion des pontages aortocoronariens. Toutefois, l'utilité du dipyridamole seul n'a pas été évaluée. Dans le but d'évaluer l'effet du dipyridamole, les auteurs ont soumis au hasard 24 patients (âgés de 47 à 76 ans) qui ont subi un pontage aortocoronarien à un traitement qui renfermait soit du dipyridamole (120 mg/j) en perfusion intraveineuse continue, soit une solution de dextrose isotonique. Ont été enregistrés: le décompte et l'agrégation plaquettaires, le taux d'hémoglobine, la perte de sang total, les dérivés sanguins et les solutés intraveineux administrés et les concentrations plasmatiques de dipyridamole, de la 8e heure jusqu'au 3e jour après l'opération.

Les deux groupes étaient comparables en ce qui a trait au temps de pompe, au temps de clampage et aux facteurs démographiques de départ. Les décomptes plaquettaires ont été similaires pendant la clampage et à la première heure après l'opération; toutefois aux jours 1, 2 et 3 après l'opération, ils étaient significativement plus élevés ($p = 0.01$ à 0.02) dans le groupe dipyridamole. Les pertes sanguines dans ce groupe ont été de 22% à 30% inférieures mais cette différence n'est pas significative. Cependant, pendant cette même période l'administration de sang entier et de plasma a été de 49% à 58% moindre dans le groupe dipyridamole ($p = 0.005$ à 0.048). Les concentrations plasmatiques de dipyridamole ont varié de 0.37 μg/ml avant l'opération jusqu'à 1.5 μg/ml dans les 3 jours qui suivirent.

Les auteurs concluent que l'administration intraveineuse de dipyridamole aux patients qui subissent un pontage aortocoronarien peut sauvegarder l'hémostase des plaquettes, réduisant de la sorte les besoins en dérivés sanguins.

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Supported in part by a grant from Boehringer Ingelheim (Canada) Ltd., Burlington, Ont.

Accepted for publication Dec. 9, 1986

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Dipyridamole (Persantine; Boehringer Ingelheim [Canada] Ltd., Burlington, Ont.) is an inhibitor of platelet function and has been used in combination with warfarin and acetylsalicylic acid to prevent thromboembolism associated with cardiac valve prostheses,[1] stroke,[2] coronary bypass graft occlusion[3] and myocardial infarction.[4] However, there have been little data establishing the effectiveness of dipyridamole alone. Claimed benefits over acetylsalicylic acid alone rest on indirect evidence[4] or have yet to be substantiated.[5,6] In order to determine whether dipyridamole alone affects platelets in coronary artery bypass grafting, we gave the drug as an intravenous infusion and measured the number of platelets and degree of hemostasis in patients who underwent elective coronary artery bypass grafting.

Patients and Methods

The study population comprised 24 patients (19 men, 5 women), ranging in age from 47 to 76 years, scheduled to undergo elective coronary artery bypass grafting. All had platelet counts in the normal range, had had no concomitant antiplatelet or anticoagulant treatment for at least 1 week before surgery and had no evidence of heart failure. Each patient gave informed consent. The protocol and consent forms were approved by the institutional Human Experimentation Committee. Only one of the authors, responsible for randomization and study organization, had access to the study code. Surgery, sample collection, postoperative patient management and assays were all carried out under blinded conditions.

The patients were randomized to two groups of 12 patients. One group received dipyridamole (120 mg/d), the other isotonic dextrose. Intravenous dipyridamole or the placebo was started at least 8 hours before operation and continued for 2 or 3 days postoperatively. The drug ampoules (Boehringer Ingelheim [Canada] Ltd., Burlington, Ont.) were mixed in a 5% dextrose in water solution

and infused at a rate of 12 ml/h to a dose of dipyridamole of 120 mg/d. The isotonic dextrose was infused at a rate of 12 ml/h. Based on pharmacokinetic considerations and oral bioavailability,[7] we estimated that this regimen would yield plasma concentrations intermediate between peaks and troughs after oral dosing of 75 mg three times daily and minimize variation due to absorption.

Anesthesia and cardiopulmonary bypass procedures were standardized. Anticoagulation intraoperatively was with heparin (3 mg/kg), cardioplegia was by cold potassium solution (25° to 28°C) and the bypass pump was primed asanguineously and operated with a bubble oxygenator. The heparin effect was neutralized postoperatively with protamine sulfate (1 mg/kg).

After operation all patients were managed routinely in the intensive care unit by the regular staff who had no knowledge of patient randomization. Hemoglobin concentrations were maintained at 100 g/L or greater by the transfusion of packed cells. Plasma, albumin or crystalloid was used for volume replacement to maintain the left atrial pressure at 8 to 10 mm Hg. No patient in either group required reoperation or infusions of platelets or cryoprecipitate to manage bleeding.

Various venous blood samples were taken pre- and postoperatively and by arterial catheter intraoperatively. Erythrocyte counts, platelet counts and hematocrit and hemoglobin levels were analysed by an automatic counter. Circulating platelet aggregates were determined by the method of Wu and Hoak,[8] and the aggregation index was calculated as the platelet count in plasma plus ethylene diamine tetra-acetic acid (EDTA) and formalin divided by the platelet count in plasma plus EDTA. Blood samples were taken 24 hours preoperatively (before infusion), immediately preoperatively (after infusion achieved a steady level), intraoperatively (during cross-clamping), 1 hour postoperatively (in the recovery room) and 24, 48 and 72 hours postoperatively. Intraoperative blood loss, postoperative fluid balance, blood losses at 0 to 12 hours, 12 to 24 hours and 24 to 48 hours postoperatively, and blood products (plasma, packed cells) and fluids given in the same postoperative periods were recorded. Dipyridamole plasma concentrations were determined by high-pressure liquid chromatography.[9]

Statistical Analyses

Statistical analyses were carried out using the BMDP statistical programs.[10] Analyses of variance techniques for a two-factor repeated-measures design with one grouping factor (drug) were employed. Unpaired *t*-tests were used for comparisons of individual time points and clinical variables. Absolute and percentage changes from baseline were used as a measure of drug effect, when baseline measurements were comparable.

Table I—Surgical Profile*

Variable	Dipyridamole (n = 12, 2 women)	Placebo (n = 12, 3 women)	p value for group difference†
Age, yr	54.5 ± 6.6 (47 – 67)	58.4 ± 9.12 (48 – 76)	0.58
Preoperative hemoglobin, g/L	142 ± 10.3 (129 – 157)	133 ± 12.1 (113 – 154)	0.08
Preoperative platelet counts, $\times 10^9$/L	228.3 ± 38.5 (166 – 286)	243.2 ± 56.1 (168 – 379)	0.46
Preoperative aggregate ratio	1.00 ± 0.14 (0.76 – 1.16)	1.07 ± 0.14 (0.89 – 1.28)	0.21
Number of bypassed vessels	3.00 ± 0.95 (2 – 4)	2.60 ± 0.51 (2 – 4)	0.26
Time on pump, min	89.9 ± 25.0 (54 – 131)	83.0 ± 18.8 (60 – 110)	0.49
Aortic cross-clamp time, min	35.3 ± 18.9 (9 – 57)	37.1 ± 18.7 (10 – 60)	0.83
Intraoperative fluids given, ml‡	4205 ± 514 (3500 – 4900)	3717 ± 1104 (2250 – 5300)	0.25
Intraoperative blood loss, ml	683 ± 510 (250 – 1900)	648 ± 332 (300 – 1400)	0.87
Postoperative fluid balance, ml§	4330 ± 1166 (2550 – 6750)	3562 ± 1172 (2400 – 6050)	0.16

*Values are means with standard deviations, numbers in brackets are value ranges.
†Test of differences is unpaired *t*-test.
‡Sum of asanguineous pump prime plus packed cells.
§Blood products and crystalloids given minus blood loss and urine output.

Table II—Hemoglobin Concentrations (g/L)*

Sample	Dipyridamole	Placebo	p value†
Preoperative, 24 h	142 ± 10.3	133 ± 12.1	0.08
Post-drug, 1 h preop	140 ± 10.3	131 ± 13.5	0.07
Intraoperative, on cross-clamp	82.5 ± 10.8	75.7 ± 13.0	0.20
Postop, 1 h	111 ± 15.0	111 ± 16.0	0.91
Postop, 24 h	108 ± 6.2	111 ± 13.2	0.50
Postop, 48 h	108 ± 16.5	115 ± 13.7	0.28
Postop, 72 h	104 ± 9.7	116 ± 15.3	0.04

*Values are mean ± standard deviations.
†p value is based on unpaired *t*-test.

Results

Table I presents details of the bypass procedure. Hemoglobin and hematocrit values and erythrocyte count were all slightly higher in the dipyridamole group but were similar to the placebo group by 1 hour postoperatively (Table II).

Changes in platelet counts and circulating platelet aggregates are plotted in Fig. 1. Overall analysis of change from baseline for platelet counts was significantly different ($p = 0.023$) for the two treatment groups, but pairwise comparison of the time points confirms the impression from Fig. 1 that this is primarily due to differences at 1, 2 and 3 days postoperatively ($p = 0.024$, 0.019 and 0.020, respectively). There was no significant change in circulating aggregates.

Cumulative blood losses over 48 hours postoperatively and blood products administered are given in Table III, and losses in the first 12 hours are shown in Table IV. Only 18 patients had complete data. Patients treated with dipyridamole lost 20% to 30% less blood than the placebo-treated patients, but the difference was not statistically significant. Nevertheless, the amount of blood products needed by the treated group to maintain the standard hemoglobin level after operation was significantly less. Dipyridamole plasma concentrations are shown in Fig. 2.

Discussion

Activated by the trauma of cardiopulmonary bypass, platelets adhere avidly to injured endothelium and synthetic surfaces. Once platelets have adhered, the sequence of activation, adhesion and the internal generation and release of bioactive materials begins. The widespread platelet consumption and partial activation of other platelets deplete their absolute number and leave many impaired

residual platelets in the circulation. These residual circulating platelets respond abnormally when tested in vitro and thus may contribute, as does the diminished number of platelets, to postoperative hemorrhage.[11]

In our study, mean (± standard error) hemoglobin concentrations dropped 42.2% ± 1.7% (dipyridamole) and 43.9% ± 2.8% (placebo) during aortic cross-clamping compared with baseline values, and platelet counts dropped 47.3% ± 2.8% and 49.2% ± 3.8% respectively. The proportionately greater loss of platelets confirms platelet sequestration or consumption. In patients treated with dipyridamole, the recovery of platelet counts was more rapid postoperatively than in patients not so treated. The time course of new platelet synthesis from megakaryocytes is in the order of 3 to 7 days after a thrombocytopenic stimulus, so that a stimulus to thrombopoiesis by dipyridamole is less likely than direct platelet-sparing.[12]

Intraoperative blood losses and blood products used (Table I) were quite similar in the two treatment groups, in agreement with the findings of Chesebro and colleagues.[3] During this period, patients received heparin anticoagulation, so the effects of an antiplatelet agent might not be detectable. After heparin reversal with protamine, both variables of platelet counts and usage of blood products separate by treatment group. A previous report[13] studied postoperative blood losses and blood products given for 15 hours after open-heart surgery in patients on dipyridamole and in a control series but detected no differences. However, the dipyridamole dose was 0.5 mg/kg given as a single dose into the heart–lung machine, about one-quarter of the dose given in this study.

The pattern of blood products used and blood losses in the recovery period mirror the observations on platelet counts. This implies that the platelets preserved by dipyridamole are functional. The platelet-sparing and improved hemostasis by dipyridamole in this study stands in marked contrast to the effects of similar drugs such as acetylsalicylic acid and prostacyclin, which may preserve platelets but at the expense of increased risks of hemorrhage and bleeding complications.[14,15]

Other groups have recorded elevations of platelet counts after dipyridamole treatment and the thrombocytopenic effect of surgery, in both animals[16,17] and man.[13,18] Miyauchi and Isomura[18] also used an intravenous dose (1 to 2 mg/kg daily) preoperatively, apparently without postoperative drug administration. None of these groups, however, monitored the postoperative use of blood products for 2 days. In one study in dogs,[17] the difference in platelet counts in dipyridamole versus control animals was paralleled by lower indium-111-labelled platelet deposition in the myocardial tissues of the dipyridamole group. This supports the suggestion of decreased platelet consumption in our study. In another animal study, Becker[16] obtained good results by adding dipyridamole to the pump prime. The plasma concentrations of dipyridamole were not monitored in any of these studies, but all the positive results were associated with intravenous use of the drug.

The infusion rate of dipyridamole was calculated to produce plasma concentrations near 0.5 μg/ml and preoperative

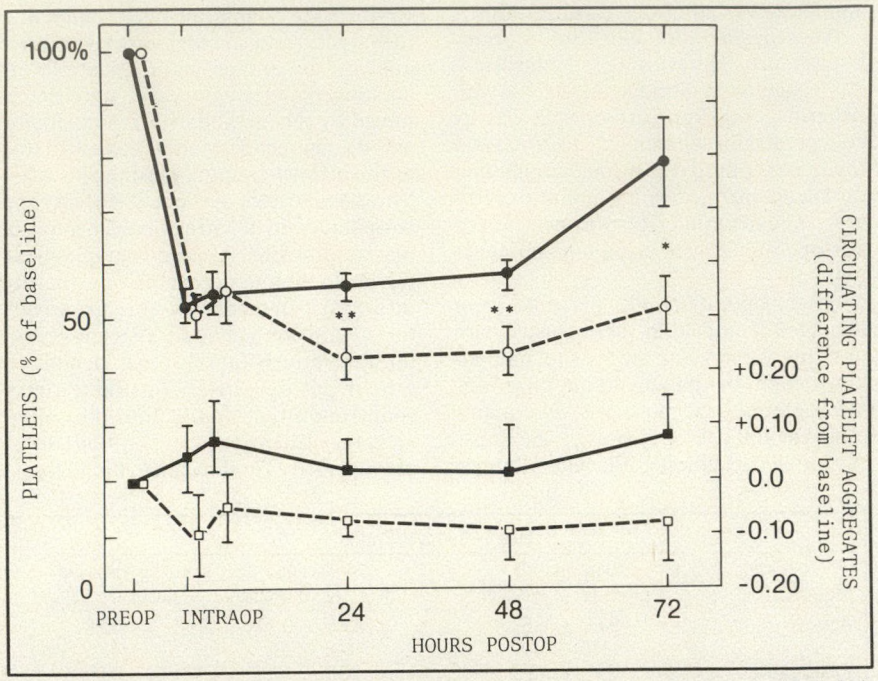

FIG. 1—Platelet counts are shown as percentage of baseline in dipyridamole (solid circles) and placebo (open circles) groups. Values are means with standard-error bars for 11 or 12 patients in each group. Asterisks indicate significant differences between treatments (*p = 0.02, **p = 0.01). Change in platelet aggregate ratio from baseline (means with standard errors) for dipyridamole (solid squares) and placebo (open squares) groups. Positive deviation indicates decrease in presence of circulating aggregates. None of differences between treatments are statistically significant.

Table III—Cumulative Blood Losses and Blood Products Given Postoperatively*

Measurement	Postop time, h	Dipyridamole (n = 10)	Placebo (n = 8)	p value
Blood loss, ml	0–12	366 ± 194	526 ± 294	0.183
	0–24	638 ± 198	841 ± 406	0.183
	0–48	738 ± 242	950 ± 511	0.261
Blood products given, ml	0–12	290 ± 222	688 ± 291	0.005
	0–24	465 ± 493	909 ± 351	0.048
	0–48	515 ± 494	1065 ± 459	0.28

*Values are mean ± standard deviations.

Table IV—Data for Transfusion and Blood Loss (ml) in the First 12 Hours Postoperatively

Dipyridamole			Placebo		
Control	Transfusion	Losses	Control	Transfusion	Losses
b24	500	315	23B	250	300
P22	50	750	21	500	450
P19	200	750	20	500	750
P16	200	Nil	18	500	925
+14	500	775	17	250	430
P13	300	525	15	250	380
P12	200	340	11	250	450
P10	250	125	9	250	600
P8	200	400	7	500	425
P5	200	100	6	—	525
P3	40	325	2	—	325
P1	50	450			

concentrations were in this range (Fig. 2). This is in the middle range of concentrations attained with a dose of 75 mg three times daily,[19,20] a regimen used in long-term trials of bypass graft occlusion[3] and secondary prophylaxis of myocardial infarction.[4] The variation among individuals with the intravenous infusion method here is much less than the 50 to 100-fold range observed with the peaks and troughs of oral dosing. If one considers the preoperative dose of 100 mg four times a day used by Cheseboro and colleagues,[3] then infusion rates must be at least doubled over our regimen to attain dipyridamole concentrations in the 1.0 μg/ml range.[20]

The postoperative changes in platelet counts in our study were associated with two to three-fold increase in dipyridamole concentrations. This accumulation phenomenon may be due to a post-bypass redistribution of fluid (there is a large positive fluid balance postoperatively), an acute phase increase in the production of α_1-acid glycoprotein,[21] which avidly binds dipyridamole, or a decreased biliary excretion of dipyridamole. From our data, we are unable to explain the mechanism of the accumulation and we cannot conclude whether the platelet and blood-product effects are due to the increased concentrations or to intraoperative platelet preservation. We could find no correlation in this study between drug concentration and platelet effects. Others have correlated both aggregation and platelet Factor IV in plasma with dipyridamole concentrations.[22]

The lack of significant effects on platelet aggregates is disappointing, but, since the publication of Wu and Hoak's method,[8] there have been only isolated demonstrations of antiplatelet effects of a drug using this end point.[23] Dipyridamole is particularly devoid of antiaggregation effects using conventional platelet aggregation models.[24] This is thought to be owing to the need for an intact in-vivo preparation for its mechanism of action to be seen. This mechanism has been variously proposed as adenosine uptake inhibition,[24] prostacyclin production[25] and phosphodiesterase inhibition.[24] The initial observation of antiplatelet effects in man occurred at dipyridamole concentrations of 3.75 μg/ml after oral dosing.[26] Extremely high doses in ex-vivo animal models have been required to decrease platelet aggregation or adhesion.[27]

Clinical evaluations of dipyridamole have yielded variable results. A combination of dipyridamole and warfarin significantly decreased the thromboembolic complications in patients with prosthetic heart valves compared with warfarin alone.[1] The combination of dipyridamole and acetylsalicylic acid significantly decreased the incidence of coronary occlusion for 2 years in patients suffering a previous infarction,[4] but comparison with acetylsalicylic acid alone has not shown added benefit. The power of the latter comparison to detect an increase of 50% in effectiveness of acetylsalicylic acid alone is about 20%, so lack of benefit cannot be established in this indication. Chesebro and Fuster[28] have used previous human data to establish the relevance of dipyridamole in bypass graft occlusion and Harajola and colleagues[29] have shown separate and additive effects of acetylsalicylic acid and dipyridamole on occlusion rates of peripheral bypass grafts. In contrast, Brown and colleagues[6] have shown no added effect of dipyridamole in coronary bypass graft occlusion, but both acetylsalicylic acid and dipyridamole were not started until 2 days after operation.

Our data show that dipyridamole alone, infused preoperatively and for 3 days postoperatively, preserves platelets and may thus decrease the necessity for transfusion of blood products in the postoperative period. The platelet-sparing activity of dipyridamole may well have important implications for reducing coronary graft occlusion and the severity of perioperative myocardial ischemia.

We acknowledge the assistance of the surgeons in the Department of Cardiovascular Surgery, Toronto General Hospital, especially Dr. R.D. Weisel. We also thank Mrs. C. Legault and Mrs. L. Deeming for data handling and computer entry and Miss Fiona Matthew for the dipyridamole analyses.

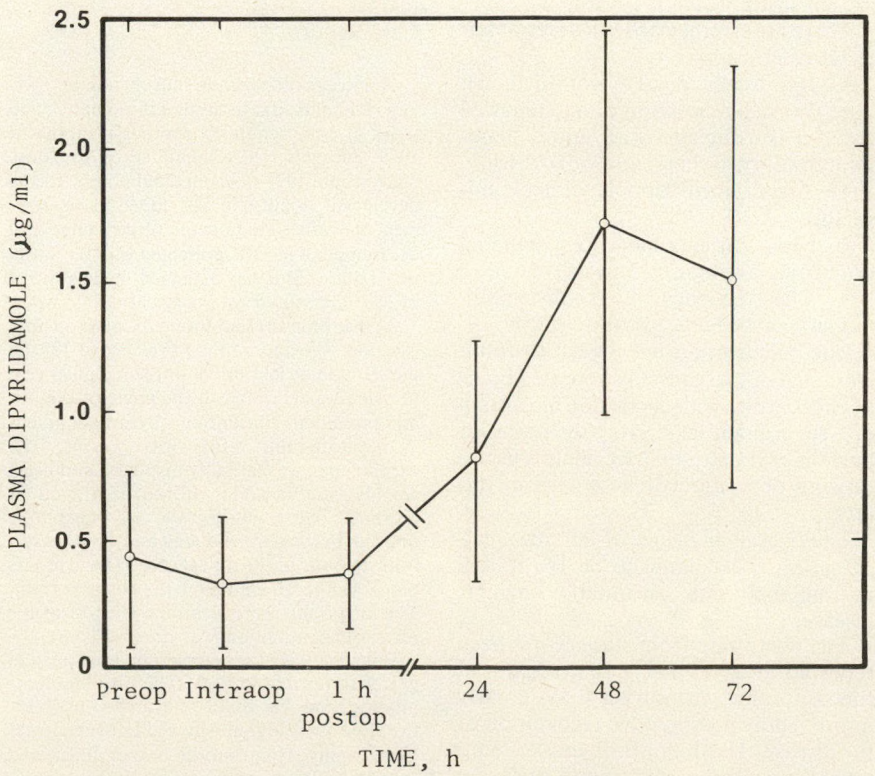

FIG. 2—Plasma concentrations of dipyridamole determined in 11 patients. Error bars show standard deviations. Drug levels rise consistently in postoperative period.

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Successful Surgical Repair in a Patient With Mitral Stenosis, Calcified Left Atrium and Severe Tricuspid Regurgitation With a Giant Right Atrium

Successful mitral valve replacement and tricuspid annuloplasty were performed on a 66-year-old woman who had a calcified left atrium, giant right atrium, calcified mural thrombus and normal pulmonary pressures. Successful repair in such cases depends on an adequate preoperative investigation and a surgical approach tailored to the individual.

On a pratiqué avec succès un remplacement de valvule mitrale et une annuloplastie tricuspidienne chez une femme de 66 ans qui avait une calcification de l'oreillette gauche, une hypertrophie de l'oreillette droite, un thrombus mural calcifié et des pressions pulmonaires normales. Dans de tels cas, le succès de la réparation dépend d'un examen préopératoire adéquat et d'un abord chirurgical individualisé.

Calcification of the left atrium was first described by Oppenheimer in 1912[1] as seen at the autopsy of a patient who died of heart failure secondary to mitral valve disease. The literature was reviewed by Harthorne and colleagues[2] in 1966, bringing the number of reported cases to 88. A further computer search has turned up only 17 more cases — probably because the condition is no longer a medical curiosity.[3–5]

There are several classic features of left atrial calcification:

- It is usually found in women (75%).
- With the exception of one reported case,[6] it is associated with mitral stenosis secondary to rheumatic valve disease.
- Atrial fibrillation is almost universal.
- Most patients have long-standing symptoms averaging 15 to 20 years.
- The frequency of embolism (pulmonary or systemic) is about 50%.

The calcification is believed to result from one of two causes — repeated bouts of endocarditis with deposition of calcium or a regurgitant jet striking the posterior portion of the atrium (McCallum's patch) causing deposition of lime salts in this area.

The overall incidence of left atrial calcification is estimated to be 1% to 2% of patients with rheumatic valvular disease.[7]

The death rate from surgery has been reported at 25%[2] and it is stressed that the outcome of surgery is greatly improved by preoperative recognition of the disease. The major problems encountered are control of hemostasis, difficulty entering the atrium and postoperative embolism.

We report an unusual case in which a huge right atrium, calcified left atrial thrombus and normal pulmonary artery pressures were all present in association with complete left atrial calcification.

Case Report

A 66-year-old woman had rheumatic fever as a child and first became symptomatic at 40 years of age, requiring commissurotomy in 1959. She was well maintained on low-dose digoxin until 1977 when mild pulmonary edema developed. A diuretic was added to her regimen. She refused to undergo surgery on several occasions and in 1983 presented with biventricular failure. She was managed medically but in 1985 consented to a surgical opinion.

At that time she had severe dyspnea on mild exertion. She had a blood pressure of 130/80 mm Hg and atrial fibrillation. Her jugular vein pressure was elevated to the angle of the jaw and prominent fibrillating waves were noted. A hepatojugular reflux was present. The cardiac apex was laterally displaced and heart sounds were variable with an occasional S3 murmur. There was a grade 3/6 pansystolic murmur in the tricuspid area and a milder systolic murmur in the mitral area. The liver was palpable 8 to 10 cm below the costal margin. The lung fields were clear and the rest of the physical examination was unremarkable.

Preoperatively the hemoglobin level was 123 g/L with normal indices. The alkaline phosphatase level was mildly elevated (134 U/L), the electrocardiogram showed left ventricular hypertrophy, right bundle branch block and atrial fibrillation at a rate of 80 beats/min. A chest x-ray film revealed cardiomegaly with a huge right atrium and enlarged left atrium

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Accepted for publication Sept. 29, 1986

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with irregular dense calcification in the left atrial cavity (Fig. 1).

Angiography demonstrated mitral stenosis and regurgitation. Her pulmonary artery pressures were 36/19 mm Hg. The left atrial calcification was not thought to involve the mitral annulus and surgical treatment was advised.

Surgical Approach

The heart was exposed through a midline sternotomy. The large right atrium was very thin and the left atrium was totally calcified. The aorta and both venae cavae were cannulated and the patient was placed on cardiopulmonary bypass. Through the right atrium a Dubost incision was performed to expose both the tricuspid and mitral valves, the former being grossly dilated but with no structural abnormalities. A calcified mass was removed from the left side of the interatrial septum and reported as a calcified mural thrombus. The mitral valve was replaced with a no. 29 Ionescu–Shiley bioprosthesis. Tricuspid annuloplasty using a no. 34 Carpentier–Edwards ring was then performed. The patient was weaned from cardiopulmonary bypass without complication. The clamp time was 134 minutes and bypass time 216 minutes.

Postoperatively, the woman required treatment for heart failure. She responded well and was transferred from the intensive care unit on postoperative day 3. Later a symptomatic pleural effusion was removed by thoracentesis and she was discharged home to the care of her family physician on day 14.

Follow-up 6 months later revealed residual class II symptoms (New York Heart Association classification). The patient complained of only mild dyspnea and there was no evidence of cardiac failure on the chest film.

Discussion

Our patient was unique in several ways. Although all of the following have been reported in association with left atrial calcification, there are no reports of all three coexisting simultaneously.

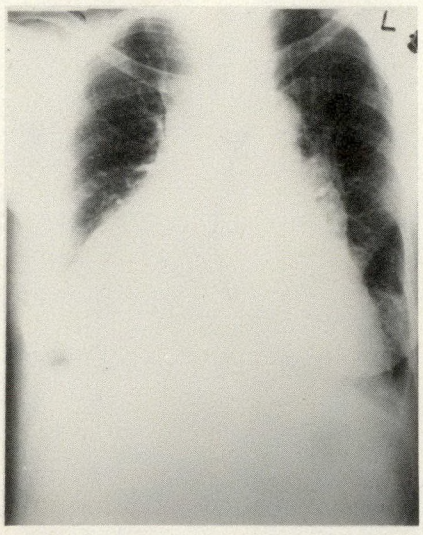

FIG. 1—Chest x-ray film reveals cardiomegaly with huge right atrium, enlarged left atrium and irregular dense calcification in left atrial cavity.

- A giant right atrium was reported by Roberts and associates[8] in a series of three patients. All died intraoperatively or soon after.
- Left atrial thrombus is not uncommon in this condition, although complete calcification is less common.
- Pulmonary artery pressures were normal. Although occurring in 20% of patients, this has never before been reported in a patient with a hugely dilated right atrium.

Proper preoperative evaluation is paramount in these patients. It must include:

(a) A chest x-ray film. The left atrium is seen just inferior to the carina. Well-penetrated films are best, and oblique and lateral projections most useful.

(b) Fluoroscopy. This is a more sensitive modality, especially in delineating the extent of calcification.

(c) Catheterization, which will help differentiate calcification in the coronary arteries.

The information gained from these investigations allows the surgeon to plan alternative approaches, two of the more common being a right thoracotomy with an incision through the posterior interatrial groove and a Dubost incision through the right atrium and interatrial septum.

The high incidence of embolism postoperatively can be minimized by removal of thrombus and by long-term anticoagulation.

We may, therefore, conclude that with proper preoperative investigation and an individualized surgical approach, severe calcification and enlargement of the atria are not contraindications to surgery in patients with severe left atrial calcification.

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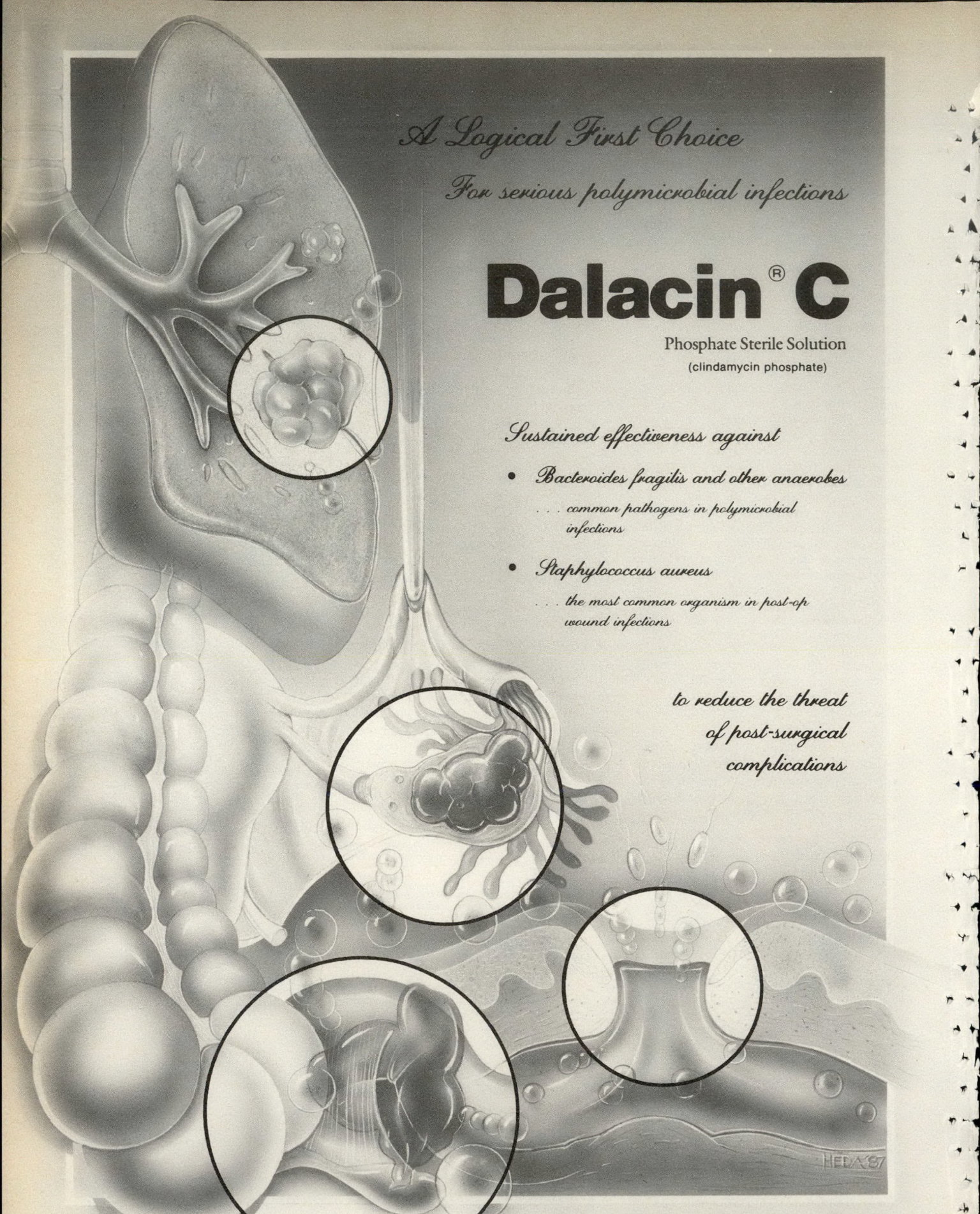

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Repair of Anorectal Incontinence in an Adolescent Boy With Neonatal Anal Atresia

Fecal continence, the ability to retain intestinal contents until evacuation is desired, requires the complex interaction of several factors. They include: the motor action of the sphincters, sensibility of skin and sphincters and function of the rectum and central nervous pathways. Appropriate and effective management is available to treat many disorders of continence. However, the success of these treatments depends on careful delineation of the various contributing factors. The authors present the case of an 18-year-old boy with disabling fecal incontinence, secondary to neonatal anal atresia and its primary management, in order to illustrate the importance of careful assessment of each of the contributing factors. His staged surgical treatment has been successful in returning him to a more normal state.

La continence anale, soit la capacité de retenir le contenu intestinal jusqu'à son évacuation volontaire, exige l'interaction complexe de nombreux facteurs. Parmi ceux-ci, on compte l'action motrice des sphincters, la sensibilité de la peau et des sphincters et le fonctionnement du rectum et des voies de transmission du système nerveux central. Il existe des traitements appropriés et efficaces pour pallier à plusieurs troubles de la continence. Néanmoins, le succès de ces traitements dépend de l'identification soigneuse des divers facteurs en cause. Les auteurs décrivent le cas d'un jeune homme de 18 ans handicapé par une incontinence anale secondaire à une atrésie anale néonatale et à son traitement initial. Ce cas leur permet d'illustrer l'importance d'une évaluation soigneuse de chacun des facteurs impliqués. Un traitement chirurgical par étape a permis à ce jeune homme un retour à une vie normale.

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Accepted for publication Mar. 16, 1987

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Complex derangements of anal incontinence can be managed in such a way as to benefit the patient. We describe the case of a teenage boy who presented with a severe sociosexual disability resulting from fecal incontinence. The derangement of the continence mechanism was complex and related to neonatal anal atresia which was managed by dilatation. His condition was substantially improved by careful delineation of the abnormalities and an operative strategy to correct each one.

Case Report

An 18-year-old boy had congenital anal atresia and an anal dimple. Treatment at birth had consisted of insertion of a probe through the dimple and dilatation. The sphincter was not incised. His mother was instructed to continue dilatations through his early childhood. His continence through childhood and adolescence was marginal. Control was achieved largely through buttock squeeze, dietary manipulation and frequent reconnoitering of accessible toilet facilities. Frequently he was unaware of the seepage. When we saw him his major problem was sociosexual, relating to his frequent leakage and fear of accidents. He refrained from all physical activities and had withdrawn from establishing any female relationships.

There were no obvious physical abnormalities other than that pertaining to the anorectum. The anus was extremely patulous with a large anterior "keyhole" defect and no observable anoderm. The anal canal was 6 cm long, but the rectal mucosa was loose and an ectropion prolapsed 3 cm into the anal canal (Fig. 1). On digital examination a thickened rigid canal was noted which could easily admit two fingers. Squeeze was present in the posterior 120° of the puborectalis and external sphincter. No squeeze could be effected anteriorly. Sigmoidoscopy and barium studies gave normal findings. Further investigations included intravenous pyelography which identified a horseshoe kidney, standard anal electromyography using a needle electrode which showed posterior activity but no activity in 67% of the anterior quadrants and anal manometry using three radially placed strain gauges (Narco Digitized Manometer; Narco Scientific Ltd., Health Dyne Co., Downsview, Ont.) (Table I).

A combined approach to the sphincter mechanisms and skin defects was designed. With the patient in the prone, jack-knife position, a curvilinear incision was made in the anterior anal verge for approximately 180°. The healthy lateral margins of normal sphincter were first dissected out to a depth of

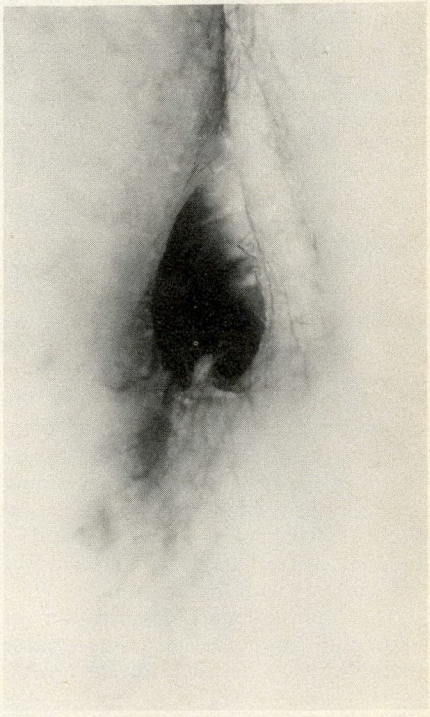

FIG. 1—Combination of defects including keyhole effect and mucosal ectropion.

Table I—Results of Manometry

Site	Pressures, mm Hg	
	Resting*	Squeeze†
Anterior	22	30
Lateral	30	45
Posterior	30	50

*Normal = 50 to 75 mm Hg.
†Normal = 50 mm Hg increase over resting pressure.

approximately 3 cm, the deep dissection was carried anteriorly to the scar. No intraoperative electromyography was used since our preoperative results had been precise and the demarcation between healthy sphincter and scar was clear. The latter was quite attenuated and difficult to dissect, but once dissection was completed the scar was divided at its midpoint in preparation for a wrap as described by Goldberg and colleagues[1] (Fig. 2). The scar ends were overlapped and sutured to the contralateral healthy sphincter with interrupted absorbable sutures (Fig. 3).

After sphincter-muscle repair, the next stage was to re-establish the anoderm of the anal canal. This was accomplished by S-shaped skin and subcutaneous flaps, approximately 5 cm × 10 cm in dimension, which were created in the lateral quadrants and rotated into the anal canal. They were anchored with absorbable sutures and the lateral subcutaneous spaces drained with a small plastic suction apparatus (Fig. 4).

One week after operation the patient was continent to solid stools and at 6 weeks he was continent to both solid and loose movements, with occasional loss of control of flatus.

Subsequently, he experienced seepage of liquid stools and was operated upon again to remove a persistent mucosal ectropion.

He is now generally continent to solid stools but still has some difficulty with loose stools and flatus, although this occurs fairly infrequently and he is aware of the seepage. Confidence in his continence mechanisms has been restored enabling him to begin an active social life. Despite significant clinical improvement, manometric improvement was minimal — approximately 20 mm Hg higher squeeze pressures, radially.

Discussion

The frequency of anorectal atresia is between 1 in 1500 and 1 in 5000 live births and is more common in males.[2–4] It is usually the result of incomplete separation by the urogenital septum of the cloaca into urogenital and anorectal components. Less frequently it is caused by failure of the anal membrane to perforate normally at the end of 8 weeks' gestation.[2] Murken and Albert[4] found a family history of anorectal atresia in 1 of 169 patients with the abnormality.

The international classification of anorectal anomalies separates these defects into low (translevator), intermediate and high (supralevator), with many subdivisions according to sex and associated fistulas.[5]

Problems commonly associated with anorectal atresia include urinary incontinence and sexual dysfunction which present a major psychosocial problem for the patient. Associated congenital genitourinary and skeletal anomalies are frequent.[6,7] In 1272 children with imperforate anus Hasse[6] found 323 urogenital malformations, 12 of which included horseshoe kidney.

The initial management of complete atresia has been directed at decompressing the obstructed bowel and subsequently producing a socially necessary degree of fecal continence.[8] In incomplete atresias, with an anal dimple, preliminary colostomy may not be necessary. An early dilatation with repeated dilatations by one parent has been a common approach.[5,9–13] Many other approaches have been advocated for higher fistulas, all of which are beyond the scope of this paper.[8,14,15]

The common factor in all forms of treatment is to provide long-term continence. Continence can be defined as the ability to retain intestinal contents until evacuation is desired. It requires the complex interaction of several factors. Schärli[16] has identified five main factors as the passive forces of continence: the motor action of the sphincters, the sensibility of the skin and the sphincters, the function of the rectum and the central nervous pathways, which produce conscious and unconscious coordination. Continence requires the ability to sense the need to defecate and to use the voluntary musculature to redirect stool proximally.[17] A rigid anorectum may impede normal continence. Stelzner[18] noted the importance of the hypersensitive skin of the anal canal in sensing the intestinal contents.

Millard and Rowe[15] identified the absence of a skin-lined anal canal from mucosal ectropion or rectal prolapse as common complications of early repair of imperforate anus, as in our patient. They noted that there is not enough skin available by push or pull to construct an ample skin-lined canal. They advocated skin flaps to eliminate tension at the border of mucosa and skin to correct the problem. They further suggested replacement of insensitive ectropic mucosa with cutaneous sensory epithelium created by local flaps to provide essential perianal sensation. Our experience with gracilis muscle replacement for damaged sphincter muscle has been unrewarding.

Although we performed manometric, electromyographic and radiologic studies, we and others[14] have found them in general to be of less benefit than clinical assessment. The one test of clinical use in our patient was electromyography which identified normal muscle and the extent of scar tissue.

We identified three deficiencies in our patient: a cutaneous sensory defect caused by the mucosal ectropion; anterior deficiency of the anal sphincter, possibly caused by the initial dilatation "missing" the anterior fibres; and anterior scarring of the anal canal, presumably due to repeated dilatations. Our surgical approach was designed to correct each of these deficiencies and was successful in restoring the young man to a near-normal state. Although we could find no similar reports using this combined approach, we speculate that, given the frequency of this problem, other patients exist who might benefit from such an approach.

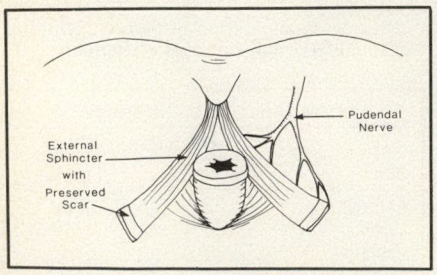

FIG. 2—Type of anterior dissection of external sphincter (adapted from Goldberg SM, Gordon PH, Nivatvongs S[1]).

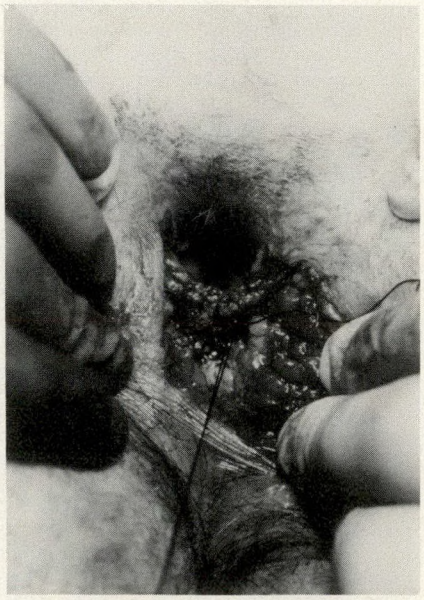

FIG. 3—Completed wrap of external sphincter with suture tags.

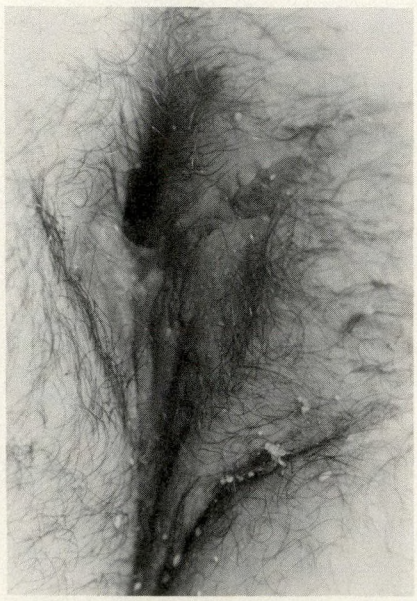

FIG. 4—Completed wrap and graft 2 months postoperatively.

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Do Symptoms Reflect a Change in Left Ventricular Function After Aortocoronary Bypass Grafting in Patients With Depressed Left Ventricular Function?

Seventy-nine patients with moderate to severe left ventricular dysfunction who underwent aortocoronary bypass grafting between 1971 and 1977 had follow-up heart catheterization at a mean interval of 3 years. Thirty-three patients (42%) had angiographic improvement in left ventricular function at follow-up and 18 (25%) had a decrease in left ventricular end-diastolic pressure. Fifty-eight patients (73%) had improvement in angina of at least one New York Heart Association class at follow-up. There was no correlation between late improvement in left ventricular function and improvement in angina. Improvement in left ventricular function did not correlate with preoperative indices of severity of coronary disease or with indices of completeness of surgical repair.

Soixante-dix-neuf patients qui avaient souffert de dysfonctionnement ventriculaire gauche moyen ou sévère et qui ont subi un pontage aortocoronarien entre 1971 et 1977 ont passé un cathétérisme cardiaque de contrôle après un intervalle moyen de 3 ans. Trente-trois patients (42%) montraient au suivi une amélioration angiographique de la fonction ventriculaire gauche et 18 (25%) avaient une diminution de la pression télédiastolique du ventricule gauche. Cinquante-huit patients (73%) présentaient une amélioration de l'angine d'au moins une classe à l'échelle de la New York Heart Association. On n'a constaté aucune corrélation entre une amélioration tardive de la fonction ventriculaire gauche et une amélioration de l'angine. L'amélioration de la fonction ventriculaire gauche n'a pas montré de rapport avec les indices préopératoires de la sévérité de la maladie coronarienne ou avec les indices d'achèvement de la réparation chirurgicale.

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Presented at the 38th annual meeting of the Canadian Cardiovascular Society, Halifax, NS, Oct. 24, 1985

Accepted for publication Jan. 29, 1987

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It has become clear over the past 15 years that symptoms of angina are relieved by aortocoronary bypass grafting for a long time in most patients,[1] but the effect of such surgery on left ventricular function is contentious. Published studies have reported improvement,[2,3] no improvement[4,5] and variable change[6,7] in resting left ventricular function after successful bypass to myocardial segments with reversible ischemia.

One problem in analysing the results of grafting is that latent dysfunction of ischemic segments may be brought out only by stress such as exercise or pacing at study before or after surgery.[5,8] Another is that segments with normal contraction preoperatively cannot be expected to show better resting function postoperatively, but there is the potential for a loss of function due to perioperative infarction. In our own experience, a comparison of segmental wall motion in 50 patients before and after operation showed a decline in resting wall motion in 29 ventricular segments, improvement in 47 and no discernible change in 174. Mean ejection fraction for the group did not differ preoperatively and postoperatively (0.48 versus 0.49).[9] On average, there appears to be modest improvement in ventricular function from aortocoronary bypass grafting alone, but there is considerable individual variation.

We surveyed the results in our hospital of aortocoronary bypass grafting along with aneurysm repair in patients with depressed left ventricular function before operation. This subgroup admittedly carries the greatest potential for a gain in ventricular function after surgery and we first wished to learn how frequently such improvement occurred. We tried to assess whether functional improvement in such patients at follow-up was manifested by better left ventricu-

lar function. Further, we questioned whether improvement in systolic and diastolic left ventricular function necessarily went together. When improvement did occur, we examined whether it could be predicted by, or associated with, certain characteristics of the patient or the operation.

Patients and Methods

Of 1831 patients who underwent aortocoronary bypass grafting at the Ottawa Civic Hospital between 1971 and 1977, 263 were considered to have class III or IV left ventricular dysfunction preoperatively. This classification is a subjective and semiquantitative assessment of dysfunction used by our group that relates to the number of 25 percentiles of normally contracting ventricular circumference at angiography (right anterior oblique projection). A class I ventricle has 75% of the circumference contracting normally and a class II ventricle 50%. Thus, a class III ventricle has only 25% of its circumference with normal inward systolic motion and a class IV ventricle has no entire segment that contracts normally. Although subjective, the system has become fairly reproducible within our group. Where such measurements are available, patients with class III left ventricles usually have ejection fractions between 0.25 and 0.45 and those with class IV ventricles less than 0.25.

This report is a survey of 98 of the 263 patients who returned for a second heart catheterization at least 1 year after surgery. In the first few years, many such patients returned because of recurrent symptoms. After 1978 some were studied in response to an invitation extended to all surviving patients. Complete pre- and postoperative catheterization data were available for 79 of the 98 patients. The interval between surgery and restudy was 2.9 ± 1.4 years (mean ± SD). Comparisons were made of ventricular function preoperatively and postoperatively for each patient. Ventricular angiograms were reviewed by an experienced cardiac radiologist without knowledge of the clinical results. Because left ventricular ejection fractions for both studies were available for only a few patients, systolic function was reported in terms of the above-noted classification. Left ventricular end-diastolic pressure was measured before angiography through fluid-filled catheters, at the nadir of the "a" wave. Change in ventricular function between the two studies and symptomatic improvement at follow-up were correlated, along with a number of preoperative clinical and operative factors (Table I). Tests of statistical significance used were χ^2 analysis, Student's *t*-test and Fisher's exact test.

Findings

Of the 79 patients, 73 (92%) were men and the mean age at the time of surgery was 49 ± 8 years. Fifty patients (63%) had class III left ventricular dysfunction preoperatively and 29 (37%) class IV. No patient had a normal electrocardiogram before surgery; 64 (81%) manifested one or more previous infarctions, and the remainder had left bundle-branch block or ischemic ST abnormalities. Almost 90% of patients had angina but only 18% had symptoms of uncompensated congestive heart failure (Table II). At the time of operation, 17 patients (22%) had plication or resection of a ventricular aneurysm and the mean number of bypass grafts placed was 3.4 ± 1.1 per patient. These figures all closely resemble those of the parent group, suggesting that patients restudied were a reasonably representative sample. The operative (30-day) death rate for the entire group of 263 patients was 8%.

Thirty-three patients (42%) were judged to have shown improvement, between studies, of at least one angiographic class of ventricular contraction (Table III). The remainder did not improve or became worse.

Changes in left ventricular end-diastolic pressure between studies are outlined in Table IV. Although the mean value for the group overall did not change, there were subgroups in which there was a physiologically and statistically significant change in end-diastolic pressure. These groups were (somewhat arbitrarily) defined — one in which the pressure was more than 16 mm Hg preoperatively and fell by at least 6 mm Hg postoperatively (18 patients) and another in which the left ventricular end-diastolic pressure was more than 16 mm Hg postoperatively and at least 6 mm Hg more than the preoperative level (21 patients). These subgroups of patients we have designated as "LVEDP better" and "LVEDP worse" respectively. In the remaining 40 patients there was no significant change.

A univariate analysis of preoperative clinical and operative factors thought likely to have a bearing on improved ventricular function was done for the 33 patients with improvement of one or more angiographic classes of left ventricular function and the 18 "LVEDP better" patients. In no instance did any of these factors have a significant correlation with such improvement ($p > 0.10$).

Improvement in angina at late follow-up was compared with angiographic or hemodynamic improvement in left ventricular function. The incidence of symptoms of uncompensated heart failure before surgery was too low (18%) for a meaningful difference to be present after surgery. Thus, improvement in angina was studied instead. Of the 18 "LVEDP better" patients, 10 (56%) had improvement in angina of one or more NYHA class. However, 16 (76%) of the 21 "LVEDP worse" patients and 32 (80%) of the 40 whose left ventricular end-diastolic pressure did not change significantly had similar degrees of improvement. Similarly, 24 (73%) of the 33 patients whose angiographic systolic left ventricular function improved had anginal improvement while 34 (74%) of the 46 whose left ventricular function was not better had similar improvement at follow-up.

Discussion

Many cardiac centres have avoided aortocoronary bypass grafting in patients

Table I—Univariate Analysis of Hemodynamic or Angiographic Improvement

Preoperative factors
Presence of angina
Presence of congestive heart failure
Angiographic class of left ventricular function
Cardiomegaly
Hypertension
Extent of coronary disease
Use of digoxin
Use of diuretics
Operative factors
Number of grafts
Graft patency at angiographic follow-up
Aneurysm repair

$p > 0.10$ for all factors.

Table II—Preoperative Clinical Status of Patients

NYHA class	Angina, no. (%)	Heart failure, no. (%)
I	9 (11)	65 (82)
II	22 (28)	9 (11)
III	28 (35)	3 (4)
IV	20 (25)	2 (3)

Table III—Change in Angiographic Class of Left Ventricular Function

Class	Preop	Postop
Normal	–	2
Class I	–	1
Class II	–	14
Class III	50	42
Class IV	29	20

Improvement in one or more class = 33 (42%).

Table IV—Change in Left Ventricular End-diastolic Pressure (LVEDP) (mean value ± SD)

Group	Preop, mm Hg	Postop, mm Hg
Total group (n = 79)	15 ± 8	16 ± 8
LVEDP worse (n = 21)	12 ± 7	25 ± 6
LVEDP same (n = 40)	13 ± 7	13 ± 7
LVEDP better (n = 18)	24 ± 6	12 ± 4

with severely depressed left ventricular function, especially those with a left ventricular ejection fraction of less than 0.25 (equivalent to our class IV). The rationale has been that the high operative death rate and high attrition rate over the subsequent few years negate the value of such surgery. This has not been our philosophy at the Ottawa Heart Institute and the acceptable surgical mortality and late survival figures for patients with depressed left ventricular function bear this out. However, the changes in left ventricular function after grafting are much less clear-cut. That 42% of patients had a discernible improvement in angiographic left ventricular function must reflect reversible ischemia as a major component of hypokinesia preoperatively. It is surprising that none of the factors reflecting clinical severity of coronary heart disease were predictive of improvement; it suggests that patients need not be denied surgery on that basis. It is more sobering that improvement in left ventricular function did not relate to those factors that usually express adequacy of surgery — number of grafts performed, number of grafts patent at follow-up and repair of an aneurysm when present. It is possible that the beta error imposed by the number of patients in this study is responsible for the apparent lack of correlation and that results in a much larger study might be different.

Of interest is the discrepancy between rates of improvement in systolic left ventricular function (33 patients) and in diastolic function (18 patients, including 9 of the above 33). As left ventricular end-diastolic pressure reflects not only end-diastolic volume but also end-diastolic chamber compliance, some ventricles must have experienced improved contraction without improvement in "stiffness" upon filling. This may be the consequence of residual scarred myocardium or an absolute reduction in chamber size.

Lastly, it was interesting that 73% of patients had persisting improvement in angina at follow-up regardless of whether left ventricular function had observably improved, even when severely depressed. In particular, anginal improvement was equally likely to be present when the resting left ventricular end-diastolic pressure was higher after surgery than when it was lower. This attests to the complexity of reasons for improvement in symptoms and, most importantly, implies that relief of angina following surgery in such patients does not necessarily indicate improvement in left ventricular function.

Clearly, aortocoronary bypass grafting can relieve angina in a majority of patients with depressed left ventricular function. Improvement in left ventricular function per se is variable and not readily predictable; it cannot be inferred from improvement in angina alone.

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BOOK REVIEWS

ACUTE PERIPHERAL VASCULAR SURGERY. Michael Staudacher. 165 pp. Illust. Springer-Verlag New York, Inc., New York, 1985. $38.50 (US). ISBN 0-387-81874-X.

This monograph is designed for the general surgeon who may occasionally be required to provide emergency care for the patient with acute vascular problems.

The contents are well organized and the procedures well illustrated with coloured schematic diagrams. A very brief description is given of the pathologic features of some vascular emergencies including arterial embolism, arterial thrombosis, venous thrombosis and the tourniquet syndrome. The descriptions of aneurysms and trauma are extremely brief.

Emergency instruments required for simple vascular repairs are adequately listed, but the description of the use of air in the Fogarty catheter will not be acceptable to most vascular surgeons.

The basic vascular surgical techniques are nicely illustrated, although the use of two stay sutures instead of the standard three triangulation stay sutures for arterial end-to-end anastomosis is not generally practised. There are specific illustrations for the management of embolectomy, simple arterial puncture wound, severe femoral artery and vein injury and false aneurysm. Venous thrombectomy is described, but I do not think this is often indicated and should not be done by the occasional operator.

Repair of the popliteal aneurysm is clearly described but oversimplified. Resectional therapy is now seldom used. Iatrogenic vascular injuries are identified in hernia repair, mastectomy and pelvic surgery. Although the management is illustrated briefly, the prevention of these injuries is not addressed.

For the occasional vascular surgeon, it seems inappropriate to include a description of the extra-anatomical repair for an infected vascular wound in the groin.

The bibliography is limited to some German and American textbooks.

This monograph may be of value to the student or junior resident as an introduction to vascular emergencies. The description of the basic vascular techniques of repairing arteries and veins will be of value to the general surgeon, but I think the operative descriptions of the emergency procedures lack the detail necessary for the general surgeon who may be called upon to treat patients with these problems.

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COLORECTAL CANCER. Current Concepts in Diagnosis and Treatment. Edited by Glenn Steele, Jr. and Robert T. Osteen. 366 pp. Illust. Marcel Dekker, Inc., New York, 1986. $59.59 (US). ISBN 0-8247-7372-1.

COLORECTAL TUMORS. Edited by Oliver H. Beahrs, George A. Higgins and Jacob J. Weinstein. 334 pp. Illust. J.B. Lippincott Company, Philadelphia, 1986. $57.50 (US). ISBN 0-397-50677-5.

Approximately 138 000 new cases of colorectal cancer were diagnosed in the United States in 1985. About half this number of people died of the disease in the same year. These statistics establish colorectal cancer as the second most common cause of cancer-related deaths, and certainly a worthy subject for a comprehensive review in textbook form. Interestingly, two separate books on the subject, each with a quite different approach, have appeared on the market at the same time. Since even the most dedicated physician is unlikely to require or desire both books, a review of the individual and relative merits of the two would seem appropriate.

Beahrs and colleagues in their book *Colorectal Tumors* have chosen a multiple-author format, with 42 contributors writing on their own areas of expertise, I gather, within the frame-

continued on page 308

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(Warfarin sodium) ANTICOAGULANT

INDICATIONS: The prophylaxis and/or treatment of venous thrombosis and its extension, pulmonary embolism, atrial fibrillation with embolization, and as an adjunct in the prophylaxis of systemic embolism after myocardial infarction. Some of the more common clinical disorders which may be associated with or predispose patients to the above indications are: Thrombophlebitis; Congestive heart failure; Surgical procedure or trauma associated with a high risk of thromboembolism; Myocardial infarction; Cerebral embolism. It may also be useful as an adjunct in the treatment of transient cerebral ischemic attacks due to intravascular clotting.

CONTRAINDICATIONS: Any localized or general physical condition or personal circumstance in which the hazard of hemorrhage might be greater than the potential clinical benefits of anticoagulation, such as: **Pregnancy:** COUMADIN[5] is contraindicated in pregnancy because the drug passes through the placental barrier and may cause fatal hemorrhage to the fetus *in utero.* Also, there have been reports of birth malformations in children born to mothers who have been treated with warfarin during pregnancy. Women of childbearing potential who are candidates for therapy should be carefully evaluated and the indications critically reviewed with the patient. If the patient becomes pregnant while taking this drug, she should be apprised of the potential risks to the fetus, and the possibility of termination of the pregnancy should be discussed in light of those risks. **Hemorrhagic tendencies or blood dyscrasias. Recent or contemplated surgery of:** 1. central nervous system; 2. eye; 3. traumatic surgery resulting in large open surfaces. **Bleeding tendencies associated with active ulceration or overt bleeding of:** gastrointestinal, genitourinary or respiratory tracts; cerebrovascular hemorrhage; aneurysms – cerebral, dissecting aorta; pericarditis and pericardial effusions; bacterial endocarditis. **Threatened abortion,** eclampsia and preeclampsia. **Inadequate laboratory facilities** or unsupervised senility, alcoholism, psychosis, or lack of patient cooperation. **Spinal puncture** and other diagnostic or therapeutic procedures with potential for uncontrollable bleeding. **Miscellaneous:** major regional, lumbar block anesthesia and malignant hypertension.

WARNINGS: The most serious risks are hemorrhage in any tissue or organ and, less frequently, necrosis and/or gangrene of skin and other tissues. Risk of hemorrhage is related to the level of intensity and duration of therapy. Hemorrhage and necrosis have in some cases been reported to result in death or permanent disability. Necrosis appears to be associated with local thrombosis and usually appears within a few days of the start of therapy. In severe cases of necrosis, treatment through debridement or amputation of the affected tissue, limb, breast or penis has been reported. Careful diagnosis is required to determine whether necrosis is caused by an underlying disease. Therapy should be discontinued when warfarin is the suspected cause of developing necrosis and heparin may be considered. Although various ones have been attempted, no treatment for necrosis has been considered uniformly effective. See below for information on predisposing conditions. These and other risks must be weighed against the risk of thrombosis or embolization in untreated cases. COUMADIN is a potent drug with a half-life of 2.5 days; therefore effects may become more pronounced as daily maintenance doses overlap. **It cannot be emphasized too strongly that treatment of each patient is a highly individualized matter.** Dosage should be controlled by periodic determinations of prothrombin time or other suitable coagulation tests. Determinations of whole blood clotting and bleeding times are not effective measures for control of therapy. Heparin prolongs the one-stage prothrombin time. When heparin and COUMADIN are administered concomitantly, refer to CONVERSION FROM HEPARIN THERAPY for recommendations. Caution should be observed when COUMADIN is administered in any situation or in the presence of any predisposing condition where added risk of hemorrhage or necrosis is present. Administration of anticoagulants in the following conditions will be based upon clinical judgement in which risks of therapy are weighed against the risk of thrombosis or embolization in untreated cases. The following may be associated with these increased risks: **Lactation. Severe to moderate hepatic or renal insufficiency. Infectious diseases or disturbances of intestinal flora** – sprue, antibiotic therapy. **Trauma** which may result in internal bleeding. **Surgery or trauma** resulting in large exposed raw surfaces. **Indwelling catheters. Severe to moderate hypertension. Known or suspected deficiency in protein C:** This hereditary or acquired condition, which should be suspected if there is a history of recurrent episodes of thromboembolic disorders in the patient or in the family, has been associated with an increased risk of developing necrosis following warfarin administration. Tissue necrosis may occur in the absence of protein C deficiency. It has been reported that concurrent anticoagulation therapy with heparin for 5 to 7 days during initiation of therapy with COUMADIN may minimize the incidence of this reaction. Warfarin therapy should be discontinued when it is suspected to be the cause of developing necrosis, and heparin may be considered. **Miscellaneous:** polycythemia vera, vasculitis, severe diabetes, severe allergic and anaphylactic disorders. Patients with congestive heart failure may become more sensitive to COUMADIN, thereby requiring more frequent laboratory monitoring, and reduced doses. Concurrent use of anticoagulants with streptokinase or urokinase is not recommended and may be hazardous. (Please note recommendations accompanying these preparations.)

PRECAUTIONS: Periodic determination of prothrombin time or other suitable coagulation test is essential. Numerous factors, alone or in combination, including travel, changes in diet, environment, physical state and medication may influence response to anticoagulants. It is good practice to monitor the response with additional prothrombin time determinations in the period immediately after discharge from the hospital, and whenever other medications are initiated, discontinued or taken haphazardly. The following factors are listed for your reference; however, other factors may also affect the anticoagulant response.

The following factors, alone or in combination, may be responsible for INCREASED prothrombin time response: ENDOGENOUS FACTORS: cancer, collagen disease, congestive heart failure, diarrhea, elevated temperature, hepatic disorders: (infectious hepatitis, jaundice), poor nutritional state, steatorrhea, vitamin K deficiency. EXOGENOUS FACTORS: alcohol,† allopurinol, aminosalicylic acid, amiodarone HCl, anabolic steroids, anesthetics, inhalation, antibiotics, bromelains, chloral hydrate,† chlorpropamide, chymotrypsin, cimetidine, clofibrate, COUMADIN overdosage, dextran, dextrothyroxine, diazoxide, diflunisal, diuretics,† disulfiram, ethacrynic acid, fenoprofen, glucagon, hepatotoxic drugs, ibuprofen, influenza virus vaccine, mefenamic acid, methyldopa, methylphenidate, metronidazole, miconazole, monoamine oxidase inhibitors, nalidixic acid, naproxen, narcotics, prolonged, pentoxifylline, phenylbutazone, phenytoin, pyrazolones, quinidine, quinine, ranitidine,† salicylates, sulfinpyrazone, sulfonamides, long-acting, sulindac, thyroid drugs, tolbutamide, trimethoprim/sulfamethoxazole, other medications affecting blood elements which may modify hemostasis. Also: dietary deficiencies, prolonged hot weather, unreliable prothrombin time determinations.

The following factors, alone or in combination, may be responsible for DECREASED prothrombin time response: ENDOGENOUS FACTORS: edema, hereditary coumarin resistance, hyperlipemia, hypothyroidism. EXOGENOUS FACTORS: adrenocortical steroids, alcohol,† antacids, antihistamines, barbiturates, carbamazepine, chloral hydrate,† chlordiazepoxide, cholestyramine, COUMADIN underdosage, diuretics,† ethchlorvynol, glutethimide, griseofulvin, haloperidol, meprobamate, oral contraceptives, paraldehyde, primidone, ranitidine,† rifampin, vitamin C. Also: diet high in vitamin K, unreliable PT determinations. Because a patient may be exposed to a combination of the above factors, the net effect on PT response may be unpredictable. More frequent laboratory monitoring is, therefore, advisable. Coumarins may also affect the actions of other drugs. Hypoglycemic agents (chlorpropamide, tolbutamide and glyburide) and anticonvulsants (phenytoin and phenobarbital) may accumulate in the body as a result of interference with either their metabolism or excretion.

ADVERSE REACTIONS: Potential adverse reactions may include: • Hemorrhage from any tissue or organ. This is a consequence of the anticoagulant effect. Signs and symptoms will vary according to the location and degree or extent of bleeding. Therefore, the possibility of hemorrhage should be considered in evaluating the condition of any anticoagulated patient with complaints which do not indicate an obvious diagnosis. Bleeding during anticoagulant therapy does not always correlate with prothrombin activity. (See SYMPTOMS AND TREATMENT OF OVERDOSAGE.) Bleeding which occurs when the prothrombin time is within the therapeutic range warrants diagnostic investigation, since it may unmask a previously unsuspected lesion, e.g. tumor, ulcer, etc. • Necrosis of skin and other tissues. (SEE WARNINGS). • Other adverse reactions are infrequent and consist of alopecia, urticaria, dermatitis, fever, nausea, diarrhea, abdominal cramping, a syndrome called "purple toes," cholestatic hepatic injury, and hypersensitivity reactions. • Priapism has been associated with anticoagulant administration, however, a causal relationship has not been established.

DOSAGE AND LABORATORY CONTROL: Administration: Administration and dosage must be individualized. Adjust the dosage according to results of the one-stage prothrombin time (PT) with commonly used rabbit brain thromboplastin. There is ample evidence that prolongation of the prothrombin time 1.2 to 1.5 times control is sufficient for prophylaxis and treatment of venous thromboembolism, minimizing the risk of hemorrhage associated with more prolonged PT values. Where the risk of thromboembolism is great, such as with recurrent systemic embolism, a PT of 1.5 to 2.0 times control should be maintained. A ratio greater than 2.0 appears to provide no additional therapeutic benefit in most patients and is associated with a higher risk of bleeding.

Table of Recommended Therapeutic PT Ranges*

Clinical State	Rabbit Brain PT Ratio**
Prophylaxis – venous thromboembolism	
High-risk surgery	1.2-1.4
Hip surgery	1.3-1.5
Treatment-deep vein thrombosis or pulmonary embolism	1.2-1.5
Prevention of systemic embolism in patients with:	
• atrial fibrillation,	
• tissue heart valves, or	
• acute myocardial infarction	1.2-1.5
Recurrent systemic embolism	1.5-2.0

*Modified from Chest, ACCP-NHLBI National Conference on Antithrombotic Therapy, volume 89, number 2, page 14S, 1986.

**For the three thromboplastins currently used in North America, a PT with rabbit brain thromboplastin of 1.3 to 2.0 is equivalent to an International Normalized Ratio (INR) of 2.0 to 4.0. For other thromboplastins the INR can be calculated as: INR = (observed PT).[ISI] The ISI (International Sensitivity Index) is available from the manufacturers of thromboplastin.

INITIAL DOSAGE: Is commonly started above anticipated maintenance dosage levels. A commonly-used regimen is 10mg/day for 2 to 4 days, with daily adjustments based on the results of PT determinations. A large loading dose (i.e., 30mg) may increase the incidence of hemorrhagic and other complications, does not offer more rapid protection against thrombi formation, and is not recommended. Lower doses are recommended for elderly and/or debilitated patients and patients with increased sensitivity (see PRECAUTIONS). **Maintenance:** Most patients are satisfactorily maintained at a dose of 2 to 10mg daily. Flexibility of dosage is provided by breaking scored tablets in half. The individual dose and interval should be gauged by the patient's prothrombin response. **Duration of therapy:** The duration of therapy in each patient should be individualized. In general, therapy should be continued until the danger of thrombosis and embolism has passed. **Treatment During Dentistry and Surgery:** Management of patients who undergo dental and surgical procedures requires close liaison between attending physicians, surgeons and dentists. In patients who must be anticoagulated prior to, during, or immediately following dental or surgical procedures, adjusting the dosage to maintain the PT at the low end of the therapeutic range, may safely allow for continued anticoagulation. The operative site should be sufficiently limited and accessible to permit the effective use of local procedures for hemostasis. Under these conditions, dental and surgical procedures may be performed without undue risk of hemorrhage. **Conversion from Heparin Therapy:** Since the onset of COUMADIN's effect is delayed, heparin is preferred initially for rapid anticoagulation. Conversion to COUMADIN may begin concomitantly with heparin therapy or may be delayed 3 to 6 days. As heparin may affect the PT, patients receiving both heparin and COUMADIN should have blood drawn for PT determination, at least: • 5 hours after the last IV bolus dose of heparin, or • 4 hours after cessation of a continuous IV infusion of heparin, or • 24 hours after the last subcutaneous heparin injection. When COUMADIN has produced the desired therapeutic range or prothrombin activity, heparin may be discontinued.

SYMPTOMS AND TREATMENT OF OVERDOSAGE: Symptoms: Suspected or overt abnormal bleeding (i.e., appearance of blood in stools or urine, hematuria, excessive menstrual bleeding, melena, petechiae, excessive bruising or persistent oozing from superficial injuries) are early manifestations of anticoagulation beyond a safe and satisfactory level. **Treatment:** Excessive anticoagulation, with or without bleeding, may be controlled by discontinuing therapy and if necessary, by administration of oral or parenteral vitamin K_1 2.5mg to 10mg. (Please see recommendations accompanying vitamin K_1 preparations prior to use.) Use of vitamin K_1 reduces responses to subsequent COUMADIN therapy. Patients may return to a pretreatment thrombotic status following the rapid reversal of a prolonged PT. Resumption of COUMADIN administration reverses the effect of vitamin K_1, and a therapeutic PT can again be obtained by careful dosage adjustment. If rapid anticoagulation is indicated, heparin may be preferable for initial therapy. If minor bleeding progresses to major bleeding, give 5 to 25mg (rarely up to 50mg) parenteral vitamin K_1. In emergency situations of severe hemorrhage, clotting factors can be returned to normal by administering 200 to 500mL of whole blood or fresh frozen plasma, or by giving commercial Factor IX complex. Packed red blood cells may also be given if significant blood loss has occurred. Infusions of blood or plasma should be monitored carefully to avoid precipitating pulmonary edema in elderly patients or patients with heart disease.

DOSAGE FORMS: COUMADIN TABLETS are single-scored, and are imprinted as follows:

SIZE:	2mg	2.5mg	5mg	10mg
CODE:	0101	0201	0301	0401
COLOR:	lavender	orange	peach	white
IMPRINT:				
Side 1:	COUMADIN	COUMADIN	COUMADIN	COUMADIN
	bisected 2	bisected 2½	bisected 5	bisected 10
Side 2:	Du Pont	Du Pont	Du Pont	Du Pont

Supplied in bottles of 100.

†Increased and decreased prothrombin time responses have been reported.

Reference:

1. O'Reilly RA, Aggeler PM: Studies on coumarin anticoagulant drugs: initiation of warfarin therapy without a loading dose. Circulation 38:169-177, 1968.

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Bacterial Colonization of Intestinal Urinary Conduit Diversion: a Morphologic and Bacteriologic Experimental Study

Cutaneous diversion of the urine through intestinal conduits results in bacteriuria with uropathogenic organisms in up to 80% of patients, many of whom suffer pyelonephritis. Analysis of the bacteriologic data from daily sampling at multiple sites and scanning and transmission electron microscopic studies of sequential loop and autopsy specimens in rabbits with functioning colonic conduits indicate that the pyelonephritis evolves in sequential stages of microbial colonization. Using these data on the natural progression of bacterial infection, we can rationally test the ability of strains of bacteria indigenous to the conduit of this animal model to prevent uropathogenic colonization of the conduit.

La dérivation cutanée de l'urine dans les canaux intestinaux résulte en une bactériurie avec organismes uropathogéniques pour un pourcentage de malades allant jusqu'à 80%; de plus beaucoup de malades sont ensuite atteints de pyélonéphrite. Les analyses des données bactériologiques de prélèvements quotidiens provenant d'emplacements multiples, ainsi que des études utilisant microscopes électroniques à balayage et à transmission avec de spécimens d'anses successives et de spécimens d'autopsie chez des lapins ayant des canaux du côlon en bon état, indiquent que l'évolution de la pyélonéphrite se fait par étages successives de colonisation microbienne progressive. Si l'on se sert de ces données sur la progression naturelle des étages successives de l'infection bactérienne, il est possible de faire des essais rationnels de l'usage de l'interférence bactérienne en utilisant des types de bactéries indigènes stabilisées dans le canal intestinal de ce modèle animal pour arrêter les pathogènes potentiels.

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Accepted for publication Jan. 27, 1987

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Cutaneous diversion of the urine is used to treat a variety of conditions that require surgical excision of the lower urinary tract. The ileal loop, which uses an ileal segment into which both ureters are implanted for draining urine to the abdominal wall,[1] has been used extensively for the past 30 years. However, the operative procedure results in later development of infection; 80% of patients are reported to have bacteriuria caused by uropathogens.[2-6] Acute and chronic pyelonephritis is common among the patients because of reflux of infected urine into the kidney resulting in progressive renal deterioration.[4,7,8] Colonic conduits incorporating a nonrefluxing ureterocolic anastomosis have recently become popular. Long-term follow-up of patients having a colonic conduit revealed that there is still a high incidence of pyelonephritis and renal deterioriation.[9] To reduce this incidence, antibiotics have been given preoperatively and postoperatively, sometimes resulting in infection with yeast and resistant bacteria.[2-6,10] Because of widespread clinical use of antibiotics, the natural history and progression of the bacterial populations associated with the intestinal urinary conduit is unknown.

Rozee and colleagues[11] have shown that the bacteria associated with the intestinal surface are trapped within the mucus blanket. In a recent study, Bruce and colleagues[10] showed virtually no bacteria adhering to the columnar cells of an intestinal urinary conduit, even though the conduit mucus was heavily colonized with bacteria. It was suggested that use of indigenous bacteria to colonize the mucus biofilm of intestinal conduits might prevent uropathogenic colonization. Before such a concept can be tested, a model must be developed to study the natural microbiologic progression of urinary conduit infections, and in which the protective effect of beneficial bacteria introduced into the conduit can be assessed. In this investigation we developed an animal model for colonic conduit urinary diversion and undertook detailed microbiologic, ultrastructural and morphologic studies of the model to determine the etiology and natural progression of urinary conduit infections from the development of bacteriuria in the sterile conduit to the appearance of destructive pyelonephritis.

Material and Methods

Operative Procedure

After partial cystectomy colonic conduits were created in 10 young, female, New Zealand rabbits, each weighing 4 to 5 kg, using a modification of the trigonal patch technique of Pond and Texter.[12] The animals were starved for 24 hours preoperatively and received saline infusions preoperatively, intraoperatively and for 36 hours postoperatively. A single dose of veterinary penicillin-streptomycin was administered parenterally 12 hours before the operation. The animals were anesthetized with ketamine and the abdomen was opened through a midline incision. A 3 to 5-cm segment of descending and sigmoid colon was mobilized and isolated by sharply transecting the proximal and distal margins, being careful to preserve the mesenteric blood supply. Continuity of the colon was re-established by a one-layer anastomosis with interrupted 4-0 chromic catgut sutures. The bladder, along with the ureters and the inferior vesical blood supply, was carefully dissected free. A subtotal cystectomy was performed, preserving the ureteral insertions into the trigone. The proximal urethra was oversewn with 4-0 chromic catgut. The isolated segment of colon was irrigated, first with a dilute piperacillin solution and then copiously by phosphate-buffered saline. The proximal end was spatulated and anastomosed to the trigonal patch with a running 4-0 catgut suture. A circular plug of abdominal wall was excised at the preselected stomal site and a nippled colonic conduit stoma constructed. After abdominal closure in

layers, the rabbits were placed in incubators with supplemental oxygen, increased humidity and a temperature of 29°C for 12 hours, after which they were returned to their cages. They received fluids parenterally for 36 hours postoperatively, and then a regular diet.

Specimen Sampling and Preparation

Catheterized specimens of urine (with or without mucus) were taken daily from the stoma (distal 1 cm of conduit) and deep conduit (more than 1 cm proximal to stoma). Aliquots (0.1 ml) of urine were spread plated on brain-heart infusion agar and sheep-blood agar plates and incubated aerobically and anaerobically for 24 to 48 hours. Bacteria were quantitated, isolated and identified using standard techniques. Bacterial counts greater than 10^4/ml were considered significant.[6] Mucus specimens extracted aseptically from the deep conduit were cultured in a similar fashion and were also prepared for transmission electron microscopy as described below.

All animals eventually became ill, stopped eating and drinking, became lethargic and had some degree of flank tenderness. With these symptoms a clinical diagnosis of pyelonephritis was made and the rabbit was killed. At autopsy, urine was aseptically aspirated from the proximal conduit and the renal pelves of both kidneys and was cultured. Specimens were taken from the stoma, proximal and distal conduit, conduit mucus, ureters and kidneys for scanning and transmission electron microscopy. Renal cortical tissue was sectioned and bisected; one portion was homogenized and cultured and the other stained with hematoxylin and eosin for histologic examination.

The specimens collected for scanning electron microscopy were fixed in 5% glutaraldehyde in cacodylate buffer (0.1 M, ph 7.0) with 0.15% ruthenium red for 2 hours at room temperature (22°C). The preparations were washed three times in the buffer and then "metallized", using osmium tetroxide and thiocarbohydrazide.[13] This was followed by dehydration in ethanol and freon-113 before critical point drying.[14] The specimens were then examined with a Hitachi S450 scanning electron microscope at an accelerating voltage of 20 kV.

The specimens collected for transmission electron microscopy were fixed as for scanning electron microscopy. They were then washed five times in buffer, postfixed in 2% osmium tetroxide in buffer and dehydrated through a series of acetone washes. All of the solutions used in processing the specimen, from the washes after gluteraldehyde fixation to dehydration with 70% acetone solution, contained 0.05% ruthenium red. After further dehydration in propylene oxide, the

Table I—Postoperative Time Elapsed Until Bacterial Colonization (10^4 bacteria/ml) of Stomal and Deep Conduit Urine and the Clinical Occurrence of Pyelonephritis

	Time, d		
Rabbit no.	Stoma	Deep Conduit	Pyelonephritis*
4	15	22	24
5	2	7	11
6	6	13	20
7	4	5	8
8	3	5	14
9	2	4	5
10	4	6	10
Mean ± SD	5.1 ± 4.6	8.9 ± 6.5	13.1 ± 6.7

*Clinical impression confirmed by bacteriologic autopsy studies.

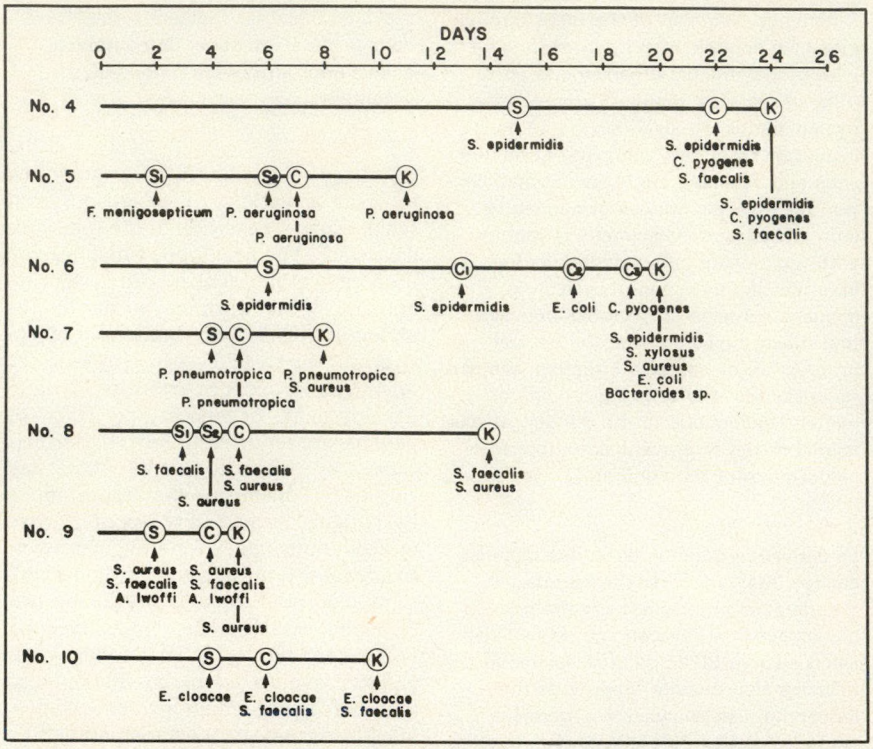

FIG. 1—Bacteriologic data from stoma (S), deep conduit (C) and kidney (K) in seven rabbits after colonic conduit urinary diversion. Identification of organisms demonstrates sequential microbiologic colonization after operation, starting with stomal area and progressing to deep conduit and finally to kidney.

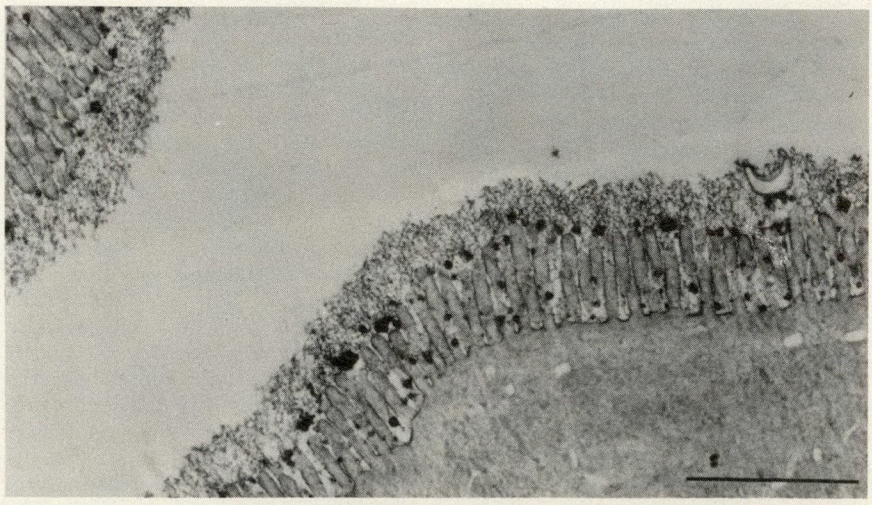

FIG. 2—Rabbit no. 7. Transmission electron photomicrograph of proximal portion of intestinal conduit 7 days after operation. Note absence of bacterial colonization of both microvillar surface of colon and overlying mucus layer. Bars on all photomicrographs indicate 1 μm.

specimen was embedded in Spurr[15] low-viscosity embedding resin. Sections were cut and stained with uranyl acetate and lead citrate,[16] reinforced with evaporated carbon and examined with a Hitachi 6000 transmission electron microscope at an accelerating voltage of 60 kV.

Results

The postoperative death rate was 30% — one animal died as a result of the anesthetic, another of pneumonia and the third of a pelvic abscess. In the remaining seven rabbits a definite pattern of microbiologic colonization and infection was demonstrated. In all, there was a progression from stomal colonization to deep conduit infection, followed by pyelonephritis. Table I shows the postoperative time lapse until demonstrable colonization (greater than 10^4 bacteria/ml) of stomal and deep conduit urine and clinical occurrence of pyelonephritis. The stomal urine was colonized by more than 10^4 bacteria/ml in a mean time ($\pm$ SD) of 5.1 $\pm$ 4.6 days, deep conduit urine infection followed 3.7 $\pm$ 2.6 days later (range from 1 to 7 days) and clinical evidence of pyelonephritis was confirmed a mean of 4.3 $\pm$ 2.8 days (range from 1 to 9 days) after that. The clinical impression of pyelonephritis was confirmed in all cases by autopsy cultures of renal pelvis urine and homogenized cortical tissue.

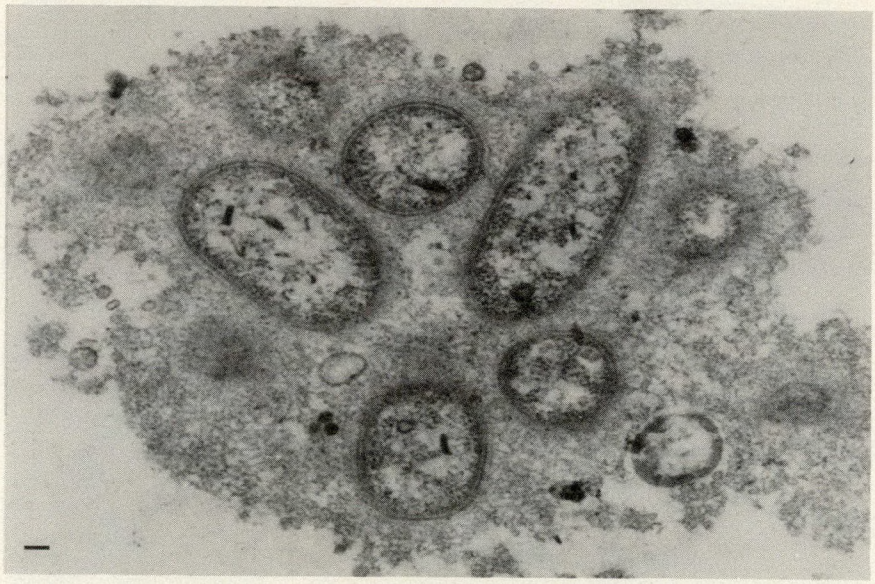

FIG. 3—Rabbit no. 4. Transmission electron photomicrograph of bacterial microcolony in mucus at site of stoma 16 days after surgery. Although cultures grew *Staphylococcus epidermidis*, and many gram-positive cocci were seen, microcolonies of this gram-negative organism were commonly found in mucus. Ruthenium-red staining has revealed fine fibrous glycocalyx material surrounding these cells which also have thin electron-dense concentric element outside outer membrane of their cell walls.

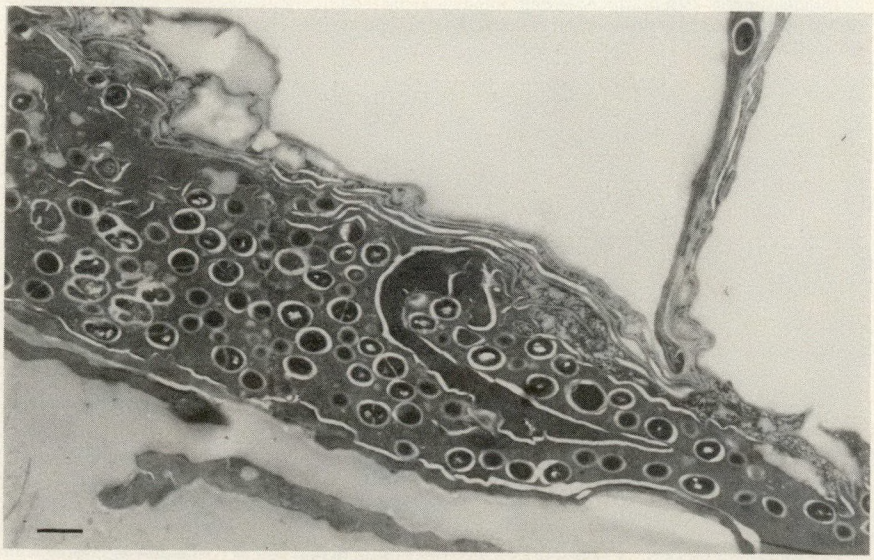

FIG. 4—Rabbit no. 4. Transmission electron photomicrograph of epidermal cells from stoma 16 days after surgery. Cultures grew *S. epidermidis* and direct microscopy showed that spaces between cells in this squamous epithelium were filled by gram-positive cocci growing in coherent glycocalyx-enclosed biofilm.

Identification of the organisms isolated from the urine specimens showed that in all animals bacteriuria developed with one or more of the following organisms: *Staphylococcus epidermidis, Staphylococcus aureus, Staphylococcus xylosus, Streptococcus faecalis, Pseudomonas aeruginosa, Corynebacterium pyogenes, Escherichia coli, Pasteurella pneumotropica, Acinetobacter lwoffi, Enterobacter cloacae, Flavobacterium meningosepticum* and *Bacteroides* sp. (Fig. 1). Analysis of the progressive data (Fig. 1) showed that organisms present in the stomal urine in the first stages of microbial colonization were usually involved in the further stages of conduit infection and pyelonephritis. In most animals polymicrobial populations developed in the later stages.

Scanning and transmission electron microscopic studies of sequential mucus specimens and autopsy biopsies of the conduits and kidneys again demonstrated the various stages of microbiologic colonization. For several days after the operation no microorganisms were found in the intestinal tissues or the mucus blanket (Fig. 2), but within a period of 5 $\pm$ 4.6 days, the stomal area, including the mucus (Fig. 3) and cells from the mucocutaneous junction (Fig. 4), was heavily colonized by glycocalyx-enclosed microcolonies of bacteria. Within days the deep-conduit mucus and urine became thickly colonized with a similar mucus-associated population of bacteria, including both gram-positive and gram-negative bacteria (Fig. 5). There was virtually no bacterial attachment to the microvillar surface of the columnar cells of the conduit (Fig. 6); however, bacterial colonization of the nearby mucus blanket was clearly demonstrated (Fig. 7). In all the animals, pyelonephritis (proven at autopsy) eventually developed. The invasive and destructive nature of the renal infection was confirmed by positive cultures of homogenized cortical tissue and histologic sections of the renal parenchyma.

Discussion

The development of pyelonephritis with renal deterioration is the major long-term complication of intestinal conduit urinary diversion.[2-10] Our data indicate that the evolution of pyelonephritis follows sequential stages of progressive microbial colonization. Entry of bacteria into the conduit undoubtedly occurs when pathogenic bacteria adhere to the mucocutaneous junction of the stoma, and this is followed quickly by colonization of the mucus and urine of the distal stomal zone. It took approximately 4 days in our animal model for the mucus and urine layer of the deep conduit to become

colonized with a polymicrobial population of pathogenic bacteria. It is likely that bacteria gain access to the kidneys through reflux of the infected urine from the conduit because reflux is present in this reconstructed urinary system.

It was interesting to note that a deep conduit urine infection did not develop immediately after colonization of the mucus and urine of the stomal zone of the conduit. The bacteria in the distal conduit were all present in the mucus/urine layer and none were seen attached to the intestinal cells. It would appear that the mucus produced by the goblet cells of the colonic conduit forms a dense flowing biofilm that entraps invading organisms and continuously forces them toward the stoma and out of the conduit. The mucus blanket might also be involved in preventing bacterial attachment to the conduit cell surface by pathogenic organisms. High levels of secretory immunoglobulin A within the conduit urine may also constitute a host-defence mechanism against urinary tract infection in patients with conduits.[17] However, this proposed defence mechanism is not totally effective, and pathogenic organisms eventually invade and reside in the mucus layer of the deep conduit. The presence of these pathogenic organisms in the proximal conduit exposes the animals to the risk of upper tract infection, which eventually develops in those subjected to this surgical modification.

Clinical studies[2-10] appear to confirm our morphologic and bacteriologic investigation, although progression through the described stages is, of course, not documented. In most patients who undergo urinary diversion, bacteriuria develops within the conduit and this appears to precede the eventual development of renal infection. The conduit mucus recovered from patients with ileal conduit bacteriuria also appears to be heavily colonized by uropathogens.[10] It has been suggested[10] that the establishment of a nonpathogen (normal flora) within the urinary conduit may competitively exclude pathogenic organisms from the conduit. Bacterial interference using indigenous strains of bacteria to inhibit potential pathogens has been successful in the intestines of human neonates,[18] in the bovine udder,[19] in the digestive tracts of newborn ruminants (unpublished data) and in the human vagina.[20]

In this investigation, the natural progression of the sequential stages of bacterial infection in the colon urinary conduit has been established in an animal model. We are planning to use this model in an attempt to stabilize indigenous bacteria such as *Lactobacillus* sp. in the mucus biofilm of the urinary conduit. In turn, we hope this may prevent subsequent stages of uropathogenic colonization.

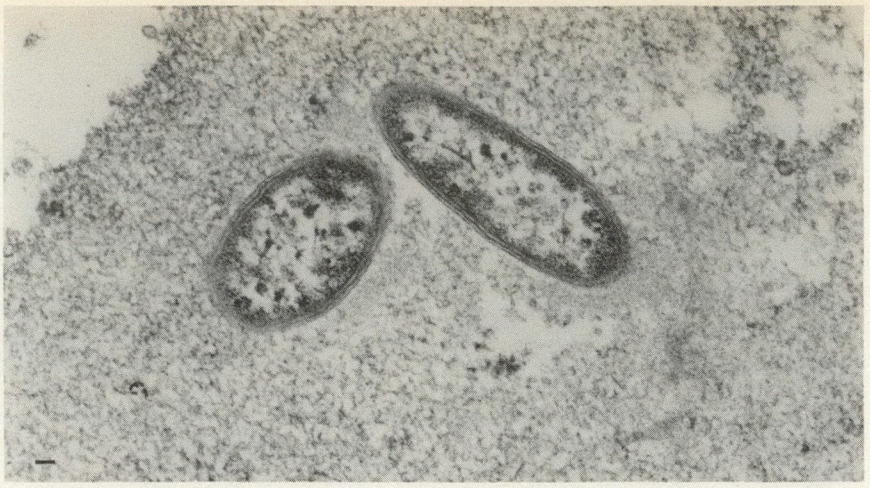

FIG. 5—Rabbit no. 6. Transmission electron photomicrograph of mucus from deep (proximal) area of colonic conduit 20 days after surgery. Both gram-positive and gram-negative bacteria were seen in mucus from this site, which grew wide variety of bacteria on culture. Gram-negative rod-shaped cells are embedded in fine glycocalyx fibres and coarse fibres of dehydration-condensed mucus.

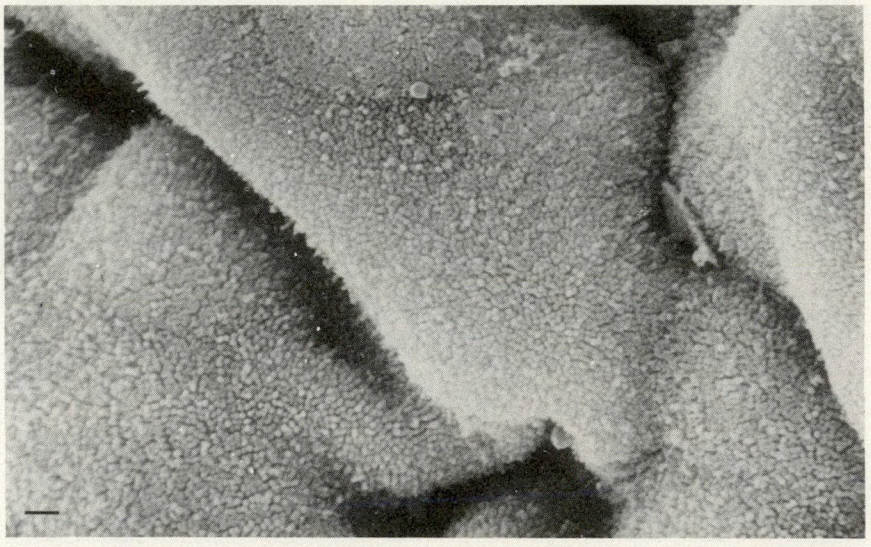

FIG. 6—Scanning electron photomicrograph of intestinal tissue from same proximal site as mucus sample seen in Fig. 5. When mucus is removed during processing, extensive examination of tissue surface shows very few bacteria and tissue appears normal in all morphologic respects.

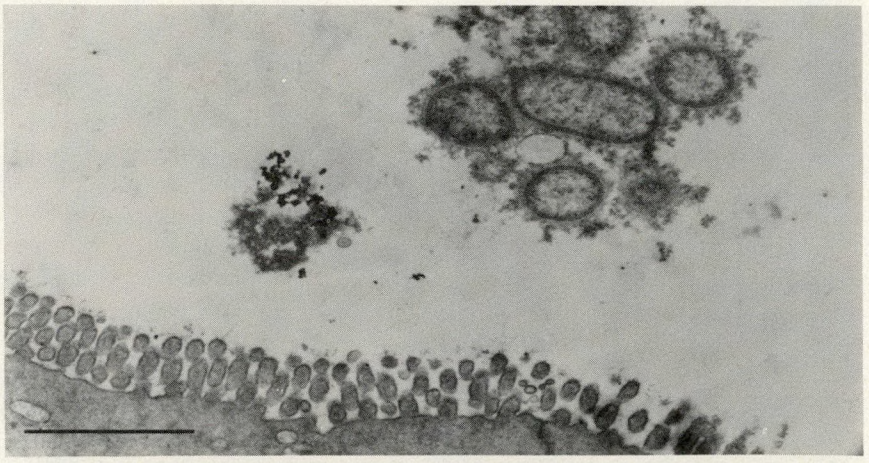

FIG. 7—Rabbit no. 7. Transmission electron photomicrograph of tissue-mucus sample from deep (proximal) site in ileal conduit. Deep portion of this conduit was rapidly colonized 8 days after surgery and this sample grew both *Pasteurella pneumotropica* and *Staphylococcus aureus* on culture. Microcolonies of both gram-positive and gram-negative (seen here) bacteria were seen in mucus layer but tissue surface was not colonized.

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DAVID F. DEXTER, MD, FRCPC, FCAP‡

Systemic Cystic Angiomatosis in a Woman With Hematuria and Splenomegaly

Cutaneous hemangiomas are frequently found on routine clinical examination and generally are not important. But when internal organs are involved, these vascular tumours assume greater importance because of associated morbidity. A case is presented of multiple organ involvement by angiomas in a woman with a history of flank hemangioma who was investigated for hematuria and splenomegaly. She subsequently underwent splenectomy. The uncommon systemic syndromes characterized by vascular tumours are discussed, and the case reported is appropriately classified. The clinical picture, pathologic features and investigations appropriate for systemic cystic angiomatosis are briefly reviewed. The authors recommend conservative management when there is splenic involvement unless the size of the spleen makes its rupture likely or when there are hematologic complications.

L'hémangiome cutané est fréquemment retrouvé à l'examen clinique de routine et il est généralement sans importance. Toutefois, quand il y a atteinte des viscères, ces tumeurs vasculaires prennent une importance plus considérable à cause de la morbidité qui y est associée. On présente ici le cas d'une femme ayant des antécédents d'hémangiome du flanc, examinée pour hématurie et splénomégalie, et chez qui on découvrit de multiples atteintes angiomateuses viscérales. Elle subit par la suite une splénectomie. On commente les syndromes systémiques rares caractérisés par les tumeurs vasculaires et les cas signalé s'y trouve classifié. On passe brièvement en revue le tableau clinique, les caractéristiques pathologiques et les examens appropriés dans les cas d'angiomatose kystique systémique. Les auteurs recommandent un traitement conservateur quand il y a atteinte de la rate à moins que la taille de celle-ci fasse soupçonner une rupture possible ou quand il y a des complications hématologiques.

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Accepted for publication Sept. 29, 1986

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Although cutaneous hemangiomas are common and usually innocuous, they can be associated with a number of uncommon systemic syndromes of potentially serious nature. In the latter context, they may serve as a clue to an underlying pathologic condition. We describe a case of the rare syndrome of systemic cystic angiomatosis, emphasizing the generalized nature of the disorder and the unusual presenting complaint of hematuria.

Case Report

A 44-year-old woman was admitted with a 5-day history of painless hematuria and mild left-flank pain. Her medical history included surgical removal of a large left-flank hemangioma as a child.

Physical examination revealed mild left-flank tenderness and scarring and an enlarged spleen, palpable near the umbilicus medially. No other abnormalities were noted.

Laboratory investigations, including complete blood count, differential count, platelet

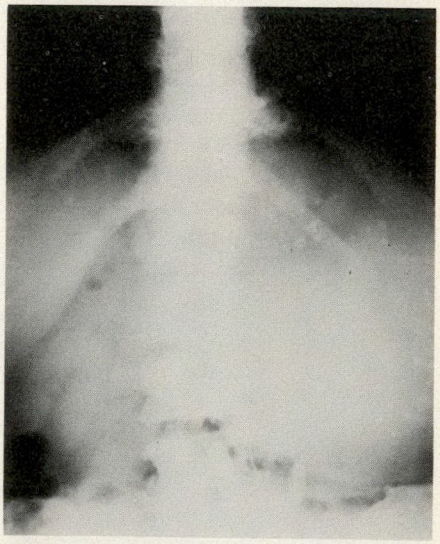

FIG. 1—Abdominal x-ray film showing calcific density near L3-4, splenomegaly with calcifications and irregularity of heads of ribs at T10 and T11, consistent with arteriovenous malformations.

count, blood smear, coagulation studies, liver transaminase determinations and creatinine measurement, were within normal limits. Urinalysis showed 150 red blood cells per high-power field with negative cytologic findings and cultures. Intravenous pyleography showed no urinary tract abnormality, but there was radiologic evidence of splenomegaly, abdominal wall calcifications and enlargement of vertebrae L3 and L4 (Fig. 1), findings consistent with arteriovenous malformation, and the left ribs appeared larger than the right on a chest x-ray film. Abdominal ultrasonography revealed multiple diffuse cystic lesions of the spleen and retroperitoneal area, believed to be hemangiomas (Fig. 2).

During cystoscopy, blood was seen to ooze from the left ureteric orifice, but a left retrograde pyelogram was normal. The hematuria stopped spontaneously after 5 days. Angiography showed hemangiomatous involvement of the left chest wall and splenomegaly without evidence of hemangioma formation. A diagnosis of splenic hemangioma was made and splenectomy advised as a prophylactic measure, because its enlarged size made rupture resulting from minor trauma a possibility. At laparotomy a large spleen, mottled by calcification, was removed. A large confluent hemangioma involved the left retroperitoneal area and encroached upon the left kidney. The liver and right kidney felt normal. No postoperative difficulties were encountered.

Pathological Findings

The spleen measured 16 × 13 × 7 cm and weighed 750 g. Multiple cystic and anastomosing vascular channels could be seen grossly, filled with blood and serosanguineous fluid (Fig. 3). These were lined by thin endothelium and surrounded by dense fibrous tissue. Some contained erythrocytes, others only proteinaceous material. Foci of calcification were present. The surrounding red and white pulp was displaced and distorted (Fig. 4). The diagnosis was lymphohemangioma.

Discussion

Vascular tumours have been described under a variety of names and syndromes: metastasizing benign angioma, angiomatosis, generalized lymphangioma, hemangiomatosis and multiple lymphangiectasis.[1] The underlying pathologic features have remained similar, consisting of a network of multiple cystic vascular and lymphatic channels.[2] The rarity with which they occur as a multiorgan problem has prevented a uniform nomenclature.

In our case, pathologic changes involved the spleen, bones, skin, retroperitoneum and the left kidney. This involvement suggests the syndrome of systemic cystic angiomatosis, described by Seckler and associates.[1] The differential diagnosis includes Klippel–Trenaunay syndrome (osteohypertrophic varicose nevus) characterized by hypertrophy of affected limbs, Osler–Weber–Rendu disease (multiple hereditary hemorrhagic telangiectasia), Sturge–Weber disease (encephalofacial hemangiomatosis), von Hippel–Lindau disease (hemangiomatosis of the retina, brain stem and other organs) and Maffucci's syndrome (cutaneous hemangioma and enchondroma of the bones). Similar splenic features may be seen in lymphangioma of the spleen,[3] lymphangioendothelioma of the spleen,[4] and peliosis of the spleen (never found without coexisting peliosis hepatis).[5] These all have features of our case and are a part of the differential diagnosis in patients with hemangioma.

Systemic cystic angiomatosis is a benign vascular tumorous formation that may be locally invasive. Only one case of truly malignant lymphangioma of the spleen has been described.[6] Involved organs may include spleen, liver, lung, pleura, subcutaneous tissues (especially the neck and axilla), thymus, mediasti-

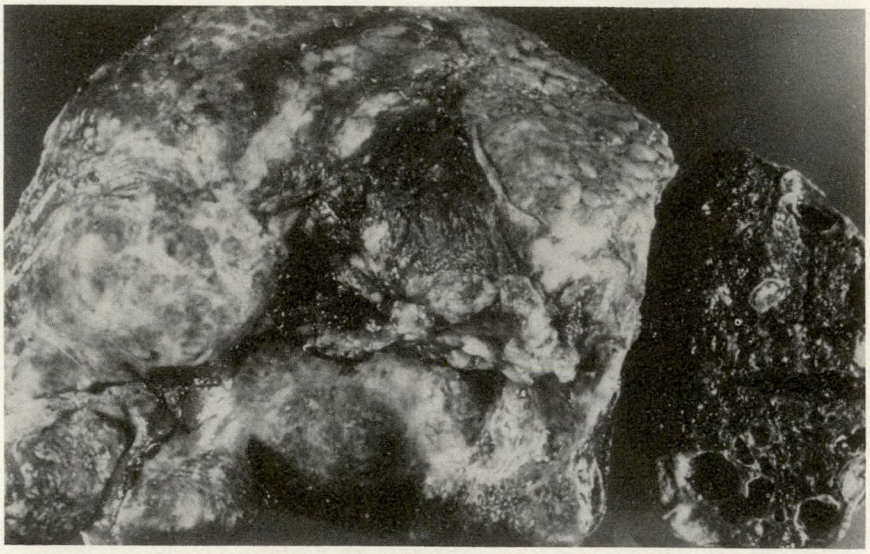

FIG. 3—Capsule of spleen (left) showing roughened pebble surface. Cut surface (right) shows cystic transformation by numerous blood-filled cavities.

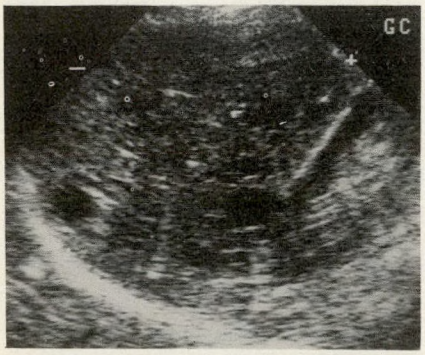

FIG. 2—Sonogram of enlarged spleen with multiple cysts of varying sizes, some with calcific foci.

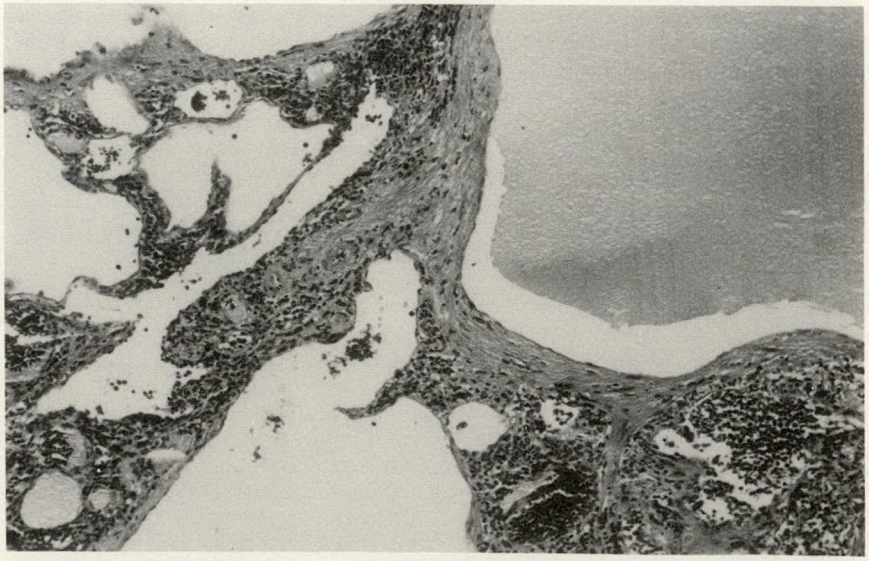

FIG. 4—Spleen shows numerous cysts lined by thin fibrous septae, some containing proteinaceous fluid (upper right), others red blood cells. Splenic red and white pulp is displaced, distorted and diminished (hemalum-phloxine-saffron stain, initial magnification × 60).

num, breast, retroperitoneum, kidney, skin and virtually any bone including the skull.[1] The multisystem involvement may reflect the congenital origin of these tumours.[7] The involvement of both lymphatic and vascular channels is more frequent than either alone, and different organs may contain different types of lesions. The congenital nature is suggested by the severe neonatal forms of the disease such as diffuse neonatal hemangiomatosis, generally a fatal condition.[8] In the older patient, the disease seems to progress more slowly.[9]

The absence of any family history and the age of our patient are not remarkable, as she had had a cutaneous hemangioma as a child, suggesting that the other lesions may have been present since then. Hematuria is an unusual presentation in this syndrome, but hemangiomas are a well-described cause of urinary bleeding.[10] Although no lesion was identified radiologically, a hemangioma is assumed because of the other lesions present and the proximity of the pathologic changes in the left retroperitoneal area. Bleeding in this syndrome normally does not result from thrombocytopenia; only one such case has been reported.[11] Despite the frequent finding of splenomegaly (50%), hypersplenism is not usual. Bleeding usually results from local tumour effects such as bruising with a pathologic fracture, hemoptysis with a lung lesion,[9] hemoperitoneum or bloody ascites from pleural or peritoneal angiomas.

No specific laboratory findings are present. However, various imaging procedures, while not always in agreement, are on the whole diagnostic, as in this case. The skeletal survey may demonstrate the extent of disease. A simple abdominal x-ray film may reveal a soft-tissue density from splenic enlargement with cyst-wall calcifications or bony involvement. Technetium-99m-labelled heat-damaged erythrocytes for radioisotope scanning provide a specific splenic imaging agent.[12] Ultrasonography may be especially helpful in splenic angioma, typically showing multiple echo-free lesions of various sizes. This is recommended as the method of choice for splenic imaging.[13] Some reviewers suggest angiography for definitive diagnosis, as a characteristic swiss-cheese appearance is pathognomonic. In our case, such an appearance was not seen. A picture of well-defined avascular lesions of varying size scattered throughout the spleen with no neovascularity, arteriovenous shunting or venous pooling is typical.[14]

Splenic involvement in this case was the reason for surgery, and perhaps other patients with apparently isolated splenic lesions have multiorgan involvement that is not recognized. Angiomas of the spleen are uncommon even if isolated, and the pathologic characteristics are essentially the same as in systemic angiomatosis.[3] The recognition of other lesions is not just of academic interest, as patients observed over time may show an increase in number and size of lesions.[11] Four of 13 patients in Seckler's study[1] died of local space-occupying effects and complications, including cervical cord compression, hepatosplenomegaly and chylous pleural effusion.

Therapy is primarily for local lesions and is surgical. Systemic therapy including chemotherapy is ineffective. The question of when to perform splenectomy might be guided by Halgrimson's observation that these lesions are frequently calcified and thought to represent a late dormant stage in the natural history of hemangioma, suggesting that they are unlikely to lead to new symptoms, and the patient can safely be observed without surgery.[15] Splenectomy is indicated when the spleen is greatly enlarged and traumatic rupture is considered likely with minor trauma, or if hematologic complications occur.

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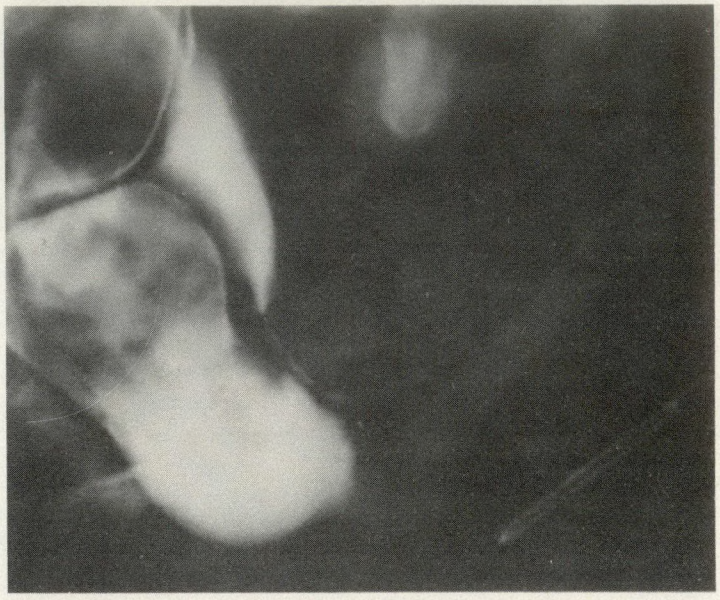
SESAP V Question



315. A baby boy born with an imperforate anus treated by sigmoid colostomy has intermittently required admission for watery diarrhea. Each hospitalization lasts three to five days, and typical serum electrolytes are serum sodium, 135 mEq/L; serum potassium, 5.5 mEq/L; serum chloride, 120 mEq/L; serum carbon dioxide content, 19 mEq/L, and pH, 7.27. The contrast radiograph shown above was performed by injection of the distal colostomy stoma when the baby was one year old. The most likely cause of this clinical picture is

(A) obstructive uropathy

(B) excessive loss of electrolytes due to gastroenteritis

(C) malabsorption caused by long-term administration of antibiotics

(D) excessive intake of liquids related to treatment of diarrhea

(E) urinary tract infection associated with passage of urine through the rectourethral fistula

For the incomplete statement above, select the one completion that is best of the five given.

For the critique of Item 315 see page 309.

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Unusual Presentation of a Pancreatic Pseudocyst: a Case Report

Although pancreatic pseudocysts are usually found in the vicinity of the pancreas, there are reports of them rupturing into the peritoneal cavity, fistulizing into the thorax or bowel and occupying unusual sites such as the mesentery, mediastinum and spleen.

This is a report of a patient with pancreatitis and a pancreatic pseudocyst presenting as a swelling of the left thigh due to dissection of the pseudocyst into the psoas muscle.

Même si les pseudokystes pancréatiques sont habituellement retrouvés dans la région du pancréas, on retrouve des articles signalant leur rupture dans la cavité péritonéale, leur fistulisation dans le thorax ou l'intestin ou leur présence dans des endroits inhabituels tels que le mésentère, le médiastin et la rate.

On signale ici le cas d'une patiente souffrant d'une pancréatite et d'un pseudokyste pancréatique qui est apparu sous la forme d'une tuméfaction de la cuisse gauche, à cause de la dissection du pseudokyste dans le psoas.

A complication of pancreatitis is the formation of pseudocysts, known to resolve spontaneously in about 41% of cases within 6 weeks.[1] However, a substantial number may cause complications or appear at unusual sites. Weaver and colleagues[2] noted that 80% of cases of pancreatic ascites resulted from pancreatic pseudocyst leakage. The pseudocysts were reported to rupture or track through the mediastinum into a pleural cavity[3] or perforate into the colon and duodenum.[4] They can present at unusual sites such as the spleen[5] and dissect into the groin.[6] We report another unusual presentation of pancreatic pseudocyst.

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Accepted for publication Sept. 29, 1986

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Case Report

A 42-year-old woman was admitted with a swollen and painful left thigh; the symptoms had developed 1 month earlier. She was known to be an alcoholic with chronic pancreatitis. Previous operations included cholecystectomy and gastrojejunostomy with vagotomy for gastric outlet obstruction secondary to pancreatitis, later converted to Roux-en-Y drainage because of bile reflux gastritis.

At examination the patient was in no distress but appeared older than her stated age. No mass or tenderness was detected on abdominal examination. Her left thigh was tender and markedly enlarged, with a circumference of 47 cm at the mid-thigh level, compared with 41 cm on the right side. The swelling was nonpitting and the skin had a bluish discolouration, most prominent near the groin.

Her hemoglobin level was 110 g/L, serum amylase level was 539 IU/L (normal 20 to 120 IU/L), alkaline phosphatase level was 498 IU/L (normal 100 to 275 IU/L) and the serum albumin level was 25 g/L (normal 30 to 50 g/L). The leukocyte count and blood urea, bilirubin, creatinine and aspartate aminotransferase levels were all normal.

A left lower limb venogram and lymphangiogram were normal. A computerized tomogram of the abdomen demonstrated diffuse pancreatic calcification and multiple radiolucent areas in an enlarged left psoas muscle (Fig. 1). In the thigh, all muscle groups and adjacent soft tissue were enlarged, with obliteration of the tissue planes (Fig. 2).

At laparotomy, the whole left psoas sheath appeared distended. It contained 1500 ml of straw-coloured fluid having an amylase content of 37 800 units/L. With the fluid removed, a communication between the left psoas sheath and pancreatic bed was discovered. The pancreas was fibrotic and thickened with chronic inflammatory changes. A splenectomy and distal pancreatectomy were performed well medial to the site where the pseudocyst clinically appeared to communicate with the pancreatic duct. An intraoperative pancreatogram revealed an ectatic duct with free flow of the dye into the duodenum and no extravasation. The duct was suture-ligated and the pancreatic stump oversewn. Penrose and sump drains were placed in the pancreatic bed and left psoas sheath.

Pathological examination of the pancreas showed chronic nonspecific pancreatitis.

Postoperatively the patient had an external pancreatic fistula that drained, at its peak, 1400 ml over 24 hours. Nasogastric suction and parenteral nutrition resulted in a substantial decrease in drainage. Three weeks postoperatively a left subphrenic fluid collection was drained surgically. She was discharged 3 weeks later in good condition, but with a persistent fistula draining up to 10 ml/d. The swelling of the left thigh had resolved at the time of discharge.

Discussion

Pancreatic pseudocysts are uncommon but well-recognized complications of acute and chronic pancreatitis. They have the ability to enlarge and migrate,[6] form an internal fistula[3] and present in unusual locations.[5,6]

Our case illustrates a unique complication of pancreatitis. Although not radiologically demonstrated, an internal fistula between the left psoas sheath and the pancreatic duct was presumably formed either directly or by way of a pseudocyst. Over time, pancreatic secretions collected in, and slowly enlarged, the psoas muscle and sheath, eventually dissecting into the left thigh where the fluid diffused into the compartments causing swelling and pain.

The computerized tomogram was instrumental in diagnosing this particu-

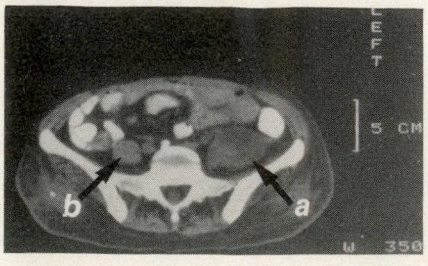

FIG. 1—Discrepancy in size between left (a) and right (b) psoas muscles.

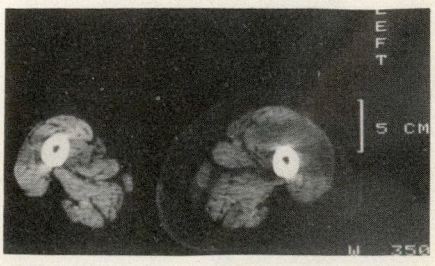

FIG. 2—Swelling and increased size of left thigh muscles.

lar problem. Ideally, endoscopic retrograde cholangiopancreatography would have been helpful in demonstrating the fistula between the pancreatic duct and pseudocyst, but unfortunately the previous surgery precluded this examination. We elected to perform distal pancreatectomy because at laparotomy we believed that the changes in the psoas sheath were closely related to the posterior aspect of the distal pancreas with a communication between the left psoas sheath and pancreatic bed even though no definite ductal disruption was visualized.

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Recognition and Management of Marlex Erosion After Horizontal Gastroplasty for Morbid Obesity

Between 1979 and 1982, 162 patients underwent horizontal gastroplasty in which polypropylene mesh (Marlex) was used to support the greater curve channel. There were no deaths and early weight loss was good. The operation was stopped because of late weight gain. Marlex erosion was recognized by endoscopy in 10 (6%) patients between 27 and 60 months after operation. Two other patients with Marlex erosion had the gastroplasty performed elsewhere.

The patients' symptoms were abdominal pain, vomiting or weight gain. The Marlex was often difficult to resect because the inflammatory mass was adherent to spleen or pancreas. Reconstruction by Roux-en-Y gastric bypass or vertical banded gastroplasty allowed continued weight loss and gastrogastrostomy, although technically easier, resulted in weight gain. The author concludes that long-term follow-up is necessary after any gastroplasty or gastric bypass procedure especially when foreign material is used to support the stoma.

De 1979 à 1982, 162 patients ont subi une gastroplastie horizontale au cours de laquelle de la toile de polypropylène (Marlex) a été utilisée pour maintenir le canal de la grande courbure. On n'a enregistré aucun décès et la perte de poids initiale fut bonne. On mit fin à cette opération à cause du gain de poids subséquent. A l'endoscopie, on a identifié une érosion de la toile Marlex chez 10 (6%) des patients de 27 à 60 mois après l'opération. Deux autres patients qui avaient subi leur gastroplastie dans un autre centre présentaient aussi une érosion de la toile Marlex.

Les symptômes consistaient en douleur abdominale, vomissements ou gain pondéral. La toile Marlex fut souvent difficile à réséquer à cause de la masse inflammatoire qui adhérait à la rate ou au pancréas. Une reconstruction par dérivation gastrique de Roux-en-Y ou par gastroplastie à sections verticales a permis de poursuivre la perte de poids alors que la gastro-gastrostomie, bien que techniquement plus facile, a résulté en un gain de poids. L'auteur conclut à la nécessité d'une surveillance prolongée après toute intervention de gastroplastie ou de dérivation gastrique, spécialement quand des matériaux étrangers sont utilisés pour soutenir le stoma.

Gastroplasty for morbid obesity is now a common operation, performed in many hospitals in North America. Late weight gain, often due to stomal dilatation, has been a problem after many types of gastroplasty and has led surgeons to support the stoma with materials such as polypropylene mesh (Marlex).

Over the last 5 years, Marlex erosion occurred in 12 of 162 patients who underwent horizontal gastroplasty. The late appearance of this problem suggests that such cases will continue to be seen for a long time.

This report emphasizes the potential problems in the use of foreign materials around gastric stomas. Long-term follow-up of patients who have had gastroplasties is recommended.

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Accepted for publication Mar. 4, 1987

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Patients and Methods

Between September 1979 and October 1982, 162 consecutive gastroplasties were performed in 140 women and 22 men, using the technique illustrated in Fig. 1 and described elsewhere.[1] One double staple line was placed in 10 patients; in all the others, two double staple lines were placed 1 cm apart. Gastric pouch volume was less than 100 ml. Three staples were removed from the greater curve end of the long staple line and two from the same end of the short staple line. The stapler was applied across the entire stomach. The resultant stoma was wrapped with a 2 × 6-cm strip of Marlex mesh held in place with a 2-0 Prolene suture passed through the mesh 5 mm from each end and through the stomach just above and below the greater curve of the staple line. The suture was tied snugly but not tightly, effectively closing the blind space between the staple lines and securing the mesh to

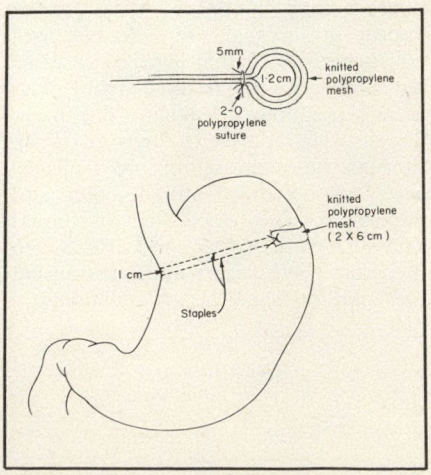

FIG. 1—Transverse gastroplasty as performed between 1979 and 1982.

the stomach. Omentum was placed over the mesh. A no. 18 nasogastric tube with extra holes above and below the staple line was passed through the opening and left in place for 48 hours.

Patients were at least 45 kg above ideal weight (mean 134 ± 20 kg) and between the ages of 17 and 58 years (mean 36 ± 8 years). Treatable endocrine disorders were excluded by preoperative assessment. Most of the 162 patients had potentially reversible disorders related to obesity, such as diabetes, hypertension, osteoarthritis and infertility. All patients were assessed preoperatively by an internist and were required to stop smoking as a condition of acceptance for gastroplasty. Heparin (5000 units) was given subcutaneously to all patients before operation and cefazolin (1 g) was given intravenously once before and twice after operation. A fluid and puréed diet was advised for the first month postoperatively and a soft diet for the second month. Patients were seen at monthly intervals for the first 6 months and every 3 months thereafter.

Revision gastroplasty was usually performed by the techniques illustrated in Figs. 2 and 3. The GIA or TA-55 staplers were used for wedge excision of the eroded Marlex, and Roux-en-Y gastric bypass was performed by the technique of Pories and colleagues.[2] For two of the later cases of staple-line disruption, vertical banded gastroplasty[3] was performed after Marlex resection. No gastrostomy was carried out.

Results

Weight loss at 1 year was 33% of the initial weight. The low early failure rate rose progressively with time. There were no postoperative deaths. One leak occurred 5 days after gastroplasty and the patient recovered after drainage and removal of the Marlex. Five (3%) patients had stomal obstruction within 2 months of operation. They required a gastrogastrostomy. In these patients Marlex was not removed. Recovery was excellent but the patients gained weight.

The late problems with Marlex are documented in Table I. Ten of the 12 patients were from our series while 2 had the initial operation elsewhere; 11 were women. Eight of the 12 patients had nausea and vomiting, 3 of these had complete stomal obstruction, 6 had weight gain and 5 abdominal pain. An upper gastrointestinal series after a barium meal demonstrated stomal obstruction or enlargement but not Marlex erosion, endoscopy being the most useful procedure. Eroded Marlex could be seen as a black mass adherent to the greater curve of the stomach. In three patients the stoma was completely obstructed. In eight, the staple line adjacent to the Marlex had disrupted leaving a large stoma, explaining the weight gain. Gastric ulceration adjacent to the Marlex was unusual but was observed in one patient. In several cases, attempts were made to remove the Marlex with forceps and traction but it was too tightly adherent to the gastric wall.

Although stomal obstruction often occurred suddenly, other symptoms often persisted for months before the diagnosis of Marlex erosion followed by reoperation. In several patients weight gain was the major sign and eroded Marlex was observed adjacent to a dilated stoma. Reoperation was carried out a mean of 44 months after initial gastroplasty (range from 27 to 60 months). Since only patients with major weight gain or severe symptoms underwent endoscopy, it is possible that the frequency of this problem has been underestimated.

Preparation for reoperation was routine except in the three patients with stomal obstruction. They required nasogastric decompression and one patient with severe weight loss was given parenteral nutrition preoperatively. Stomal dilatation was attempted as previously described[4] but was unsuccessful. At operation, the eroded Marlex was part of an inflammatory mass that was often difficult to resect. Although the Marlex had been covered by omentum in most cases, the mass was often adherent to pancreas or spleen. Careful dissection was necessary and bleeding was sometimes troublesome. In patient 6, splenectomy was necessary and discharge was delayed by hematoma formation in the left upper quadrant. Patient no. 10 underwent the original gastroplasty at another hospital. The Marlex did not appear to have been covered with omentum; it had retracted upward and was adherent to the diaphragm. The Marlex was left in place without apparent ill effect and the patient was well 4 months later. He requested the safest operation and underwent only a

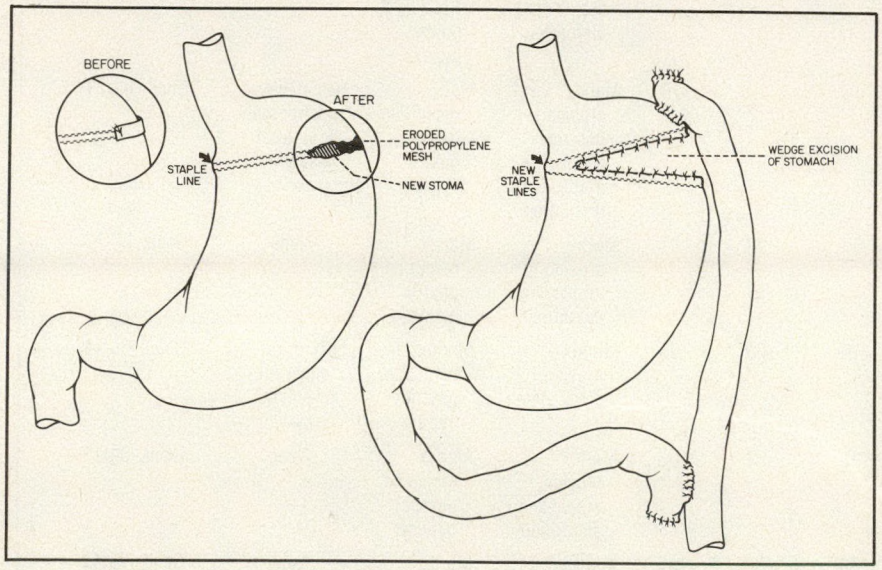

FIG. 2—Method of reconstruction in most patients after Marlex erosion.

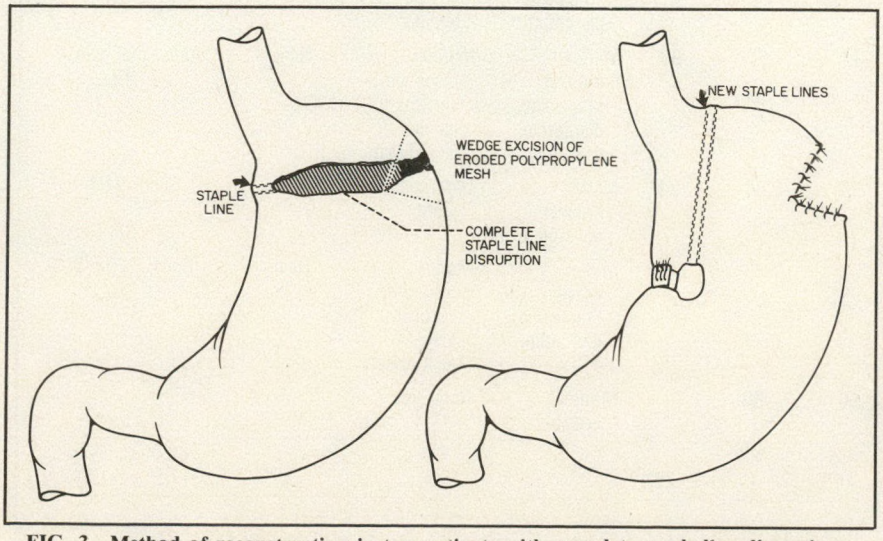

FIG. 3—Method of reconstruction in two patients with complete staple-line disruption.

gastrogastrostomy. Stomach adjacent to the eroded Marlex was often thickened and inflamed. Care was necessary to get a secure staple line, and staple lines were usually oversewn. In two of the later cases the transverse staple line had disrupted almost completely allowing a vertical banded gastroplasty to be performed. Care was taken to avoid constriction or obstruction of the stomach at the point where transverse and vertical staple lines intersected. The appearance of the eroded Marlex is shown in Figs. 4 and 5. In patient 12, Marlex erosion was diagnosed recently by endoscopy. Although the patient had been vomiting, the stoma was patent. So far, she has been willing to accept her symptoms because of the risks of reoperation and the potential for weight gain.

Although the operations were often

Table I—Data for Patients With Marlex Erosion After Gastroplasty

Patient no.	Age, yr	Sex	Preop weight, kg	Symptoms/ signs	Months to re-operation	Minimum weight, kg	Revision weight, kg	Findings	Treatment	Complications	Present condition, weight (kg)/duration of follow-up (mo)
1	28	F	147	Vomiting, stomal obstruction	34	76	76	Marlex erosion, no stoma	Marlex resection, gastrogas-trostomy	None	Weight gain, 103/11
					45		103	Large stoma	Gastric bypass	None	Good, 85/35
2	49	F	123	Vomiting, stomal obstruction	45	57	57	Marlex erosion, stomal obstruction	Marlex resection, Roux-en-Y, gastric bypass	None	Good, 82/36
3	49	F	125	Left upper quadrant pain, weight gain	27	93	104.5	Marlex erosion, staple-line disruption	Marlex resection, Roux-en-Y, gastric bypass	None	Good, 86/24
4	31	F	115	Abdominal pain	46	76	76	Marlex erosion, gastric ulceration, staple-line disruption	Marlex resection (refused gastric bypass)	Hematemesis, gastrectomy 1 yr later (elsewhere)	Good, 61/21
5	34	F	120	Vomiting, abdominal pain, weight gain	37	72	107	Marlex erosion, staple-line disruption	Marlex resection, gastric bypass	None	Good, 80/18
6	38	F	102.5	Vomiting	49	63	63	Marlex erosion, small stoma	Marlex resection, gastric bypass	Left upper quadrant bleeding, splenectomy	Good, 69/14
7	33	F	119	Vomiting, abdominal pain, weight gain	35	81	103	Marlex erosion, staple-line disruption	Marlex resection, gastric bypass	None	Good, 84/13
8	52	F	115.5	Nausea, weight gain	60	88	107	Marlex erosion, staple-line disruption	Marlex resection, gastric bypass	None	Good, 75/12
9	37	F	111	Weight gain, abdominal pain, burping	47	77	93	Marlex erosion, staple-line disruption	Marlex resection, vertical banded gastroplasty	None	Abdominal pain, 71/12
10	37	M	227	Vomiting, stomal obstruction	36	84	84	Marlex erosion, no stoma	Gastrogastro-stomy	None	Good, 91/4
11	54	F	177	Heartburn, weight gain	58	89	145	Marlex erosion, staple-line disruption	Marlex resection, vertical banded gastroplasty	None	Good, 129.5/3
12	36	F	139	Vomiting	60	60		Marlex erosion	None yet		
Means	39.9 ± 9.1		135.0 ± 36.8		44.4 ± 10.5	76.3 ± 11.1	92.3 ± 25.1*				83.0 ± 17.7/ 17.5 ± 11.3†

*Initial revision weight only for patient 1.
†Final weight and follow-up date only for patient 1.

difficult, recovery was uncomplicated apart from the case of left upper quadrant hematoma after splenectomy. Weight loss was good with gastric bypass and satisfactory with vertical banded gastroplasty. Patient 1 with a gastrogastrostomy had substantial weight gain which was controlled by gastric bypass. Patient 10 is doing well after gastrogastrostomy although weight gain is expected.

Discussion

Erosion of the gastrointestinal tract by foreign materials ranging from sump drains to the Angelchik prosthesis[5] has been described. It is not surprising that Marlex sutured to the stomach should sometimes erode, and our data suggest that erosion may not be recognized for many years after the initial operation. Symptoms and signs may vary widely from complete and surprisingly sudden stomal obstruction to abdominal pain and weight gain. Erosion of the Marlex at the end of a staple line appears to weaken it and may initiate the disruption. It is possible that erosion has occurred without symptoms in other patients who have not been subjected to endoscopy. The act of fixing the Marlex to the stomach with sutures penetrating the gastric wall may lead to erosion. In vertical banded gastroplasty the Marlex is passed through the circular window and fixed to itself. The lack of fixation to the stomach may reduce the likelihood of erosion.[3]

The reoperations proved difficult although there were no serious complications. The major problem was removal of the Marlex which was obscured by an inflammatory mass adherent to spleen or pancreas. The lack of symptoms after leaving the Marlex in place in one patient with stomal obstruction suggests that Marlex need not always be removed as long as obstruction is relieved. Even in those with a large stoma it may be possible to leave the Marlex below new staple lines, although staples may not hold well when placed in or near an inflammatory mass. Gastric bypass or vertical banded gastroplasty allow continued weight loss and can be performed safely, although gastrogastrostomy is a much easier operation for the high-risk patient and when technical difficulties occur. We did not use gastrostomy or jejunostomy but would recommend either procedure if the revision operation was particularly difficult.

Erosion of Marlex is not the only factor that can cause late obstruction and vomiting. Other possible causes include inflammatory adhesions to the Marlex mesh with torsion of the stomach, stomal ulceration, impaired emptying of the pouch due to pooling of contents in a large dependent pouch or poor motility and stricture formation under the mesh. Dilatation is the easiest way to manage stomal obstruction, but this may not be possible when the stoma is wrapped with Marlex.[4]

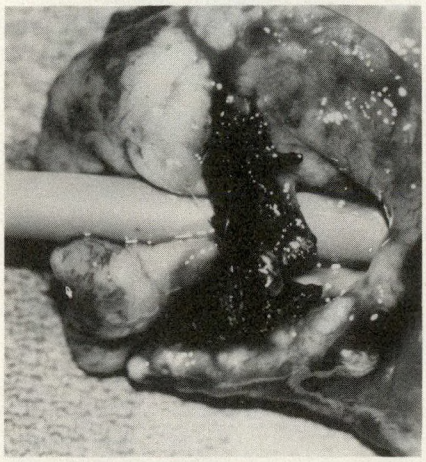

FIG. 4—Appearance of eroded Marlex after resection.

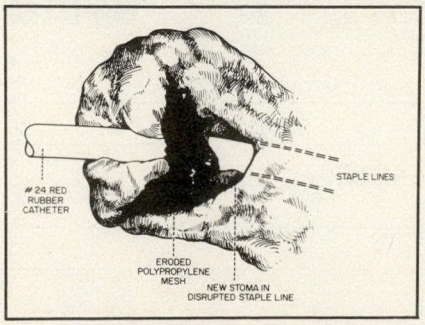

FIG. 5—Artist's depiction of eroded Marlex.

Summary and Conclusions

Marlex erosion has been detected as a late complication in 12 patients after transverse gastroplasty for morbid obesity. The symptoms included vomiting, abdominal pain and weight gain. The problem was identified by endoscopy but not by upper gastrointestinal series. The patients were managed by Marlex resection and Roux-en-Y gastric bypass or vertical banded gastroplasty, with good results. Horizontal gastroplasty is an unsatisfactory operation, and any patient with a gastric partition or gastric bypass requires long-term follow-up, especially when foreign materials are used to support the stoma.

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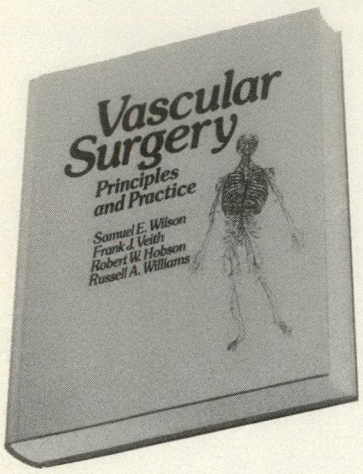

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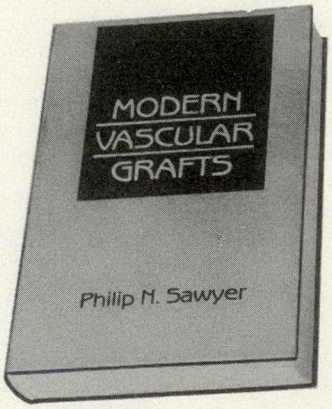

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Giant Villous Adenoma of Second Portion of Duodenum: Case Report

Villous adenomas of the duodenum are rare. In this report the authors describe the case of a 75-year-old woman who had an extensive adenoma involving the second portion of the duodenum and ampulla of Vater. The patient was successfully managed by a pancreaticoduodenectomy. The authors describe the histopathology of the lesion and suggest guidelines for surgical consideration.

Les adénômes villeux du duodénum sont peu fréquents. Ce rapport décrit une lésion très étendue comprenant toute la deuxième partie du duodénum ainsi que l'ampoule de Vater chez une femme de 75 ans. L'investigation ayant mené au diagnostic, le traitement chirurgical et les conclusions histo-pathologiques sont présentés. Quelques pensées au sujet de la tenue à prendre envers ces lésions sont offertes.

Villous adenomas, although they are found throughout the gastrointestinal tract, are rare in the duodenum. Fewer than 75 such cases have been reported in the English medical literature since 1893.[1] These lesions have a propensity for malignant transformation, especially when they involve the duodenum and the ampulla of Vater simultaneously,[1,2] and their detection is often incidental. The size of villous adenomas may or may not correlate with malignant change.[3–5] Jaundice,[6] obstruction[1] and bleeding[7] are the most frequent clinical features in patients with villous adenomas of duodenum and ampulla of Vater.

The patient whose case is described in this paper was referred to the gastrointestinal service with vague abdominal distress and was found to have a large duodenal and ampullary villous adenoma, containing foci of both in-situ and invasive adenocarcinoma.

Case Report

A 75-year-old white woman complained that she had had a vague gnawing sensation in the abdomen for more than 2 years. Physical examination and results of hematologic and biochemical studies failed to provide a diagnosis, but ultrasonography demonstrated a 1.5-cm dilatation of the common bile duct. She underwent endoscopic retrograde cholangiopancreaticography (ERCP), which demonstrated an extensive lesion involving the entire mucosa of the second portion of the duodenum and the ampulla of Vater, the orifice of which was obscured, preventing cannulation. Multiple biopsies were taken and later a barium study was performed to determine the extent of the lesion. Results of liver function tests and amylase levels were normal. A computerized tomogram demonstrated a thickening of the wall of the second part of the duodenum and dilatation of the common bile duct. Selective celiac and mesenteric arteriograms showed a lesion of the second portion of the duodenum with increased vascular supply from branches of the gastroduodenal artery.

At laparotomy a diffuse soft mass was palpated, filling the second portion of the duodenum. There was no proximal dilatation. The gallbladder was thickened and distended, but the pancreas felt normal. The second portion of the duodenum and head of the pancreas were mobilized medially. Lymph nodes from the retroperitoneal space were examined by frozen section and no malignant cells were found. Once the common duct, portal vein and vena cava had been exposed, a pancreaticoduodenectomy was feasible and safely performed. The postoperative course was uncomplicated and the patient was discharged 11 days later.

Pathological Findings

The operative specimen showed a circumferential mucosal villous tumour, 8 cm long, extending equidistant from the ampulla in the second portion of the duodenum. The wall of the ampulla of Vater was thickened, its orifice narrowed and the common duct distended to 1.5 cm in diameter (Figs. 1 and 2).

The tumour, a villous adenoma, was characterized by folds of hyperplastic surface epithelium protruding into and narrowing the duodenal lumen (Fig. 3). Multiple sections (32) revealed one focus of adenocarcinoma in situ, as demonstrated by a cribriform proliferation of the surface epithelium with cellular atypism (Fig. 3). In another zone of the adenoma was an area of submucosal invasive adenocarcinoma with marginal intramucosal carcinoma (Fig. 4).

The villous adenoma involved the

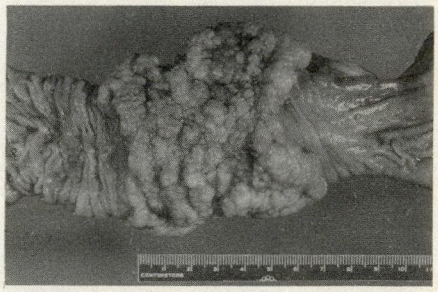

FIG. 1—Duodenal villous tumour involving entire circumference of mucosa of second portion of duodenum and ampulla of Vater.

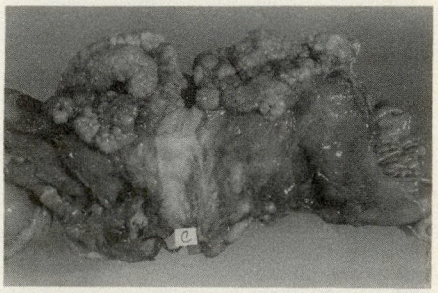

FIG. 2—Villous tumour located equidistant from papilla. Papilla and ampulla of Vater are thickened and common duct lumen (C) is slightly widened.

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Accepted for publication Sept. 29, 1986

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ampulla of Vater (Fig. 5). No metastasis was noted in the 20 lymph nodes identified.

Discussion

Although villous adenomas have been observed in the first and third parts of the duodenum,[3,6,8] the majority are located in the second portion,[8,9] the number possibly being inflated by the inclusion of localized ampullar adenomas. Some observers believe that the incidence of malignant transformation in villous adenomas of the duodenum and small intestine parallels their increase in size. In a recent review, Perzin and Bridge[5] detailed a retrospective study of 51 patients with adenomatous lesions of the small bowel among 392 000 surgical pathological examinations. The benign adenomas were observed to have an average diameter of 2.65 cm versus 3.7 cm for adenomas incorporating a malignant component.

The type of surgical resection chosen for villous tumours of the duodenum depends on a number of factors: the demonstration of a focus of invasive adenocarcinoma within an existing predominantly villous adenoma, the location and confines of the adenoma in the duodenum, the size and type (pedunculated versus sessile), and on the simultaneous involvement of the duodenum and ampulla of Vater.

Although preoperative biopsies of the lesion in our case did not demonstrate malignant change, the surgical procedure selected was a pancreaticoduodenectomy, a decision based on the size and extent of the tumour, its sessile rather than pedunculated nature, the involvement of the ampulla of Vater with radiologic evidence of luminal narrowing and, finally, the difficulty in demonstrating by frozen section a possible site of invasive carcinoma in a lesion of this size.

Our case demonstrated the coexistence of focal, in situ, intramucosal and invasive carcinoma, a finding that has frequently been observed in large villous adenomas of the duodenum.

In our patient the difficulty in making an early diagnosis related to a paucity of symptoms. The narrowing of the duodenal lumen did not lead to obstruction, the increased vascularity of the tumour did not cause bleeding and the ampullary involvement did not result in jaundice or cholangitis.

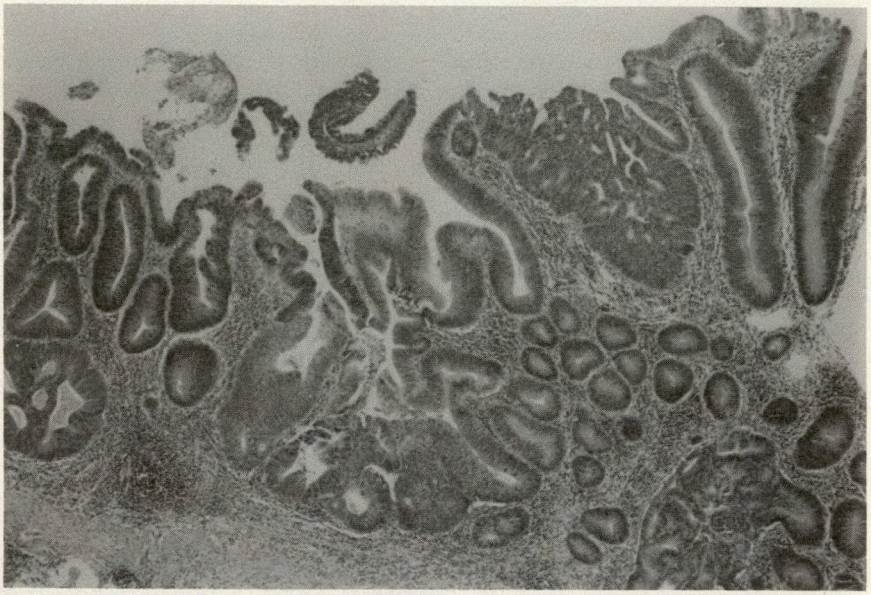

FIG. 3—Microscopic focus of adenocarcinoma within lesional alterations of villous adenoma. Surface epithelium forms cribriform proliferative pattern with evidence of intact basal layer (hematoxylin and eosin, original magnification × 67).

FIG. 4—Adjoining foci of villous adenoma, intramucosal adenocarcinoma and submucosal invasive carcinoma (hematoxylin and eosin, original magnification × 27).

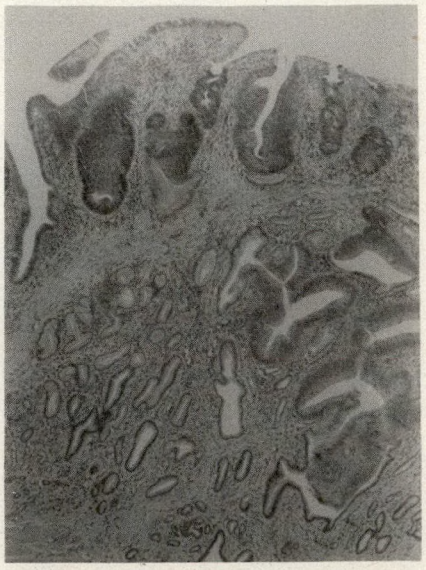

FIG. 5—Villous adenoma also involving ampulla of Vater (hematoxylin and eosin, original magnification × 69).

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Radiocarpal Osteoarthritis Associated With Bilateral Bipartite Carpal Scaphoid Bones: a Case Report

Bipartite carpal scaphoid is a rare congenital anomaly. The authors report on a 56-year-old man who presented with bilateral wrist pain without a history of trauma. X-ray films demonstrated bilateral symmetric bipartition of the scaphoid into a large distal ossicle and a smaller proximal ossicle. There was degenerative change in the articulation between the distal ossicle and the radial styloid. The joint space between the proximal ossicle and the scaphoid fossa was preserved. The absence of periscaphoid degenerative change has been suggested as a criterion for the diagnosis of congenital bipartite scaphoid. This patient fulfilled all other criteria for the diagnosis and the authors suggest that degenerative change developed due to abnormally high contact force between the distal ossicle and the radial styloid. An identical pattern of degenerative change has been observed in long-standing nonunion of the scaphoid, likely due to a similar mechanism. Radiocarpal osteoarthritis can develop in patients with congenital bipartition of the scaphoid and is similar to that observed in long-standing nonunion of the scaphoid.

Le scaphoïde carpien bipartite est une anomalie congénitale rare. Les auteurs commentent le cas d'un homme de 56 ans vus souffrant de douleurs bilatérales aux poignets alors qu'il n'y avait pas d'antécédent de traumatisme. Les radiographies ont démontré une division en deux parties symétrique bilatérale du scaphoïde donnant lieu à un osselet proximal gros et à un osselet distal plus petit. On constatait une modification dégénérative de l'articulation entre l'osselet distal et le styloïde radial. L'espace articulaire entre l'osselet proximal et la fossette scaphoïde n'était pas touché. L'absence de changement dégénératif periscaphoïde a été proposée comme critère diagnostique du scaphoïde carpien bipartite. Le malade qui nous occupe remplissait tous les autres critères diagnostiques et les auteurs suggèrent que les changements dégénératifs observés étaient dûs à la force de contact élevée entre l'osselet distal et le styloïde radial. Une évolution identique des modifications dégénératives a déjà été observée dans des cas d'absence d'ossification de longue date, probablement à cause de mécanismes similaires. Une arthrose radio-carpienne peut apparaître chez les patients souffrant de scaphoïde bipartite congénital et celle-ci ressemble à celle qu'on observe quand il y a absence prolongée d'ossification du scaphoïde.

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Accepted for publication Dec. 8, 1986

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Many authors[1-13] have reported bipartite carpal scaphoid as a rare congenital anomaly. The condition may be isolated or it may occur in conjunction with other congenital anomalies of the hand or forearm.[14] The differentiation of scaphoid bipartition from nonunion of the scaphoid is important so that unnecessary immobilization and surgery can be avoided.[13] Furthermore, accurate recognition of the condition is important in compensation cases in order to avoid inappropriate judgements. The absence of periscaphoid degenerative change has been suggested as a criterion for the diagnosis of congenital bipartite scaphoid.[10,13,14] We recently treated a patient who had radiocarpal osteoarthritis in association with bilateral bipartite carpal scaphoid bones.

Case Report

A 56-year-old right-handed male factory worker complained of bilateral wrist pain. The pain had developed spontaneously 2 years earlier. There was no history of trauma or other joint problem. On specific questioning, the patient denied any history of even trivial injury to or immobilization of either wrist joint. There was no family history of a joint problem in general or of the wrist in particular. The pain was aching in nature, exacerbated by activity and localized to the dorsal radial aspect of both wrists. The patient had been employed at a foundry for 15 years where he regularly had to push a heavy hopper down a track.

There was tenderness over the dorsal radial aspect of both wrists. The range of motion was 45° dorsiflexion, 40° volar flexion, 15° ulnar deviation and 10° radial deviation bilaterally. Pronation and supination were full. Discomfort was felt at the extremes of motion. Maximum grip strength measured with a Jaymar dynanometer was at the third setting and measured 28 kg on the right and 18 kg on the left. Other findings on examination were within normal limits.

X-ray films demonstrated identical findings in both wrists (Fig. 1). The scaphoid consisted of two separate ossicles with smooth margins. The distal ossicle was larger than the proximal one. There was no evidence of cyst formation, increased density or osteoporosis. Lateral radiographs (Fig. 2) showed no evidence of intercalary instability. The joint space between the radial styloid and the distal ossicle was narrow and the subchondral bone was sclerotic. The joint space between the proximal ossicle and the scaphoid fossa was preserved. A bone scan (Fig. 3) showed increased activity in the region of the scaphoid and lunate bilaterally consistent with a degenerative process. Use of a nonsteroidal antinflammatory agent partially relieved the patient's symptoms. Bilateral leather wrist gauntlets were prescribed to be worn at work. One year later the patient still complained of pain, but he had not changed his occupation.

Discussion

Bipartite carpal scaphoid is a rare congenital anomaly. Gruber[15] found four bipartite scaphoid bones among 3007 dis-

sections and Pfitzner[1] found nine in 1456. It has been proposed that scaphoid bipartition results from failure of the distal (radial) and proximal (ulnar) components of the scaphoid to fuse. However, the very existence of scaphoid bipartition has been questioned.[16] In view of the absence of a history of trauma and the symmetrical radiologic findings, we believe that our patient had congenital bilateral bipartition of the scaphoid. He fulfils Bunnell and Boyes' criteria[10] for the diagnosis, with the exception of degenerative change within the radiocarpal articulation. However, the presence of degenerative change in association with scaphoid bipartition is consistent with current knowledge of carpal biomechanics and the patient's long history of manual labour. Others[5,9,11,17] have stated that arthritic changes may occur with bipartite scaphoid.

Radiocarpal osteoarthritis is usually associated with chronic ligamentous instability,[18] malunion of displaced intra-articular fractures[19] and nonunion of the scaphoid.[20,21] Watson and Brenner[18] noted that degenerative arthritis of the carpus develops when small cartilaginous contact surfaces are subjected to high shear loading. They also noted that the process of degenerative arthritis associated with long-standing nonunion of the scaphoid begins between the radial styloid and the distal scaphoid fragment. In contrast, the proximal pole fragment in the scaphoid fossa is always spared. This radiographic pattern was observed in our patient, suggesting that degenerative arthritis associated with bipartition of the scaphoid develops by a mechanism similar to that observed in nonunion of the scaphoid. We suggest that scaphoid bipartition increased the contact force on the articulation between the radial styloid and the distal ossicle resulting in the development of degenerative arthritis. The radiographic pattern of arthritic change was identical to that observed in long-standing nonunion of the scaphoid.

Bone grafting of the scaphoid was not recommended in our patient because of the relatively advanced arthritic changes present when we saw him. It has been clearly shown that long-standing nonunion of the scaphoid leads to progressive degenerative carpal arthritis.[20,21] Since degenerative change also developed in our

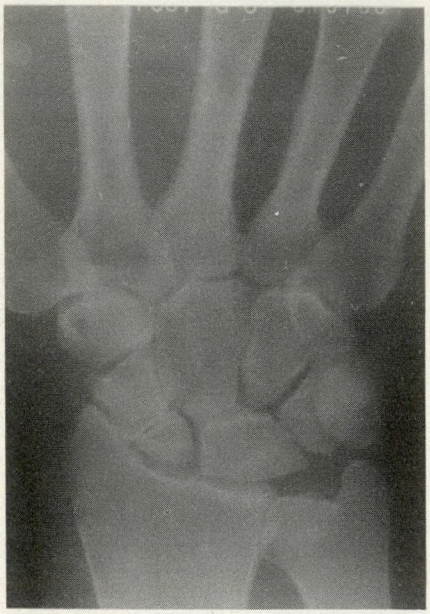

Fig. 1a

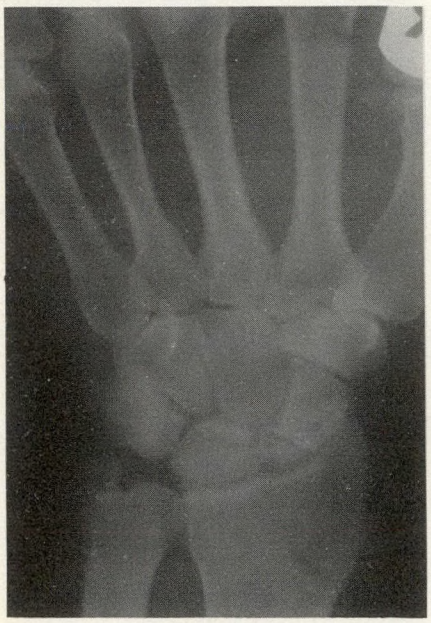

Fig. 1b

FIG. 1—Posteroanterior views of (a) left and (b) right wrists of patient. Both scaphoids are bipartite. Distal ossicles are larger than proximal and edges are rounded. There is joint-space narrowing and subchondral sclerosis between each distal ossicle and radial styloid. Joint space between proximal ossicle and scaphoid fossa is preserved.

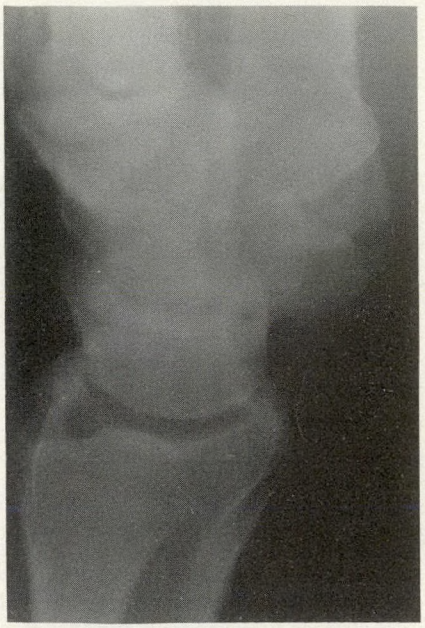

Fig. 2a

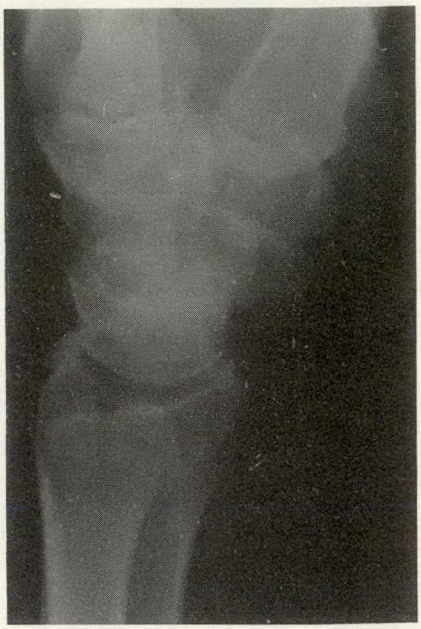

Fig. 2b

FIG. 2—Lateral views of (a) left and (b) right carpi do not show evidence of intercalary instability.

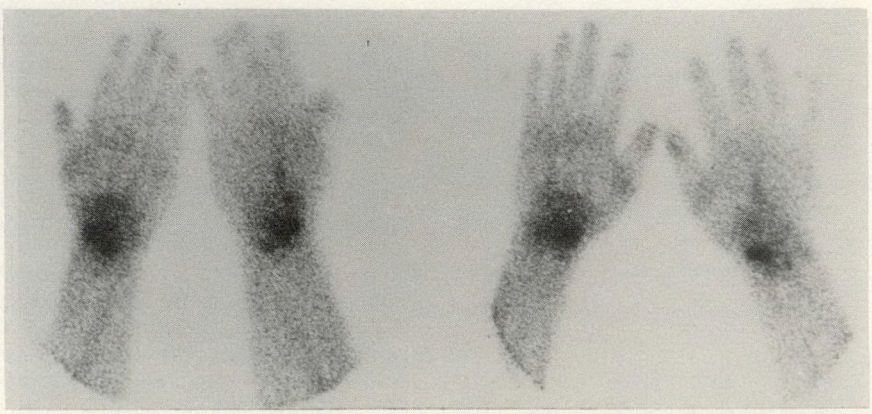

FIG. 3—Delayed technetium-99m bone scan demonstrating increased uptake affecting radioscaphoid articulation. Pattern of uptake is consistent with degenerative process.

patient, bone grafting might be considered for patients with symptomatic scaphoid bipartition who present without degenerative changes. Although differentiation of nonunion of the scaphoid from scaphoid bipartition is important diagnostically, it would appear, on the basis of this case report, that both conditions can result in identical patterns of carpal arthritis.

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JOHN R. TANNER, MD, FRCSC

St. Anthony's Fire, Then and Now: a Case Report and Historical Review

A rare case of morbid vasospasm, together with striking angiographic findings, is described secondary to the ingestion of methysergide by a 48-year-old woman. A brief review of the literature on similar cases is presented.

A discussion of the history of ergot includes its original discovery, the epidemics of gangrene that it has caused through the ages and its past and present role in the management of migraine headache. Despite the advent of calcium channel blockers and β-adrenergic antagonists, ergot preparations continue to play a major role in migraine therapy, so that the danger of St. Anthony's fire persists.

On décrit un cas rare de vasospasme pathologique survenant de concert avec des modifications angiographiques marquées chez une femme de 48 ans qui avait pris du méthysergide. On fait une brève revue d'articles traitant de cas similaires.

Le commentaire sur l'histoire de l'ergot de seigle, porte sur sa découverte originale, les épidémies de gangrène dont il fut la cause à travers les âges et son rôle passé et actuel dans le traitement de la migraine. Malgré l'avènement des bloqueurs calciques et des antagonistes β-adrénergiques, les préparations de dérivés de l'ergot continuent de jouer un rôle majeur dans la thérapie de la migraine de sorte que, encore de nos jours, le danger du feu de Saint-Antoine persiste.

Epidemic gangrene has been recorded since the 9th century AD and a "noxious pustule in the ear of grain" (i.e., ergoty rye) was described on an Assyrian tablet in 600 BC.[1] Written records of epidemic gangrene appeared in the 10th and 11th centuries AD. The Greeks and Romans did not eat rye; it was the conquering Teutons who popularized its consumption, and thus were responsible for the spread of ergotism throughout Western Europe. The connection with St. Anthony of Egypt (251–356 AD), "patriarch of monks and healer of both men and animals", developed some time later, probably with the translation of his relics and the establishment of a shrine at La Motte in the southeast of France around 1100 AD.[2] Victims of ergotism likely obtained relief at the shrine because of a change of diet. On modern French maps, there are two spots marked "La Motte" in the Dauphinée region. Synonyms for the disease, such as "mal des ardents", "ignis sacer" and "feu sacré", reflect this French influence. Houses of the Order of St. Anthony, devoted to the care of the victims of epidemic gangrene, apparently had flame-red walls. Alternatively, the "fiery" pain of peripheral vasospasm, which prompted sufferers to expose their limbs to the open air, and the subsequent development and bloodless separation of charcoal-like lesions on the extremities (i.e., dry gangrene) may also be responsible for the name of the disease, St. Anthony's fire.

In 1676, it was proven that epidemic gangrene was caused by the sclerotium of the fungus *Claviceps purpurea* (Fig. 1), which grows on rye, particularly in wet weather, and is now subject to strict public health regulations. Despite these, the problem has persisted into the 20th century.

Isolated instances of morbid vasospasm, on the other hand, may occur through the use of ergot preparations for treating migraine. They tend to follow one of three patterns:[3]

- Long-term ingestion with gradual increase to toxic levels.
- Acute excessive ingestion.
- Acute idiosyncrasy to a small dose.

The first pattern is the most common, and ergotamine usually is the causative agent.

Methysergide, a semisynthetic drug that is structurally related to the natural ergot compound, has a relatively weak

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Accepted for publication Jan. 2, 1987

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vasoconstrictive action and is less often implicated in vasospasm than ergotamine. Methysergide has some pharmacologic antiserotonin activity, which may account for its clinical prophylactic value against migraine. Moreover, vasospasm with methysergide has been overshadowed in importance by a graver complication — retroperitoneal fibrosis.[4] The following report describes a case of methysergide-induced vasospasm, documented both by angiography and by noninvasive studies, which suggest that significant ergot-like activity is present in methysergide, in spite of synthetic alteration.

Case Report

A 48-year-old woman was referred with a 1-month history of rest pain in the feet. One year before, pentoxifylline (Trental) had been prescribed for claudication, primarily in the left calf. For 2 years she had been taking methysergide as prophylaxis for migraine headache. Dosage ranged up to 16 mg/d, but she had two 3-month periods of abstinence on the advice of her physician. At the time of referral, however, she was taking close to her maximum dose. She had stopped smoking 5 years before. Her history included hysterectomy for fibroids in 1966 and an allergy to codeine.

The patient was a tired, anxious woman of medium build, who was having much difficulty walking. Carotid pulses were normal without bruits, femoral pulses were weak with bruits and there were no distal pulses in the lower limbs. The feet were pale, cold and slightly mottled, with elevation pallor and dependent rubor; ankle pressure was 30 mm Hg on the right, with no recordable Doppler signal on the left. Angiography (Fig. 2) demonstrated severe symmetrical narrowing in the superficial femoral and popliteal arteries, and multiple areas of occlusion in the distal leg vessels.

Methysergide was discontinued. Within 48 hours the feet were warm and pink, with bounding pulses and ankle pressure of 150 mm Hg (brachial pressure 145/80 mm Hg). Angiography was not repeated. On follow-up 2 months later, ankle pressures were normal, although there was some numbness in the distal part of the right foot. She was having intermittent nocturnal headaches, satisfactorily controlled with ice packs.

Discussion

Because, historically, epidemic gangrene involved such a large population, the epidemiology of ergotism was difficult to define. It became apparent that poor farmers were particularly vulnerable and that wet weather could be followed by an outbreak. The fact that ergot loses its potency with storage probably also confused the picture. Once the connection was established between St. Anthony's fire and ergoty rye, preventive public health measures were introduced. They have not met with unmitigated success: between 1889 and 1890, Kortneff observed over 2000 cases of ergot poisoning.[5] A spectacular outbreak with convulsions, psychoses and death occurred in rural France in the early 1950s when a baker used contaminated illegal rye to circumvent taxation, and, as recently as 1979, an epidemic was reported in Ethiopia.[6] Agriculture Canada[7] grades rye on the basis of ergot content, from no. 1 (0.05%) to no. 3 (0.33%). Higher contents than this are labelled ergoty rye; this usually involves less than 1% of rye in a normal year but up to 36% in a wet year. Other cereal and feed grains that are less susceptible to ergot infection than rye have similar limitations imposed by the Canadian Grain Commission.

The effect of ergot in pregnancy has been known for at least 2000 years. A quote from the sacred book of the Parsees, written between 400 BC and 300 BC, translates as follows: "Among the evil things created by Angro Maynes are noxious grasses that cause pregnant women to drop the womb and die in childbed".[1] The Moorish physician Avicenna (980–1037) apparently knew of ergot's potential, although there is no direct mention of it in his writings. In 1582, Lonicerus wrote of the painful effect of ergot on the uterus. In the early 1800s, Stearns, Akerly and Spalding described the use of ergot in parturition, and by 1822 an enquiry into its misuse resulting in stillbirth was conducted for the Medical Society of New York.[5]

In 1884, Dr. William H. Thomson of New York City described the use of fluid extract of ergot for frequent periodic headache.[8] The dosage was 2 to 4 mg orally or rectally every hour for 3 hours or until effective, accompanied by quinine for nausea. According to Dr. Thomson,

FIG. 1—Three ears of rye on left of this early 19th-century drawing contain ergots, sclerotia of fungus *Claviceps purpurea*. First two ears contain great number of ergots, more detailed examples of which are shown in detached ergots. Third ear of stout rye contains only one large ergot. Ear at right, which also bears one ergot, is wheat, grain rarely contaminated by fungus. (From Prescott, O Jr: *A Dissertation on the Natural History and Medicinal Effects of the Secale Cornutam, or Ergot*, Cummings and Hilliard, Boston, 1813).

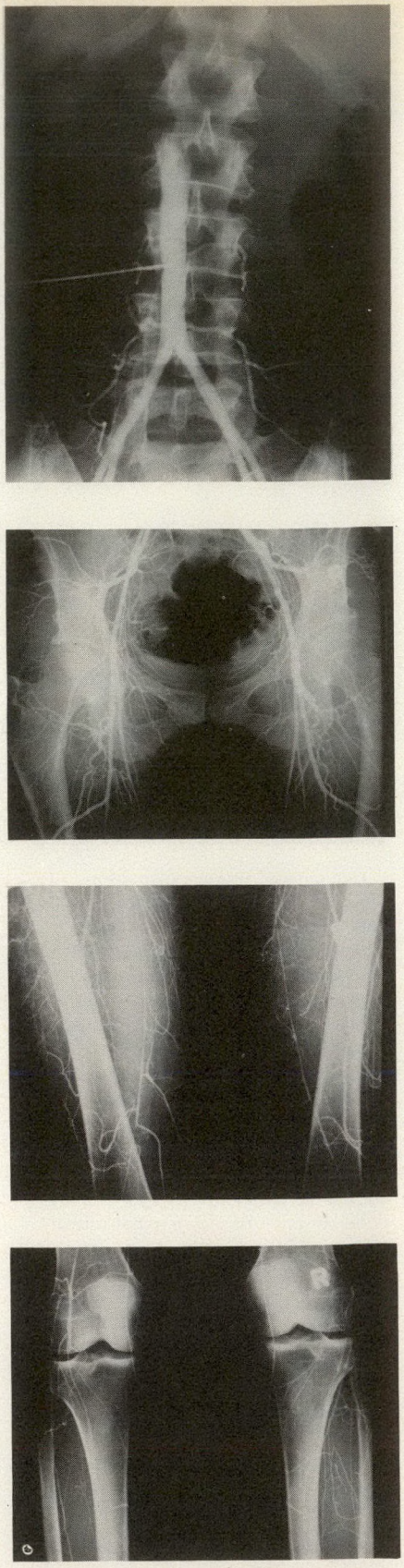

FIG. 2—Four films of translumbar aortogram demonstrate typical picture of ergot-induced vasospasm: symmetrical and diffuse narrowing, principally in superficial femoral, popliteal and limb arteries. Collateral vessels are frequently larger than main channels. Aorta and iliac arteries appear normal.

it "rarely fails". Twentieth-century reports of ergotamine use in migraine appeared first in the French[8] and German[5] literature, but the definitive proposal for the mechanism of ergot's beneficial effect was by Graham and Wolff[9] in 1938: the long-acting vasoconstriction produced by ergot prevents the vasodilatation of arteries of the scalp, which gives rise to the pain of migraine. *Harrison's Principles of Internal Medicine*[10] cites ergotamine as the principle therapeutic agent for aborting an acute attack of migraine, and methysergide as the most useful drug in the prophylaxis of migraine. With such a testimonial from one of the most popular and widely read medical textbooks in the world, one might infer extensive prescription of ergot compounds.

Some of the most important investigations related to the actions and chemistry of the ergot alkaloids were carried out by Dale[11] and coworkers in the early 1900s. Using the receptor protection technique, Innes[12] demonstrated that ergotamine acts both as an α-adrenergic antagonist and as a partial agonist, in a variety of in-vitro animal smooth-muscle preparations. The α-adrenergic blocking effect was confirmed by Mikkelsen and associates[13] who showed that ergotamine could block the effect of norepinephrine in human in-vitro mesenteric arteries harvested at cancer surgery. The partial agonist activity has been corroborated both in migraine patients and in normal volunteers,[14] in whom there is a reproducible fall in ankle-to-brachial pressure ratio with intravenous administration of 0.5 mg of ergotamine.

Goodman and Gilman[1] have described ergot as a "veritable treasure house of pharmacology". As well as ergot, the sclerotium of *Claviceps purpurea* contains amines such as histamine, tyramine and acetylcholine; also quarternary ammonia bases, amino acids, carbohydrates, sterols, glycerides and inorganic compounds. Ergot itself can be classified as follows: amino-acid alkaloids, amine alkaloids, and dihydrogenated compounds of the above.

Ergotamine is an amino-acid alkaloid, whereas methysergide, which is more easily absorbed through the gastrointestinal tract, is an amine alkaloid (Fig. 3). Dihydrogenation of ergotamine reduces its vasoconstrictive effect. All these compounds are structurally related to lysergic acid diethylamide (LSD).

The problem of iatrogenic ergotism, particularly in migraine sufferers, was first brought to the attention of the medical community by Labbe and colleagues in 1929.[5] Since then, over 100 case reports have appeared describing morbid vasospasm with the therapeutic use and abuse of ergot compounds.[15] Table I outlines documented sites of involvement. A partial review of these case reports suggests that the majority of patients recover completely. A few suffer minor tissue loss, the rare patient requires a major amputation, and when death ensues there is a complicating factor such as recent major surgery. In a few cases, such as the one we described, the only treatment was withdrawal of the medication, but most physicians have felt obliged to institute at least some additional care (Table II). Since all these reports are anecdotal, the only control has been in patients in whom a constant intravenous infusion of nitroglycerin[16] or nitroprusside[17] has been interrupted with return of symptoms and signs. Otherwise, it is impossible to determine whether the relief of vasospasm is spontaneous or treatment-induced.

Migraine headache is an extremely common phenomenon, and its management can be time-consuming, frustrating and moderately dangerous, as illustrated by this review. More recently there has been a trend to the use of β-blocking agents[18] and calcium channel blockers[19] in migraine therapy, although the sale of ergot preparations seems to be continuing relatively unabated (Sandoz Ltd, Montreal: Personal communication, 1986). Thus, the potential for St. Anthony's fire is still with us, and likely will be for the forseeable future. It is important that the practising physician be aware of morbid vasospasm from ergot drugs, so that disasters can be averted.

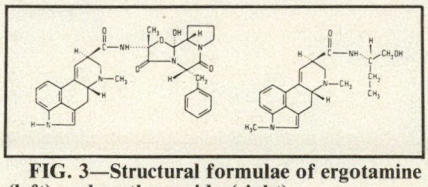

FIG. 3—Structural formulae of ergotamine (left) and methysergide (right).

Table I—Iatrogenic Ergotism: Sites of Involvement[15]

Carotid vessels
Retina
Upper limb
Coronary vessels
Mesentery
Kidney
Lower limb

Table II—Treatments for Iatrogenic Ergotism[15]

Vasodilatation (pharmacologic)	Anticoagulation
α-adrenergic block	Heparin
Phenoxybenzamine	Streptokinase
Phentolamine	Dextran (?)
Tolazoline	
Prazosin	
Reserpine	
β-adrenergic block	
Propranolol	
Direct action	Volume expansion
Papaverine	Dextran
Nitroglycerin	Mannitol
Nitroprusside	Crystalloid
Calcium channel block	Vasodilatation (mechanical)
Nifedipine	Fogarty
	Grüntzig
Ganglionic block	
Epidural bupivacaine	
Cervical ganglionectomy	
Lumbar sympathectomy	

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Dalacin® C Phosphate Sterile Solution

(clindamycin phosphate)

Antibiotic

Recommended Applications

Action: Clindamycin exerts its antibacterial effect by causing cessation of protein synthesis and also by causing a reduction in the rate of synthesis of nucleic acids.

Indications: Dalacin C Phosphate (clindamycin phosphate) is indicated for the treatment of infections where the oral route is not indicated or feasible.

Dalacin C Phosphate is indicated in the treatment of serious infections due to sensitive anaerobic bacteria, such as Bacteroides species, peptostreptococcus, anaerobic streptococci, Clostridium species and micro-aerophilic streptococci.

Dalacin C Phosphate is also indicated in serious infections due to sensitive Gram-positive organisms (staphylococci, including penicillinase-producing staphylococci, streptococci and pneumococci) when the patient is intolerant of, or the organism resistant to other appropriate antibiotics.

Contraindications: The use of Dalacin C Phosphate (clindamycin phosphate) is contraindicated in patients previously found to be hypersensitive to this compound, the parent compound, clindamycin, or clindamycin palmitate. Although cross-sensitization with Lincocin® (lincomycin hydrochloride) has not been demonstrated, it is recommended that Dalacin C Phosphate not be used in patients who have demonstrated lincomycin sensitivity.

Until further clinical experience is obtained, Dalacin C Phosphate is not indicated in the newborn (infants below 30 days of age), or in pregnant women.

Warnings: Some cases of severe and persistent diarrhea have been reported during or after therapy with Dalacin C Phosphate (clindamycin phosphate). This diarrhea has been occasionally associated with blood and mucus in the stools and has at times resulted in acute colitis. When endoscopy has been performed, some of these cases have shown pseudomembrane formation.

If significant diarrhea occurs during therapy, this drug should be discontinued or, if necessary, continued only with close observation. Significant diarrhea occuring up to several weeks post-therapy should be managed as if antibiotic-associated.

If colitis is suspected, endoscopy is recommended. Mild cases showing minimal mucosal changes may respond to simple drug discontinuance. Moderate to severe cases, including those showing ulceration or pseudomembrane formation, should be managed with fluid, electrolyte, and protein supplementation as indicated. Corticoid retention enemas and systemic corticoids may be of help in persistent cases. Anticholinergics and antiperistaltic agents may worsen the condition. Other causes of colitis should be considered.

Studies indicate a toxin(s) produced by Clostridia (especially Clostridium difficile) may be a principal cause of clindamycin and other antibiotic-associated colitis. These studies also indicate that this toxigenic Clostridium is usually sensitive *in-vitro* to vancomycin. When 125 mg to 500 mg of vancomycin were administered *orally* four times a day for 5 - 10 or more days, there was a rapid observed disappearance of the toxin from faecal samples and a coincidental recovery from the diarrhea.

It should be noted that serious relapses have occurred up to one month after apparently successful treatment. A relatively prolonged period of continuing observation is therefore recommended.

Precautions: Dalacin C Phosphate (clindamycin phosphate), like any drug, should be prescribed with caution in atopic individuals. Dalacin C Phosphate must be diluted for intravenous administration. (See Dosage and Administration)

The use of antibiotics occasionally results in overgrowth of nonsusceptible organisms - particularly yeasts. Should superinfections occur, appropriate measures should be taken as dictated by the clinical situation.

As with all antibiotics, perform culture and sensitivity studies in conjunction with drug therapy.

Since abnormalities of liver function tests have been noted occasionally in animals and man, periodic liver function tests should be performed during prolonged therapy. Blood counts should also be monitored, during extended therapy.

Dalacin C Phosphate may be used in anuretic patients. Since the serum half-life of clindamycin in patients with impaired hepatic function is greater than that found in normal patients, the dose of Dalacin C Phosphate should be appropriately decreased. Hemodialysis and peritoneal dialysis are not effective means of removing the compound from the blood. Periodic serum levels should be determined in patients with severe hepatic and renal insufficiency.

Adverse Reactions: ***Local***

(a) *Intramuscular Injections:* Of 404 patients treated with Dalacin C Phosphate (clindamycin phosphate) intramuscularly (with a solution containing 150 mg/ml), six (1.5%) demonstrated local reactions as follows: Two complained of pain at the injection site, two demonstrated induration at the injection site and two developed sterile absesses.

(b) *Intravenous Infusions:* Of 192 patients treated with Dalacin C Phosphate by intravenous infusion, 14 (7.3%) demonstrated local reactions. Eleven patients developed superficial thrombophlebitis and one patient developed both superficial and deep thrombophlebitis. The majority of these cases developed in conjuction with the use of indwelling I.V. catheters and it is difficult to know how much the drug contributed to the irritation. Two patients developed localized erythema, swelling and pain at the site of the infusion.

Systemic Side Effects: Twenty-eight patients of 596 treated with Dalacin C Phosphate (clindamycin phosphate) by either the intramuscular or intravenous routes developed systemic side effects as follows:

	Number of Patients
Rash	7
Urticaria	1
Pruritus	1
Fever, Leucocytosis	1
Nausea, with or without vomiting	1
Diarrhea (See also under "Warnings")	4
Hypotension	1
Hypertension	1
Shortness of Breath	1
Superinfection*	4
Cardiac arrest**	1
Bad or bitter taste in mouth	5

* Superinfection is a complication of antibiotic therapy in general and is not necessarily a true side effect of clindamycin phosphate.

** Due to underlying myocarditis in this patient.

Clinical and Laboratory Findings: Patients treated during clinical trials of Dalacin C Phosphate (clindamycin phosphate) were followed with clinical laboratory tests, including complete hematology, urinalysis and liver and kidney function tests. Some of these tests were abnormal initially and returned to normal during therapy with Dalacin C Phosphate, while others were normal initially and became abnormal, during therapy. Overall evaluation of clinical laboratory values in these patients does not indicate that Dalacin C Phosphate therapy has a toxic effect on the hematopoietic, hepatic or renal systems. Transient elevations of serum transaminases have occured in some patients, but other liver function tests (alkaline phosphatase, serum bilirubin) have not shown any tendency to increase and there have not been clinical signs of drug-induced hepatic toxicity.

Symptoms and Treatment of Overdosage: No cases of overdosage have been reported. No specific antidote is known. Doses as high as 1200 mg every six hours (4800 mg/day) by infusion for five days have been given without adverse effects.

DOSAGE AND ADMINISTRATION

Adults

Intramuscular Injection: 600 mg/day in 2 equal doses.
Moderately severe infections: 600 to 1200 mg/day in 2 or 3 equal doses.
Severe infections: 1200 to 2400 mg/day in 2, 3 or 4 equal doses. Intramuscular injections of more than 600 mg into a single site are not recommended.

Intravenous Administration: Dalacin C Phosphate (clindamycin phosphate) must be diluted prior to I.V. administration to a dilution of 300 mg in 50 mL of diluent (6mg/mL) or more, and infused in not less than 10 minutes. Administration of more than 1200 mg in a single 1 hour infusion is not recommended. Dalacin C Phosphate should not be injected intravenously undiluted as a bolus.

Moderately severe infections: 900 to 1800 mg/day by continuous drip or in 2 or 3 equal doses, each infused over 20 minutes or longer.

Severe Infections: 1800 to 2700 mg/day by continuous drip or in 3 or 4 equal doses, each infused over 20 minutes or longer. In life-threatening infections, doses of 2700 to 4800 mg/day by continuous drip or in 3 or 4 equal doses each infused over 20 minutes or longer may be given.

Dilution and infusion rates:

Dose	*Diluent*	*Time*
300 mg	50 mL	10 min.
600 mg	100 mL	20 min.
900 mg	150 mL	30 min.
1200 mg	200 mL	45 min.

Alternatively, drug may be administered in the form of a single rapid infusion of the first dose followed by continuous I.V. infusion as follows:

To maintain serum clindamycin levels	*Rapid infusion rate*	*Maintenance infusion rate*
Above 4 mcg/mL	10 mg/min. for 30 min.	0.75 mg/min.
Above 5 mcg/mL	15 mg/min. for 30 min.	1.00 mg/min.
Above 6 mcg/mL	20 mg/min. for 30 min.	1.25 mg/min.

Children: (Over one month of age)

Intramuscular injection: 10 to 15 mg/kg/day in 2, 3 or 4 equal doses.
Moderately severe infections: 15 to 20 mg/kg/day in 3 or 4 equal doses.
Severe infections: 20 to 30 mg/kg/day in 3 or 4 equal doses.

Intravenous Administration:
Moderately severe infections: 15 to 25 mg/kg/day by continuous drip or in 3 or 4 equal doses, each infused over 20 minutes or longer.

Severe infections: It is recommended that children be given no less than 300 mg/day regardless of body weight. (Dilute Dalacin C Phosphate Sterile Solution in the same manner as for adults.)

Dilution and Compatibility:
4 mL (600 mg) Dalacin C Phosphate when diluted with 1000 mL of the following commonly used infusion solutions was found to be physically compatible and demonstrated no significant change in pH or antimicrobial potency over a period of 24 hours:

Sodium chloride injection
Dextrose 5% in water
Dextrose 5% in saline
Dextrose 5% in Ringer's Solution
Dextrose 5% in half-strength saline plus 40 mEq potassium chloride
Dextrose 2½% in Lactated Ringer's Solution (Hartmann's Solution)

Dalacin C Phosphate was not stable when added to Dextrose 5% in water plus vitamins. Therefore it is not recommended that Dalacin C Phosphate be mixed with any infusion solution containing B vitamins.

Supplied:
Dalacin C Phosphate contains the following per mL of sterile solution:
Clindamycin phosphate equivalent to clindamycin base 150 mg
Benzyl alcohol 5 mg
Disodium edetate 0.5 mg
Water for injection q.s.
When necessary the pH is adjusted with sodium hydroxide and/or hydrochloric acid to maintain a pH range of 5.5 to 7.0.
Dalacin C Phosphate is available in 2 mL and 4 mL ampoules.

NOTE: Do not store below 15°C.

Product Monograph available upon request.

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R191

MARLEX MESH IMPROVES THE RESULTS OF INCISIONAL HERNIA REPAIRS. P. Makarewicz, M. Al-Shehri, J. Freeman. Division of General Surgery, Ottawa General Hospital, University of Ottawa, Ottawa, Ont.

One percent of all abdominal surgery is complicated by a postoperative hernia and 8% of such repairs fail. Significant defects in muscle and fascia or infection cause failure after simple closure. Reinforcement with prosthetic materials may reduce recurrence but increases risks. We established a hernia follow-up clinic to study recurrence and complications in 116 patients with massive primary or recurrent hernias repaired with (60) or without (56) Marlex. There were 68 women and 48 men aged 54 ± 14. The hernia was incisional in 91/116 (78%); 39 of these 91 (34%) were recurrent. There were 58 upper, 41 lower abdominal hernias, 8 inguinal, 12 umbilical and 5 others. Thirty-six hernias were massive (greater than 8 × 8 cm). Fifty-nine patients were obese, 25 had diabetes and 65 smoked. Postoperative drains were placed in 64/116; local antibiotics were used in 43/116 and systemic antibiotics in 60/116. Patients were followed for 18 – 40 months. In the Marlex group, 10/60 (17%) complained of pain without objective findings while at least half of these were proven secondary to Prolene sutures subsequently removed. Two patients developed wound sinus necessitating removal of Marlex. Recurrent hernia occurred in 9/60 (15%). In those repaired *without* Marlex, 9/56 (16%) complained of pain and recurrence occurred in 22/56 (39%). There was a strong correlation between recurrence versus obesity, diabetes or previous surgery.

Marlex mesh repair of abdominal hernias is advisable for all large and/or recurrent hernias or in the presence of obesity. The recurrence rate with Marlex is lower compared to primary repair (15 versus 39%). Incisional pain is identical in patients with and without Marlex and may be secondary to Prolene knot granulomas.

R192

PRELIMINARY RESULTS OF COLOANAL ANASTOMOSIS. Denis Bernard, Daniel Tassé, Ramses Wassef, Stephen Morgan. Department of Surgery, University of Montreal, Division of Gastrointestinal Surgery, Hôpital St-Luc, Montreal, PQ

Coloanal anastomosis is the ultimate procedure to preserve the patients' sphincters and avoid a permanent colostomy. Carcinomas (CA) of the midrectum and sometimes of the lower third do not require excision of the pelvic floor and sphincters for cure. Coloanal anastomosis was achieved in 29 patients after rectal excision for CA in 23, for adenomas in 4 and for 2 other conditions. Dukes' staging of the CA were A: seven, B: six, C: nine and resection was palliative in one. The mean distance from the anus to the CA was 6.4 cm. A temporary defunctioning colostomy was established in all. There were no deaths and 52% (n: 15) had postoperative complications (major: six, minor: nine). Mean follow-up is now 29 months (9 – 61 months). Only one had pelvic recurrence; one is alive with metastatic disease and one died of an unknown cause at 30 months with no evidence of recurrent disease. All others are alive and well. Colostomy was closed in all but two. Stenosis was the most common long-term problem: it occurred in 13 and required more than one dilatation in 8. Six months after the procedure the mean number of daily bowel movements is 4.3; 95% are continent to solid stools and 16% are incontinent to liquid stools or gas, but only one would prefer re-establishment of a colostomy. In selected cases with little or no extramural spread, we feel a resection and coloanal anastomosis is a good alternative with acceptable functional results and a low rate of recurrent tumour comparable to complete rectal excision.

R193

PREVENTION OF INTRA-ABDOMINAL ABSCESSES WITH FIBRINOLYTIC AGENTS. O.D. Rotstein, J. Quinto, J. Kao. Department of Surgery, University of Toronto, Toronto, Ont.

Fibrin deposition during peritonitis predisposes to subsequent abscess formation by protecting bacteria from host defences. To determine whether the use of intraperitoneal (ip) fibrinolytic agents could prevent ip abscess formation, Wistar rats were implanted intraperitoneally with fibrin clots (100 mg/dl) containing either *B. fragilis* (BF, 2×10^9/clot) alone or *E. coli* (EC, 2×10^6) plus BF (2×10^8) and beginning at surgery, animals received daily 5 ml ip injections of either phosphate-buffered saline (PBS), trypsin (TRYP 1.0 mg/ml or 0.5 mg/ml) or recombinant tissue plasminogen activator (t-PA, 0.25 mg/ml, Genentech Co). Abscess formation and bacterial numbers were assessed at 5 days.

Treatment	*B. fragilis*[a] Abscess formation	*E. coli* + *B. fragilis* Mortality	Abscesses
PBS	87% (n = 15)	8.3% (n = 12)	90%
TRYP (1 mg/ml)	20%[b] (n = 20)	25% (n = 12)	11%[b]
TRYP (0.5 mg/ml)	60% (n = 5)	16% (n = 12)	18%[b]
t-PA	0% (n = 15)	ND	ND

a: No mortalities occurred, b: p 0.01 versus PBS control, ND: not done.

The viable bacteria (log CFU/abscess) in BF abscesses were 7.7 ± 0.4 (n = 6) in the PBS group versus 4.8 ± 2.5 (n = 4) in the TRYP 1 mg/ml group. In-vitro studies demonstrated *no* direct microbicidal activity of TRYP or t-PA on BF or EC, suggesting that enhanced bacterial clearance was responsible for reduced abscesses and bacterial numbers. Controlled fibrinolysis at operation may be a useful adjunct to surgery and antibiotics in preventing postoperative ip abscess formation.

R194

LOBULAR CARCINOMA OF THE BREAST. W.J. Orrom, B.M. Mielke, O.G. Thurston. Department of Surgery and Department of Pathology, University of Alberta Hospital, Edmonton, Alta.

Lobular carcinoma of breast has distinct biological differences from other breast cancers affecting treatment, follow-up and prognosis. This study is a review of our experience with 22 cases of lobular carcinoma in situ and 136 cases of invasive lobular carcinoma seen at the University of Alberta Hospital, 1955 – 1985.

Lobular carcinoma totalled 10% of all breast cancers in this period. When compared to patients with invasive duct carcinoma, those patients with invasive lobular carcinoma were younger — 55 versus 58 years ($p < 0.01$) and had a higher incidence of bilaterality — 20% versus 5% ($p < 0.001$). Actuarial survival was better for invasive lobular than invasive duct cancers ($p < 0.001$). The mean age of patients with lobular carcinoma in situ was 7 years younger than patients with invasive lobular carcinoma ($p < 0.01$) whereas patients with duct carcinoma in situ were similar in age to patients with invasive duct carcinoma. The surgical treatment of invasive lobular carcinoma was evenly distributed between radical, modified radical and simple mastectomy. There were no survival differences between the three groups.

The results of this study indicate a biological behaviour to lobular carcinoma that is significantly different than the more common duct carcinoma. The younger age, improved prognosis and higher frequency

of bilaterality in patients with lobular carcinoma have important implications for treatment and follow-up. The 7-year lag between the in-situ and invasive forms of lobular cancer is strong evidence that this is a precursor lesion rather than a high-risk marker of invasive cancer. This is in contrast to duct carcinoma, where the in-situ and invasive forms appear virtually simultaneously.

R195

ENHANCEMENT OF CELLULAR KINETICS IN THE PROXIMAL SMALL INTESTINE OF THE RAT FOLLOWING RUMENECTOMY. R. Odze, B. Mitmaker, P.H. Gordon. Lady Davis Research Institute, Department of Surgery, Sir Mortimer B. Davis-Jewish General Hospital, McGill University, Montreal, PQ

To determine the effect of excision of the rat rumen (forestomach) on the cellular kinetics of the proximal small intestine, young male Sprague–Dawley rats were randomly assigned to receive either control gastrotomy (Group A) or rumenectomy (Group B). After preliminary results indicated a proliferative response in Group B, the following two surgical groups were added to differentiate if this effect is dependent on luminal or extraluminal factors: pyloric ligation and gastrojejunostomy (Group C) and rumenectomy, pyloric ligation and gastrojejunostomy (Group D). Fourteen days following surgery each animal received an intraperitoneal injection of ^{3}H-thymidine and was sacrificed 1 hour later. Radioautographic slides were prepared from sections of duodenum and jejunum for determination of crypt column cell counts (CCC), villous column cell counts (VCC), crypt labelling index (Li) and proliferative index (Pi). In the duodenum, the results showed a CCC of 31.5 ± 0.3 (SEM) in Group A and 40.0 ± 0.4 in Group B ($p < 0.01$). Bypassed duodenum from Groups C (32.5 ± 0.3) and D (35.9 ± 0.4) showed no significant change in this parameter. In the efferent jejunum, the CCC showed no difference between Group A (30.9 ± 0.3) and B (33.1 ± 0.2) but significant increases were observed in both Groups C (40.3 ± 0.5) and D (46.9 ± 0.4) when compared to control ($p < 0.01$) and to each other ($p < 0.05$). The Li, Pi and VCC showed little variation between any surgical group. These results indicate that the intestinal mucosa in direct proximity to the stomach exhibits a luminal dependent crypt cell hyperplastic response following excision of the rumen.

R196

RECONSTRUCTION OF THE TOTAL THORACIC ESOPHAGUS WITH REVASCULARIZED JEJUNUM: AN EXPERIMENTAL MODEL. J. Hobson, G. Pliagas, G. DeRose, R. Finley. Division of General Surgery and Division of Vascular Surgery, Victoria Hospital, University of Western Ontario, London, Ont.

Historically, total esophageal reconstruction with pedicled jejunum has proved to be unreliable. Ischemia of the proximal graft leads to anastomotic leakage and increased morbidity and mortality. Augmentation of the blood supply to the proximal jejunum should prevent these complications.

A canine model was used to assess this technique of total esophageal reconstruction with autologous revascularized jejunum. Pedicled, isoperistaltic jejunum is passed retrosternally to the neck and anastomosed to the cervical esophagus and distally to the stomach. In the experimental groups ($n = 15$) the proximal jejunal arcade is revascularized by anastomosing the artery to the side of the common carotid artery and vein to side of external jugular vein. The control group ($n = 6$) had no revascularization.

Successful revascularization occurred in 14/15 experimental animals. The esophagojejunal anastomosis leaked in 5/14 experimental (no strictures) and 6/6 control animals ($p < 0.01$). None of the control animals were able to sustain an oral diet.

Using this technique, 10 other dogs ($n = 10$) underwent total thoracic esophageal reconstruction with revascularized jejunum. Regional mesenteric blood flow in the jejunum was measured using the radioactive microsphere technique (I-125 and Cr-51). Blood flow to the jejunum at the esophagojejunal anastomosis without revascularization measured 0.011 ± 0.009 ml/g/min. With revascularization jejunal blood flow at the anastomosis measured 0.415 ± 0.129 ml/g/min ($p < 0.001$).

Revascularization of the proximal jejunum in the neck does result in a statistically significant increase in blood flow to the jejunum at the esophagojejunal anastomosis and decreases the incidence of esophagojejunal anastomotic leakage in the neck.

R197

THE VALUE OF CURATIVE RESECTION FOR CARCINOMA OF THE ESOPHAGUS. F. Rubens, F. Shamji, S. Brown, H. Sachs. Division of Cardiothoracic Surgery, University of Ottawa, Ottawa Civic Hospital, Ottawa, Ont.

We managed 71 patients with esophageal cancer between December 1983 and February 1987. Thirty-two had squamous cell and 39 had adenocarcinoma of which 13 arose in Barrett's esophagus. Using predefined criteria, 45 patients were considered operable. Thirty-seven of these were deemed resectable; 29 of these were curative and 8 were palliative.

Twenty-one of the 29 patients who had curative resection are alive 1 to 36 months postoperatively (mean 20 months) whereas 6 of the 8 patients who had palliative resection died 1 to 13 months (mean 4.8 months) after surgery. Four of 45 operated patients died (8.9%); 3 of these were amongst the palliative resection group. However, only 1/29 patients (3%) resected for cure died. The eight patients in the operable but unresectable group lived 2 to 24 months (mean 9). Eight of 26 inoperable patients are still alive 2 to 27 months (mean 11.5) after diagnosis.

Of the 32 patients with squamous cell cancer, 9 were inoperable or unresectable and were treated with 5-FU and radiation. Four of nine of these patients have survived longer than 13 months.

Conclusions: Curative resection of carcinoma of the esophagus results in mean 20-month survivorship which is superior to palliative or non-resectional therapy and has an operative mortality of 1/29 (3%). Conversely, palliative resection for carcinoma of the esophagus carries a high operative mortality with a low survival. Combination chemoradiation therapy provides reasonable palliation for patients with inoperable squamous cell lesions.

R198

PALLIATIVE ND:YAG LASER THERAPY FOR OBSTRUCTING ESOPHAGEAL CANCER. K. Woolfson, C.D. Mercer. Hotel Dieu Hospital, Kingston, Ont.

Nine patients with unresectable carcinoma of the esophagus (one hypopharyngeal, one Barrett's adenocarcinoma, two squamous cell midesophageal and five adenocarcinoma of the gastroesophageal junction), ranging in age from 41 to 87 years (mean 68 years) underwent palliative vaporization of the tumour with the Nd:YAG Laser. Fourteen treatments were required in these patients. A maximum of 7870 joules (J) of energy was used in a single session (mean 4400 J). Pulse settings were energy 40 watts, pulse duration 0.5 seconds. Operative time ranged from 5 to 48 minutes. No intraoperative complications occurred. Postoperative morbidity occurred in two patients having previously undergone radiotherapy. (Procedure-related morbidity rate was 14%.) One developed transient laryngeal edema requiring intubation and one developed a tracheoesophageal fistula controlled with an internal esophageal stent. Mean follow-up is 3 months (range 2 - 6 months). Significant improvement of dysphagia occurred in seven patients and one patient with a bleeding tumour was controlled with the laser. Two patients had minimal transient relief of dysphagia. Two patients died of metastatic disease during follow-up. The Nd:YAG Laser is an effective alternative to internal esophageal stenting and palliative resection or bypass in patients with obstructing esophageal malignancy.

R199

NORMOGLYCEMIA AFTER IMPLANTATION OF PURIFIED ISLETS OF LANGERHANS IN DOGS. G.L. Warnock, R.V. Rajotte. Department of Surgery and Department of Medicine, University of Alberta, Edmonton, Alta.

To promote clinical transplantation, effective methods for the isolation of purified islets will be needed. Canine pancreata removed at total pancreatectomy were cannulated via the ducts and perfused with collagenase solution (1900 U/ml) at 37°C until mucoid. The gland was dissociated into a tissue suspension by teasing and trituration. Islets were separated on ficoll with gradient densities of 1.045, 1.075 and 1.085, which reduced the mean packed cell volume from 37 ± 2 to 1.1 ± 0.4 ml (±SEM, $n = 15$). Counts of islets identified at microscopy revealed a recovery of 103.6 ± 12 × 10^3 per pancreas. Mean islet diameter was 128 ± 7 μm. Histology showed highly purified islets. A dose of ≥ 3200 purified islets/kg body weight was autoimplanted into splenic veins (six dogs) or embolized via the portal vein to the liver (five dogs). Portal venous pressure was unchanged in the former and rose transiently

by 3.4 ± 1.3 cm H_2O in the latter group. Mean fasting plasma glucose (PG, mg/dl) was 122 ± 4 at 1 week, 91 ± 6 at 1 month and 86 ± 4 at 6 months. During glucose tolerance tests, K values were nondiabetic (> 1.0) at 1.9 ± 0.2 (1 month) and 2.0 ± 0.3 (6 months). Six months after splenic implantation, basal splenic vein insulin was 20 ± 4 mU/L versus 4 ± 1 in the artery; PG rose to > 350 after splenectomy and immunohistology showed intrasplenic islets. In four other dogs, < 3200 islets/kg were refluxed into the spleen; PG rose to 235 ± 16 at 1 week. In three apancreatic controls, PG was 337 ± 9. These data define the critical number of purified dog islets of constant size which is necessary to induce prolonged normoglycemia. Sufficient pure islets can be collected from a single dog pancreas to reverse diabetes after implantation into the liver or spleen.

R200

THE OPERATING ROOM: AN UNEXPLOITED UNDERGRADUATE TEACHING RESOURCE. J.L. Provan, R. Cohen. Department of Surgery, University of Toronto, Toronto, Ont.

During the 1985-86 clerkship at the University of Toronto, 236 students completed the end-of-rotation Clerkship Assessment Form (CAF). The CAF contains 27 closed-ended (1 to 5 scale) items that assess the quality of the learning experience and the exposure to specific activities on all surgical clerkship rotations. Analysis of these data (correlation coefficients and stepwise multiple regression) revealed that six of the specified activities correlated well to moderately well with perceived success of the student's learning experience, i.e., quality of time spent in the OR (r = 0.68), amount of scut work the clerk had to do (r = 0.53), frequency of review of histories and physical examinations (r = 0.46), opportunity to follow patients admitted previously to the OR (r = 0.45), amount of responsibility for patient care (r = 0.41) and the opportunity to follow patients postoperatively (r = 0.33). Stepwise regression revealed that one activity, i.e., quality of time spent in the OR explained most of the variance on the dependent variable (perceived success of learning experience) for all surgical subspecialties: general surgery 45% (total variance explained 56%), neurosurgery 41% (total explained 61%), orthopedics 28% (total explained 51%), thoracic 43% (total explained 50%), urology 51% (total explained 65%), plastic surgery 53% (total explained 60%), and vascular surgery 44% (total explained 56%) at $p < 0.001$ (for all). The results of this study indicate that the quality of teaching students receive in the OR is important in determining the success of a surgical clerkship as a learning experience. Clerkship coordinators need to give more attention to developing the OR as a structured undergraduate learning experience.

R201

COMPLICATIONS OF HICKMAN–BROVIAC CATHETERS. M.J. Monaghan, R.F. Pace. Department of Surgery, Queen's University, Kingston General Hospital, Kingston, Ont.

The use of indwelling Silastic central venous catheters (Hickman–Broviac) has simplified many problems of long-term venous access. A review of 103 catheters in 79 patients (12 192 patient days) has disclosed a significant incidence of catheter-related morbidity, with occasional mortality. The indications for catheter use were chemotherapy administration in 48, total parenteral nutrition in 19 and fluid or antibiotic administration in 12 patients. Mean catheter use in successfully catheterized patients was 120.7 days (range 1 to 827 days) and 11 patients required more than one catheter to complete therapy. Although 69% (71/103) of catheters functioned satisfactorily until elective removal, patient death or study conclusion, only 45% (46/103) were free from complication. Catheter removal was necessary in 31% (32/103) because of complications which did not respond to treatment, the most common indications for premature removal being catheter occlusion (6), subcutaneous tunnel infection (5) and catheter sepsis (10).

Although tunnel sepsis usually required catheter removal (5/7), the more ominous onset of catheter sepsis could often be controlled with intravenous antibiotics, and catheter removal was avoided in 14/24 cases. Coagulase-negative staphylococci predominated in cases of catheter sepsis (16/20 cases). Postoperative hemorrhage, venous thrombosis and catheter sepsis each contributed to one patient death and resulted in significant morbidity in surviving patients. Despite the high incidence of complications associated with Hickman catheters, patient acceptance was high, and their use facilitated treatment in a high-risk patient group.

R202

RESTORATIVE PROCTOCOLECTOMY FOR ULCERATIVE COLITIS AND FAMILIAL POLYPOSIS — THE CALGARY EXPERIENCE. J.A. Heine (R-II), J.M. Langevin, R.M. Preshaw, W.D. Wong. Division of General Surgery, Department of Surgery, Faculty of Medicine, University of Calgary, Calgary, Alta.

Fifty-five patients with ulcerative colitis (51 patients) or familial polyposis (4 patients) have undergone restorative proctocolectomy employing either an S or J ileal pouch–anal anastomosis. There were no postoperative deaths. Major complications included small-bowel obstruction requiring laparotomy in two patients (4%) and pelvic abscess in three patients (5.5%). Minor complications included small-bowel obstruction managed conservatively in 10 patients (18%); "pouchitis" in 11 patients (20%); and anastomotic stenosis in 12 patients (22%), requiring anal dilatation under general anesthesia in 5 patients (9%).

Patients were followed up for a mean of 14.7 months. Fifty-four patients (98%) could defecate spontaneously and mean stool frequency was 6.1 ± 1.9 bowel movements per 24-hour period. Four patients (7%) were having nine or more bowel movements per 24-hour period. Major daytime incontinence was a complaint of two patients (4%). Functional outcome was judged to be good to excellent in 50 patients (91%). These results suggest that restorative proctocolectomy is a viable surgical alternative in selected patients requiring surgical management of ulcerative colitis or familial polyposis.

R203

SURGICAL TREATMENT OF CHRONIC PANCREATITIS (CP): 12 YEARS' EXPERIENCE. B.G. Karam, M.J. Wexler. Department of Surgery, Royal Victoria Hospital, McGill University, Montreal, PQ

Forty patients with cp underwent 42 operations between 1973 and 1984. Intractable pain or symptomatic cysts were the primary indications. Fifty percent were alcoholic, 23% diabetic and 18% had steatorrhea. All patients had ERCP or operative pancreatogram. Selection of procedure was tailored to ductal pathology with attempts to preserve pancreatic tissue. Late results were classified as A, B or C: "A" = complete relief of pain, no worsening of pancreatic function (pf); "B" = only occasional pain and/or controlled pf; "C" = failure to relieve pain or poorly controlled pf. Mean follow-up was 5.6 years. Three patients developed pancreatic carcinoma and four had late unrelated deaths. Perioperative mortality and morbidity rates were 2.5% and 15% respectively. Procedures and results are summarized:

	#	A	B	C
Whipple or total pancreatectomy	3	1	1	
40-80% distal resection ± retrograde drainage (Duval)	17	3	5	5
Lateral pancreatojejunostomy	5	2	1	1
Internal cyst drainage	8	5		1
Sphincteroplasty Wirsung	8	5		
Other	1		1	

Relief of pain without iatrogenic worsening of pf can be achieved with 80% success and minimal morbidity and mortality, using a selective approach based on the ductal pathology. Results are poorest when major resectional procedures are required.

R204

IMPROVED SURVIVAL WITH SURGICAL TREATMENT OF PANCREATIC ABSCESS. R.G. Keith, J.R. Brow, H. Miller, J. McKee, R. Reid. Department of Surgery, University of Toronto, Toronto, Ont.

Twenty-two consecutive patients with pancreatic abscess were treated by operation designed to preserve maximal pancreatic tissue. Infected pseudocysts and aseptic necrosis were excluded from this study. All patients presented with sustained sepsis and nutritional failure; 10 patients had single organ failure and 1 had multiple organ failure (MOF).

Accurate localization of septic foci was obtained by diagnostic imaging; CT scanning proved superior to ultrasound (sensitivity 90%:45%). Multiple abscesses were identified in 17 cases. Operation in each case was extensive débridement and drainage of all suppurative necrosis and abscess, specifically minimizing pancreatic resection. Postoperative intensive care was supplemented by mechanical ventilation in 9 cases and total parenteral nutrition in 15 cases.

There were only two deaths: 7 days from MOF; 60 days from myocardial infarction. Both had multifocal sepsis, preoperative organ failure but a short interval from diagnosis to operation (1:7 days).

Postoperative complications were common, correlating with multifocal sepsis, but not with interval from diagnosis to operation. Fifteen patients developed 31 complications; 7 required reoperation, 2 for residual abscesses.

Follow-up of pancreatic function in 20 survivors at mean duration of 54 months documented preservation of endocrine (16/20 nondiabetic) and exocrine (17/20) function, which correlated with intact whole organ on scanning (16/20).

Improved long-term survival is possible when débridement surgery conserving pancreatic parenchyma is effectively combined with intensive perioperative care.

R205

THE RESTING ENERGY EXPENDITURE OF THE MORBIDLY OBESE. L.K. Tin, M. Gagner, H.M. Shizgal, R.A. Forse, L.D. MacLean. Royal Victoria Hospital, McGill University, Montreal, PQ

Previous reports on the resting energy expenditure (REE) of the morbidly obese are inconsistent. This study was undertaken to determine the effects of obesity on the REE. In 18 morbidly obese patients, body composition was determined by multiple isotope technique, and the REE simultaneously measured by indirect calorimetry. Control data were obtained in 16 normally nourished volunteers. Resting energy expenditure was expressed as a function of body weight (BW), body surface area (BSA) and body cell mass (BCM). The degree of obesity was determined using an obesity index (OI) defined as body fat (FAT) per BCM representing the amount of fat supported for a given BCM.

	BCM	FAT	REE	REE/BW	REE/BSA	REE/BCM	OI
Control	24.6 ± 1.6	16.7 ± 1.3	1427 ± 74	21.6 ± 0.7	789 ± 24	56.6 ± 1.7	0.70 ± 0.05
Obese	30.7 ± 1.4*	61.2 ± 2.4*	1961 ± 77*	15.9 ± 0.5*	870 ± 6*	64.7 ± 2.4*	2.06 ± 0.10*

*Significantly different from control by unpaired Student's *t*-test.

Morbid obesity is associated with an increased REE not only due to an increased BCM but also and separately due to the degree of obesity. A multiple linear regression equation was developed and was found to be significant ($p < 0.0001$, $r = 0.89$).

$$REE = 139.3 \pm 47.0\ (BCM) \pm 180.5\ (OI)$$
$$(p < 0.0001) \quad (p < 0.001)$$

These data indicate that morbid obesity is associated with a hypermetabolic state which is apparent when REE is expressed as a function of BCM, the metabolically active component of the body. The REE is related to the size of the BCM and the degree of obesity.

R206

POST NISSEN SYNDROME. D.E. Low, C.D. Mercer, E.C. James, L.D. Hill. Virginia Mason Medical Center, Seattle, WA.

Introduction: Nissen fundoplication (NFP) is the most common antireflux operation performed in North America. We reviewed 305 patients who had undergone 356 previous antireflux procedures but required reoperation for recurrence or complication from 1964 to 1986. The 116 patients who required reoperation following NFP were specifically examined for etiology of failure and complications. *Results:* Reasons for reoperation included recurrent reflux ± stricture (80%), severe dysphagia (60%) and esophageal dysmotility (48%). Mean time interval between failed NFP and remedial surgery was 51.4 months, but mean time to onset of recurrent symptoms was only 12.8 months. Prior to the patient's original NFP, dysphagia was present in 23% compared to 60% of patients requiring remedial surgery. Analysis at operation demonstrated the cause of NFP failure to be "slipped Nissen" (48%), patulous repair (21%), complete (16%) and partial (13%) disruption, in addition to life-threatening complications including gastrobronchial, gastrocutaneous, gastropericardial and gastroaortic fistulas. Remedial surgery was individualized; however, most patients underwent Hill posterior gastropexy. Operative mortality was 2.6%. With a mean follow-up of 19 months, the overall results were rated excellent/good (86%), fair (11%) and poor (3%). *Summary:* We suggest three reasons for recurrent symptoms and complications following NFP. (1) The procedure is done blindly with no attempt at intraoperative LES calibration, (2) it relies on sutures placed in the esophageal muscularis, (3) the wrap is not anchored and therefore is more prone to migration. The popularity of the NFP should be tempered due to these weaknesses in technique and increased risk of recurrence and serious complications.

R207

SUTURELESS BOWEL ANASTOMOSIS USING ND:YAG LASER. C.D. Mercer, B. Pauli. Department of Surgery, Hotel Dieu Hospital, Queen's University, Kingston, Ont.

Phase I: Nd:YAG Laser produced tissue-welded bowel anastomoses in rabbits. A 0.5-cm enterotomy was closed using sutures or non-contact laser energy. Small-bowel anastomoses (n = 84) were performed 15 cm apart; bursting pressure (BP) of the occluded anastomotic segment was measured. Bursting pressures of laser anastomoses less than 300 J were weaker ($p < 0.001$) than sutured anastomoses. Bursting pressures of sutured and laser anastomoses using 300 - 500 J were similar. Phase II: Enterotomies were closed with sutures or laser at 400 J. All rabbits (n = 6) recovered without complication. At 7 or 14 days, all anastomoses were intact. Bursting pressures of sutured (mean 203 ± 7 mm Hg) and laser anastomoses were not different (mean 200 ± 10 mm Hg). Histology demonstrated fibrous union at 7 days but incomplete mucosal approximation with laser anastomosis. At 14 days, laser anastomoses were indistinguishable from normal bowel.

Technique	N	Energy (J)	Mean BP ± SD (mm Hg)
Sutures	10	—	65.6 ± 15.3
Non-contact laser	7	100	29.1 ± 6.5
	19	200	41.3 ± 11.9
	21	300	50.2 ± 20.9
	20	400	64.0 ± 25.7
	7	500	65.0 ± 25.8

Nd:YAG Laser tissue welding is an effective method of anastomosing small-bowel enterotomies in rabbits.

R208

VOLVULUS OF THE COLON: EARLY LOCALIZATION OF SITE AN ESSENTIAL STEP FOR PROPER MANAGEMENT. E. Farkouh, M. Allard, H. Atlas, R. Denis. Department of Surgery, Sacré-Coeur Hospital, University of Montreal, Montreal, PQ

This paper summarizes our experience with this uncommon and serious form of intestinal obstruction over a period of 12 years, pointing out the importance of early recognition of the bowel segment involved for the selection of the appropriate treatment. Forty patients with colonic volvulus form the basis of this study, 24 male and 16 female, average age 59 years. They were divided into three groups according to site: 1. *Sigmoid volvulus* (n: 24): Deflation by rectosigmoidoscopy was attempted on 20 patients. It was successful in 11 patients, and 5 of those had eventually elective surgery at a later date. Nine patients where deflation failed had emergency surgery. Early in the series four patients had emergency surgery without attempt of deflation endoscopy. The type of operation used: sigmoid resection and anastomosis in eight, Hartmann's procedure in five and simple detorsion in five. In this group the primary treatment recommended is deflation by rectosigmoidoscopy, with elective surgery at a later date. 2. *Cecal volvulus* (n: 13): All patients had emergency surgery, five had right hemicolectomy, six had detorsion and cecostomy, and two had cecopexy. 3. *Transverse colon volvulus* (n: 3): Two patients had colon resection and anastomosis; and one patient had resection and ileostomy.

Groups 2 and 3 should be recognized early and subjected to immediate surgery to avoid early necrosis and perforation. In the three groups, seven patients died postoperatively (mortality rate 17.5%).

To lower this high mortality rate associated with colonic volvulus, early localization of the bowel segment involved and the immediate appli-

cation of the appropriate treatment are essential. On the left side of the colon deflation endoscopy should be the primary treatment, while on the right side emergency surgery is the treatment of choice.

R209

IMMUNOSUPPRESSION WITH CYCLOSPORIN A (CsA) DOES NOT AFFECT RESISTANCE TO MURINE HEPATITIS VIRUS (MHV-3) IN A/J MICE. M. Abecassis, G.A. Levy, J.A. Falk, L. Makowka, R.E. Falk. Department of Medicine and Department of Surgery, University of Toronto, Toronto, Ont.

It has been recently suggested that CsA is associated with a lower incidence of viral infections when compared to conventional immunosuppressive therapy. These studies were designed to examine whether CsA alters innate T-cell dependent resistance to MHV-3 infection in the fully resistant A/J mouse. Mice, 6 – 8 weeks of age, were treated daily with cyclophosphamide (CY — 75 mg/kg), methylprednisolone (MP — 500 mg/kg) or CsA (50 mg/kg) and were infected with MHV-3. Trough serum CsA levels were measured by radioimmunoassay 12 hours (4830 ± 650 ng/ml) and 24 hours (628 ± 315 ng/ml) following administration, and CsA, at this dosage, prevented rejection of allogeneic skin grafts. Infected mice treated with CsA developed specific antibody (IgM and IgG) to MHV-3 and demonstrated no biochemical or histologic evidence of liver disease. In contrast, infected mice immunosuppreseed with either CY or MP developed both histologic and biochemical evidence of fulminant hepatitis associated with failure to produce antibody to MHV-3 and 100% mortality. These results suggest that CsA does not alter innate resistance to MHV-3 in A/J mice, whereas immunosuppression with either CY or MP results in conversion of resistance to susceptibility. We conclude that the increased incidence of viral infections associated with the use of CsA may be related to the concomitant use of other immunosuppressive agents.

R210

UTILITY OF INTRAOPERATIVE ULTRASONOGRAPHY IN THE SURGICAL TREATMENT OF HEPATIC TUMOURS. R. Lapointe, M. Lafortune, L. Lapointe, R. Déry, P. Lavoie. Department of Surgery and Department of Radiology, Hôpital Saint-Luc, Université de Montréal, Montreal, PQ

Intraoperative ultrasonography was performed in 14 patients with various liver tumours: 9 colorectal metastasis, 3 nodular focal hyperplasia, 1 hepatoma and 1 primary hepatic cystadenocarcinoma. All patients had preoperative work-up including ultrasonography of the liver as well as computerized tomography of the liver (13 patients) and selective hepatic arteriography (12 patients). At surgery, various kinds of liver surgery were carried out on these 14 patients after inspection, palpation and operative ultrasound examination of the liver had been performed; segmentectomy in 4, hepatic lobectomy in 4, subsegmentectomy in 2 and biopsy in 4. Intraoperative ultrasonography proved to be a useful method not only for revealing and permitting guided biopsy of unsuspected preoperative tumours deep in the hepatic parenchyma but also for establishing the exact relationship between the vascular structures and the tumour. It is therefore an important exploration mean for therapeutic decision in liver surgery.

R211

SEPTIC STRESS AND THE ADIPOCYTE BETA-1 ADRENERGIC RECEPTOR. D. St. Vil, M. Gagner, R.A. Forse. Royal Victoria Hospital, McGill University, Montreal, PQ

Catecholamine exposure results in down-regulation of beta-adrenergic receptors (decreased response to stimulation in vitro). The object of this study was to determine if there were differences in the down-regulation of beta-1 receptors of adipose tissue from septic patients who survived (SURV) and those who died (NON-SURV). The adipose tissue was biopsied from the abdominal wall and the intestinal mesentery during open abdominal dressing for peritonitis. Normal tissue was biopsied during elective surgery (Normal). Lipolysis was measured using an in-vitro dual isotope technique and expressed below as the lipolysis factor (LF). The beta-1 receptors were stimulated with isoproterenol (ISO). The maximal LF at any concentration was recorded as the V_{max}. The results are:

ISO	10^{-8}	10^{-7}	10^{-6}	10^{-5}	10^{-4}	V_{max}
Mesentery						
Normal	5.3 ± 2.7	22.1 ± 7.3	51.3 ± 9.0	50.7 ± 4.5	43.6 ± 5.8	62.1 ± 7.9
SURV	5.6 ± 5.6	26.4 ± 9.3	43.7 ± 25.3	38.3 ± 14.0	34.0 ± 9.6	53.4 ± 23.2
NON-SURV	18.0 ± 2.1*	16.4 ± 5.2	5.9 ± 2.3*	10.4 ± 8.4*	7.4 ± 6.2*	21.2 ± 0.4*
Abdominal wall						
Normal	7.6 ± 4.6	17.8 ± 4.4	40.5 ± 6.2	39.1 ± 4.6	40.0 ± 7.4	48.4 ± 6.5
SURV	12.6 ± 4.9	27.7 ± 7.3	46.4 ± 15.3	42.4 ± 15.3	42.3 ± 9.2	53.1 ± 23.2
NON-SURV	0.3 ± 3.2*	14.2 ± 6.8	16.5 ± 4.2*	20.4 ± 12.3*	7.1 ± 6.9*	25.5 ± 9.9*

*Significantly different from normal by analysis of variance.

The LF at each ISO concentration and the V_{max} were significantly decreased in the NON-SURV, reflecting the degree of catecholamine stimulation. The APACHE II and APS scores were similar between SURV and NON-SURV. The data thus indicate that the beta-1 receptor of adipose tissue can provide an early measure of septic stress and be an early indicator of septic outcome.

R212

RESULTS OF PARKS' SPHINCTEROPLASTY FOR POST TRAUMATIC ANAL INCONTINENCE. S. Morgan, D. Bernard, D. Tassé, R. Wassef. Department of Surgery, University of Montreal, Division of Gastrointestinal Surgery, Hôpital St-Luc, Montreal, PQ

Surgical and obstetrical trauma is one of the commonest causes of fecal incontinence. Controversy still exists as to the type of repair and the need for a temporary diverting colostomy. We report on 45 patients (40 F, 5 M): 17 (38%) had one or more previous attempts at repair. Duration of incontinence following obstetrical trauma averaged 13 years versus 4 years following other trauma. Diverting colostomy was established in every patient either shortly after the trauma for the incontinence or immediately before attempting repair and was closed 3 months after the repair in every case. Sphincteroplasty was performed according to Parks' technique by dissection of the sphincters over half of its circumference, dividing the centre of the fibrotic ring, and doing an overlapping type of repair and leaving the wound open for second-intention healing. There was no mortality. Results for incontinence were judged good to excellent in 82%, fair in 9% and failures in 9% (four cases). Three of the failures were repaired according to the same technique and the result was excellent in two and fair in the other. Previous reports mentioned three factors of increased risk of failure: age > 65, previous repairs and over 10-year duration of incontinence. Assessment of these risk factors reveals no difference in our results, possibly because of the presence of a colostomy. The rate of failures was significantly higher in the first 15 cases (47%) than in the last 30 (3%) ($p < 0.01$). Sphincter repair following Parks' principles is sound; a colostomy is recommended after major trauma or sepsis and should be considered when risk factors of failure are present.

R213

THE RESULTS OF TREATMENT USING COMPOSITE RESECTION IN PATIENTS WITH SQUAMOUS CELL CARCINOMA OF THE ORAL CAVITY FOLLOWING FAILED IRRADIATION THERAPY. R.E. Robins, S. Schlagentweit. Cancer Control Agency of British Columbia and University of British Columbia Medical School, Vancouver, BC

In our institution, the majority of patients with T1 and T2 N0 and selected T3 and T4 N0 squamous carcinoma of the oral cavity receive irradiation as primary therapy. There is close follow-up and surgical treatment for failure or recurrence. A retrospective study of 98 consecutive patients undergoing composite resection from 1971 to 1983 was done to determine and compare treatment results in those irradiated (76) and the total group including non-irradiated (22) patients. In the 22 patients treated primarily by composite resection, 21 had T3 or T4 lesions. All were followed at least 2 years and 84% more than 3 years. A computer-assisted analysis of the data was performed yielding product limit and actuarial survival curves.

The commonest sites were floor of mouth (35), tongue (19) and lower alveolus (19). There was no significant difference in the overall survival between the irradiated and total group. The 2, 3 and 5 years' survival for all patients was 58%, 56% and 48%; for irradiated patients 56%, 54% and 47%. There was also no difference in the behaviour of the irradiated group as compared to the total in comparison of tumour size, positive margins or node status. In both groups, 25% had positive margins at surgery. This was followed by ultimate failure as at 3 years there were no survivors with positive margins; for clear margins there was 70% survival at 3 years and 61% at 5 years. One-half of both

groups had positive cervical nodes. Three and 5 years' survival with negative nodes was 65% and 61%; with positive nodes 46% and 37%. The level of nodal involvement was highly significant.

R214

SPECIFIC AND NONSPECIFIC IMMUNITY IN THE RESPONSE TO *PSEUDOMONAS* INFECTION FOLLOWING THERMAL INJURY. J.C. Marshall, H. Rode, D. Eshelby, J.L. Meakins, N.V. Christou. Department of Surgery, Royal Victoria Hospital, McGill University, Montreal, PQ

The role of specific and nonspecific immune mechanisms in the response to pulmonary infection with *Pseudomonas* following thermal injury was studied in a rat model. Male Lou/Lewis rats (mean weight 320 g), presensitized to keyhole limpet hemocyanin (KLH), underwent a 30% TBSA scald burn or sham burn. Twenty-four hours later, animals were immunized IP with 10^8 heat-killed *Pseudomonas aeruginosa*. Seven days following immunization, blood was drawn for assay of specific anti-*Pseudomonas* IgG antibody using an ELISA to whole organisms, then animals were challenged intratracheally with 2×10^8 CFU live *Pseudomonas*. Animals were sacrificed 24 hours later, and the right lung was homogenized for quantitation of residual numbers of viable *Pseudomonas*. The mean diameter (mm) of the delayed-type hypersensitivity (DTH) response to recall skin testing with KLH was used as a measure of nonspecific immune responsiveness following the burn. Specific anti-*Pseudomonas* IgG was expressed in units/ml measured against a standard serum arbitrarily defined to have 10 U/ml of activity. Results follow (mean ± SEM).

Group	24 h DTH	72 h DTH	1 wk DTH	Anti-pseudo IgG	Log_{10} CFU pseudo/ml
Controls	6.8±0.3	10.2±0.2	9.0±0.3	69.5±11.0	1.97±0.1
Burn	1.8±0.5[a]	3.6±0.5[a]	4.2±0.3[a]	61.5± 7.7[b]	2.30±0.1[c]

[a]$p < 0.001$, [b]NS, [c]$p = 0.01$.

A 30% TBSA burn injury produced marked and persistent impairment of nonspecific host defences; in contrast specific anti-*Pseudomonas* antibody production was normal. Pulmonary clearance of a bacterial challenge was depressed, despite an intact humoral immune response.

R215

SURGERY FOR CHRONIC CONSTIPATION. J. Engel, J. Duff, D. Grant, B. Taylor. Department of Surgery, University of Western Ontario, London, Ont.

We reviewed 25 patients who underwent surgery between 1976 and 1986 for severe disabling constipation not controlled by laxative and enema regimens. There were 22 females and 3 males with a mean age of 43 years (range 5 - 86). They had an average of 1.2 bowel movements per week (range 0 - 7). Additional symptoms included abdominal bloating 22 (88%), abdominal pain 20 (80%), straining at stool for greater than 5 minutes 14 (56%) and nausea 14 (56%). The preoperative diagnosis was colonic inertia in 23 (92%) and anal sphincter spasm in 2 (8%). Barium enemas performed in 22 patients showed: no abnormality - 8, megacolon - 7, dolichocolon - 6. Transit studies demonstrated colonic inertia in 14 and outlet obstruction in 2 patients.

The following operations were performed: subtotal colectomy with ileorectal anastomosis - 15 (60%), total colectomy with ileostomy - 4 (16%), subtotal colectomy with ileosigmoid anastomosis - 3 (12%), extended internal sphincterotomy - 2 (8%) and partial colectomy with cecorectal anastomosis - 1 (4%). The mean follow-up was 19 months (range 1 - 60 months). Adhesive small-bowel obstruction occurred in five (20%) patients; three required reoperation. Constipation was completely relieved in 14 (56%) patients. Ten (40%) patients were improved: 7 (28%) required regular laxatives and 3 (12%) required occasional laxatives. One patient remained constipated following an extended internal sphincterotomy. No one suffered from disabling diarrhea.

We conclude that total colectomy alleviates disabling constipation due to colonic inertia.

R216

HUMAN COLONIC MUCIN IN INFLAMMATORY AND NEOPLASTIC DISORDERS. B.E. Lukie. Department of Physiological Sciences, University of Newcastle-Upon-Tyne, UK

A mucus alteration of unknown significance has been observed in neoplastic and inflammatory disorders of the colon. The objective of this work was to characterize the molecular size distribution of this abnormal mucus. Mucus obtained from fresh colectomy specimens was solubilized and analysed by Sepharose CL-2B gel filtration chromatography. Two glycoprotein peaks were regularly observed: a high molecular weight peak (mucin) which eluted at the void volume and extended to the initial included volume; a late-eluting low molecular weight peak (LMWP) of higher protein content. The distribution of glycoprotein between these two peaks was unaffected by prior heating at 100°C for 10 minutes, incubation at 37°C for 48 hours or 10^{-3}M phenylmethylsulfonyl fluoride. Both ulcerative colitis (UC) and cancer (CA) samples showed an increase in LMWP protein content when compared to a mucus from a patient with sigmoid volvulus. Mucin subunit which elutes between the mucin and LMWP peaks was not seen in any of the CA samples but was present in one of the two UC samples studied. Thus, CA and UC mucus are associated with an increased protein content of the LMWP. Mucin subunit formation was not detected in CA, but may be a feature of some cases of UC.

R217

MANAGEMENT OF MALIGNANT COLORECTAL POLYPS. D. Bernard, S. Morgan, D. Tassé, R. Wassef. Department of Surgery, University of Montreal, Division of Gastrointestinal Surgery, Hôpital St-Luc, Montreal, PQ

Management of patients with endoscopically removed malignant intestinal polyps remains controversial. The risk of residual disease, estimated at 10.1% by Wilcox (*Cancer* 1986; 57: 160–171) should be assessed against the risk of a surgical operation. We report 30 cases of malignant polyps (7.3% of 409 colonoscopically removed adenomas). Fourteen contained carcinoma in situ and had no further treatment and 16 had invasive carcinoma (5 sessile, 11 pediculated). Among these 16, 5 were not submitted to surgery: because of old age in 1, minimal invasion in 3 and low rectal location in the other. Eleven (3 sessile, 8 pediculated) underwent surgical resection and residual disease was present in 3 (27%), 1 with positive nodes. Reported criteria of increased risk of residual disease — CA in lymphatics or veins, incomplete excision, tumour at resection margin, sessile and villous — were present in seven. All three patients with residual disease had one or more of these criteria in their malignant polyps. Evidence of residual disease (10.1%) in the colonoscopy era studies comes from 14 series (*Cancer* 1986; 57: 160–171), such as this one, totalling 188 cases. The presence of criteria of increased risk justifies surgical resection unless the patient is an otherwise poorer operative risk.

R218

SCINTIGRAPHIC BALLOON PROCTOGRAPHY FOR ASSESSMENT OF SEVERE CONSTIPATION. M.E. Pezim, J.H. Pemberton, S.F. Phillips. Department of Surgery and Gastroenterology Unit, Mayo Medical School, Rochester, MN

To determine why some constipated patients remain unchanged following colectomy and ileorectal anastomosis, we examined the role of the anorectal angle during defecation. *Method:* Fourteen subjects (5 healthy controls, 9 severely constipated patients) were studied at rest and during defecation with an intrarectal balloon probe containing 5 mCi $^{99m}TcO_4^-$ in 70 ml water, imaged by gamma camera. Patients in Group A (n = 5) often used digital evacuation or perineal compression to facilitate defecation. Patients in Group B did not. *Results:* Group A differed significantly from both controls and Group B by having a more acute resting anorectal angle, *and* a reduced ability to straighten the angle during defecation. In contrast, Group B resembled controls in both resting and defecation anorectal angles.

Table: Anorectal Angle (°) (Mean ± SEM)

	Rest	Defecate	Change
Control	113 ± 4	133 ± 5	20 ± 4
Group A	101 ± 4*	109 ± 4	8 ± 2**
Group B	118 ± 5	142 ± 6	23 ± 2***

*Group A vs Group B rest; p = 0.02.
**Group A vs control change; p = 0.02.
***Group B vs control change; NS.

Conclusion: Scintigraphic balloon proctography differentiates constipated patients into two groups. Patients unable to straighten their anorectal angle during defecation have an expulsion disorder which would persist following colectomy. Those with normal anorectal angle dynamics have a colonic motility disorder for which colectomy is rational. Scintigraphic balloon proctography appears to be an important tool in selecting patients for appropriate surgical treatment.

R219

STRICTUREPLASTY FOR CROHN'S DISEASE. P.J. Whelan, F.G. Saibil, L.B. Cohen, A.W. Harrison. Division of General Surgery and Gastroenterology, Sunnybrook Medical Centre, University of Toronto, Toronto, Ont.

The threat of short-bowel syndrome secondary to resectional surgery for Crohn's disease has led us to test the safety and efficacy of strictureplasty for the obstructive complications of Crohn's disease.

In the past 21 months we performed strictureplasties on eight patients with Crohn's disease. The main indication for operation in all was chronic obstruction. Six patients had a history of previous bypass and/or resection.

Thirty-two strictureplasties have been carried out, encompassing 55 strictures. Two of the eight patients underwent limited resection concurrently with multiple strictureplasties.

The length of follow-up ranges from 4 to 21 months. One patient has required reoperation for recurrence of an enterovesical fistula. Two patients had evidence of postoperative leaks; both responded to conservative management. All patients are able to ingest a full, normal diet despite discontinuation of steroids. The nutritional status of all patients has improved or continued in the normal ranges as determined by body weight, serum protein and hemoglobin.

Strictureplasty is a safe and effective therapy for Crohn's disease.

R220

PERCUTANEOUS ENDOSCOPIC GASTROSTOMY (PEG) BY THE "PULL" AND "PUSH" METHODS. M. Deitel, T. To, E. Spratt, C. Burul. Department of Surgery and Department of Nutritional Sciences, University of Toronto, St. Joseph's Health Centre, Toronto, Ont.

After 28 patients studied prospectively underwent PEG by the Ponsky "pull" technique, a subsequent 28 patients underwent PEG by the Russell "push" technique. These were compared with the prior 28 patients retrospectively who had undergone Stamm gastrostomy as an independent operation by the same group of surgeons. All procedures were done within a 4-year period. *"Pull" Method:* After gastroscopic insufflation, a suture or guidewire passed through a Medicut was snared and secured to the end of a phlanged 14F mushroom tube which was pulled retrograde out the abdominal wall. *"Push" Method:* Through a needle, a guidewire was inserted over which a dilator-sheath was rotated; the dilator was removed, and a lubricated 14F Foley was pushed through the intragastric-viewed sheath which was peeled away.

Table: (n = 28 per group)	Stamm	"Pull" PEG	"Push" PEG
Age ($\bar{x}$ ± SD yr):	68 ± 12	69 ± 14	73 ± 15
Use: Neurologic diseases	15	16	14
Pharyngeal blockage	3	4	6
Cachexia, respiratory, burn	10	8	8
No. under local anesthesia:	5	13	17
Time for procedure (min):	55 ± 7 p < 0.001	27 ± 8 p < 0.001	16 ± 3
Wound infection:	5	6	0
Death within 30 days:	4	2	2

Feeding was started 3 days after Stamm and 24 hours after PEG, initially elemental diet, progressed to non-digested liquid diet. "Pull" PEG requires removal and reinsertion of the scope, and drags oral bacteria through abdominal wall which may account for peritubal infections. Percutaneous endoscopic gastrostomy was less costly than Stamm; "push" PEG was simpler, faster and safer.

R221

EFFECTS OF PROLONGED INTESTINAL TRANSIT TIME ON BILIARY LIPIDS IN THE PIG. N. Causton, O. Thurston, K. Walker. The Surgical-Medical Research Institute, University of Alberta, Edmonton, Alta.

Previous studies indicate that cholesterol saturation of bile is associated with prolonged intestinal transit time (so-called colonic stasis) characteristic of populations eating a low-fibre/high-refined-carbohydrate diet. This study was undertaken to determine the effect of prolonged intestinal time on biliary lipids (cholesterol, phospholipid and total bile salts).

Young domestic pigs weighing 10 – 30 kg had an antiperistaltic loop of colon fashioned to prolong intestinal transit time mechanically, as measured by the passage of radiopaque markers. Gallbladder bile for determination of biliary lipids was obtained by needle aspiration at celiotomy prior to reversal of a 10- to 35-cm colonic segment and at 4 and 8 weeks after reversal. The number of animals studied at 0, 4 and 8 weeks was 19, 13 and 9 respectively. The animals were on a cholesterol-free diet throughout the study.

Intestinal transit time in control animals was 53.5 ± 23.1 hours ($\bar{x}$ ± SEM) and was 175.2 ± 22.1 hours in animals with a reversed colonic segment ($p < 0.001$). Cholesterol saturation index of bile was 0.40 ± 0.03 in the control period and 0.38 ± 0.03 and 0.33 ± 0.02 at 4 and 8 weeks after colonic reversal. None of the observed differences was statistically significant.

Under the conditions of this study a marked prolongation of intestinal transit time did not increase the cholesterol saturation index of gallbladder bile. This result does not support the commonly held belief that prolonged intestinal transit time is a factor leading to cholesterol gallstone formation.

R222

THE EFFECT OF CYCLOSPORINE ON LIVER REGENERATION. R. Black, D. Grant, R. Zhong, W. Wall, P. Keown, J. Duff. Department of Surgery and Department of Medicine, University of Western Ontario, London, Ont.

Liver regeneration can be critical to the success of orthotopic liver transplantation when the donor organ has been compromised. Many drugs interfere with liver regeneration. The present study examined effect of oral cyclosporine (CsA) on liver regeneration. Thirty-two male Sprague-Dawley rats were placed into four groups after a standard 70% hepatectomy. The rats were sacrificed on the 10th postoperative day, and the following were measured: hepatic regenerative rate (RR), total food intake (FI) and the change in body weight (BW).

	Treatment	RR	FI	BW
Group 1	Olive oil	105%	186 g	+13%
Group 2	CsA 10 mg/kg/d	104%	159 g	+9%
Group 3	CsA 20 mg/kg/d	87%*	124 g*	−5%*
Group 4	Olive oil/pair-fed with Group 4	86%*	124 g*	−3%*

* = $p < 0.05$ compared to Group 1.
The correlation coefficient between FI and RR was +0.75 ($p < 0.001$).

We conclude: (1) there is a significant correlation between FI and liver regeneration, (2) low-dose CsA does not impair liver regeneration, (3) high-dose CsA indirectly impairs liver regeneration by inducing anorexia.

R223

LONG-TERM RESULTS OF ENDOSCOPIC SCLEROTHERAPY FOR BLEEDING ESOPHAGEAL VARICES: EFFECTIVENESS, COMPLICATIONS, AND SURVIVAL. R.J. Tabah, M.J. Wexler, A.P.H. McLean. Department of Surgery, Royal Victoria Hospital, McGill University, Montreal, PQ

Endoscopic sclerotherapy as initial and primary treatment of symptomatic esophageal varices in 77 consecutive, unselected patients over a 6-year period is reviewed in order to define the effectiveness, complications and survival associated with this form of treatment. Each of the 186 individual sclerotherapy sessions was performed through a Williams overtube under general anesthesia. Alcoholic cirrhosis was present in 69%. Of those with cirrhosis, 41% were classified as Child's C. No specific additional treatment other than repeat sclerotherapy was required in 58%. Only 23% ultimately required surgery for control of recurrent variceal hemorrhage. The rate of rebleeding following an individual sclerotherapy session was 29%. Forty-one percent of recurrent bleeding occurred within 1 week following sclerotherapy. Complications directly attributable to sclerosis were generally mild and well tolerated. These included superficial esophageal ulceration (11.8%) and fever (25%). Deep esophageal ulceration was observed in 17% of patients. Two deaths were directly attributable to sclerotherapy. Life-table analysis resulted in a 3-year survival of 71%, 63% and 27% in Child's A, B and C patients managed under our protocol. Major causes of death included hepatic failure, sepsis and hemorrhage. We believe that endoscopic sclerotherapy as performed under general anesthesia is a safe, effective means of initial and long-term management of the patient with bleeding esophageal varices.

Canadian Society for Vascular Surgery

R523

CAROTID ENDARTERECTOMY: LATE RESULTS. C.R. Lye. University of Manitoba, Health Sciences Centre, Winnipeg, Man.

The late results of carotid endarterectomy have been assessed in a consecutive series of 380 patients who underwent 444 carotid repairs between 1975 and 1986. Follow-up to date, subsequent stroke or death was obtained in 357 patients (94%).

There were 49 late deaths (30 cardiac, 7 cerebral), with a 7-year cumulative survival (life-table method) of 68.4%. There have been 18 late strokes (7 fatal), of which 3 were ipsilateral to the original repair. Cumulative freedom from all stroke events (including operative stroke) at 1, 4 and 7 years was 94%, 90% and 81% respectively. Freedom from fatal or disabling stroke at the same intervals was 97%, 96% and 89% respectively. Only three patients have experienced late disabling non-fatal strokes.

Late angiograms have been obtained in 53 patients and have demonstrated occlusion (5) and restenosis (6) of the original repair as well as marked progression of disease in the opposite carotid (13).

Repair of recurrent stenosis was carried out in four patients, two of whom presented with recurrent symptoms. Late contralateral repair was performed in 13 patients, 8 of whom were symptomatic.

Although many patients die with intercurrent coronary artery disease, carotid endarterectomy can result in prolonged freedom from disabling stroke. Late recurrent stenosis is less frequent and troublesome than contralateral disease progression.

R524

INFLUENCE OF SIZE ON SURVIVAL AFTER RUPTURED ABDOMINAL AORTIC ANEURYSM REPAIR. J.L. Murphy, G.G. Barber, N.V. McPhail, T.K. Scobie. Division of Vascular Surgery, Ottawa Civic Hospital, Ottawa, Ont.

One hundred and seventy-two consecutive patients underwent repair of ruptured abdominal aortic aneurysms (AAA) between 1970 and 1985. There were 146 males and 26 females with an average age of 69.8 years. The overall mortality rate was 49.4%. Fourteen of 51 variables were clinically and statistically important, the most significant being urine output under 100 ml in the Operating Room, hypotension (< 90 mm Hg) in the OR, arrest, history of collapse and initial systolic BP (< 90 mm Hg). Discriminant analysis correctly classified 90.2% of survivors with a false (+) rate of 9.8% and 83.7% of nonsurvivors with a false (−) rate of 16.3%. The principal contributors ($F > 4$) to the prognostic formulae were parameters of shock, not age or associated cardiopulmonary disease.

Size was documented in 133 aneurysms. Thirteen (9.8%) were less than 6.0 cm and 23 (17.8%) were 6.0 cm or less, while the average size of the larger (> 6.0 cm) AAA was 9.51 cm. The 13 smallest aneurysms (< 6.0 cm) were correctly diagnosed initially in only 46.2% compared to 76.7% overall ($p < 0.05$) and the time from arrival in the hospital to the Operating Room was 6.71 versus 2.37 hours for the rest ($p < 0.05$). The mortality for these small aneurysms was 76.9% compared to 45.0% for the remainder ($p < 0.06$). Small aneurysms were known to be present prior to rupture in 8/23 (34.8%) of cases, including 5/10 aneurysms measured at 6.0 cm.

This review confirms the continuing poor results after ruptured abdominal aortic aneurysm repair and supports a more aggressive approach to diagnosis and treatment including abdominal aortic aneurysms between 4.0 and 6.0 cm in size.

R525

THE SELECTIVE MANAGEMENT OF SMALL ABDOMINAL AORTIC ANEURYSM WITH SERIAL ULTRASOUND SIZING. J.R. Gutelius, R. Pattenden, P.M. Brown. Division of Vascular Surgery, Department of Surgery, Queen's University and Kingston General Hospital, Kingston, Ont.

Management of the small asymptomatic abdominal aortic aneurysm is controversial. The authors have attempted to clarify this problem by a sizing program for all patients with asymptomatic aneurysms less than 5.0 cm in diameter.

In the past 6 years, 163 patients have entered the sizing program. After the initial ultrasound, repeat studies were done on a 6-month basis. Operation was performed if the aneurysm became symptomatic, increased more than 0.5 cm in diameter in 6 months, or reached 5 cm in greatest AP or lateral diameter. Twenty-eight patients have required surgery for symptoms (3), increase of greater than 0.5 cm in 6 months (5), and size of greater than 5 cm in diameter (20). There were no operative deaths and no patients ruptured prior to surgery. The operative patients were followed a mean duration of 17 months prior to surgery with a yearly increase in size of 1.1 cm.

One hundred thirty-five patients have been followed without surgery. Eighty-five of these patients have had two or more documented sizings with an average follow-up of 24 months. The mean yearly increase in size in this group was 0.2 cm. No ruptures occurred and there were only two deaths (CA of esophagus and myocardial infarction). Five patients were lost to follow-up.

Abdominal aortic aneurysm with a diameter of less than 5.0 cm can safely be followed. Even low-risk patients may be optimally managed conservatively and followed with serial ultrasounds with minimal risk of rupture.

R526

POPLITEAL ANEURYSMS: AN INDEX OF GENERALIZED VASCULAR DISEASE. A.M. Thijssen, G.G. Barber, N.V. McPhail, T.K. Scobie, C. Wm. Cole. Division of Vascular Surgery, Ottawa Civic Hospital, Ottawa, Ont.

Experience with 59 popliteal aneurysms in 38 patients, accrued over 10 years, was reviewed. Information is complete for 31 patients (50 legs) available for follow-up or who were followed until they died. Seven patients were lost to follow-up after several months and are included only so far as reliable information is known. Age at presentation ranged from 93 to 39 with the median age of patients with unilateral aneurysms being 5 years younger than those with bilateral disease (67 versus 72).

There was only one female. Associated vascular pathology was noted with increasing frequency as age progressed including the development of a second popliteal aneurysm in patients presenting initially with unilateral findings. Two such patients were found to have developed an aneurysm in the opposite leg on subsequent follow-up 5 and 7 months after initial treatment. Seventeen patients with unilateral popliteal aneurysms remain asymptomatic with respect to the opposite leg, but the follow-up experience is considerably shorter than for the group who presented with bilateral disease. Initial management consisted of a bypass graft in 34/59, immediate amputation in 9/59, while 16/59 unreconstructable but viable limbs were treated without surgical intervention. Bypass material was ePTFE in 2/34 and saphenous vein in the remainder. Thirty of 34 grafts were patent at last follow-up or were determined to be patent at the time of death. Four grafts occluded, one in the perioperative period and the others at 4, 5 and 32 months resulting in three amputations, only one of which was below-knee. These data illustrate that popliteal aneurysms may serve as a gauge of generalized vascular disease, with a higher probability of occlusive as well as aneurysmal disease in other vessels when bilateral.

Vascular Pathology Associated With Popliteal Aneurysms

No. of patients	17 (unilateral)	21 (bilateral)
Age (median)	67	72
Follow-up (median)	21 (range 1–95 mo)	35 (range 1–96 mo)
MI	24 (%)	33 (%)
CABG	6	10
Stroke	12	19
AAA	24	38
Fem/iliac aneurysm	18	29

R527

THE RATE AND DISTRIBUTION OF BLOOD FLOW FOLLOWING PROLONGED SKELETAL MUSCLE ISCHEMIA. I. Forrest, T. Lindsay, A. Romaschin, D. Mickle, P. Walker. Division of Vascular Surgery, Toronto General Hospital, Toronto, Ont.

The pattern of muscle blood flow upon re-establishment of circulation to the lower extremity, following an acute arterial occlusion, is unclear, and yet may determine the extent of resulting necrosis. The purpose of this study was to document total blood flow and its distribution within the muscle, following prolonged periods of ischemia. The bilateral canine gracilis muscle preparation was used. Total blood flow was measured by timed venous collections. Distribution of blood flow was determined using a multiple microsphere technique. Measurements were made preischemia and during the reperfusion phase at 3, 18, 33 and 48 minutes following 4 ($n = 8$) and 5 ($n = 8$) hours of ischemia. Necrosis was quantitated using nitroblue tetrazolium staining in conjunction with computerized planimetry. The muscles were divided into six slices and blood flow to each slice, as well as to the alive and dead portions, was calculated.

We demonstrated that preischemic flow is homogeneous throughout the muscle (4.4 ± 0.02 ml/min/100 g). On reperfusion, total flow was 6–10 times higher and the distribution was preferentially to the middle slices. Blood flow in each slice was correlated to necrosis ($r = 0.4$) ($p < 0.05$). The degree of reactive hyperemia was greater following 4 hours of ischemia than 5 hours (12.5 cc/min/100 g versus 20.3 cc/min/100 g, $p < 0.05$) but there was no correlation between blood flow to alive versus dead portions, in each slice (p = NS).

We conclude that upon reperfusion muscle blood flow is no longer homogeneous. The rate of reactive hyperemia is inversely related to the length of ischemia. The extent of returning blood flow may not be the sole predictor of muscle viability.

R528

EVALUATION OF CEFAZOLIN AND CEFAMANDOLE TISSUE CONCENTRATIONS DURING AORTOFEMORAL BYPASS GRAFT PROCEDURES. A. Downs, T. Louie, C. Lye, D. Howard, G. Gray, H. Fong, W. Krulicki. Department of Medicine, Department of Medical Microbiology and Department of Surgery, University of Manitoba, Winnipeg, Man.

Coagulase-negative staphylococci (CNS), primarily *S. epidermidis*, are increasingly recognized as pathogens causing prosthetic graft infections. We measured serum and tissue concentraions of cefazolin (CFZ) or cefamandole (CFM) during the surgical procedure. One gram of antibiotic was infused over 15 - 20 minutes beginning 60 minutes prior to surgery. Aortic blood (for preclotting the Dacron graft), a biopsy of the aorta at the proximal anastomosis and soft tissue from the region of the distal anastomosis were obtained at approximately 100, 115 and 150 minutes respectively, after initiating the antibiotic infusion. Results are shown below:

Mean Concentration ± SE of Antibiotic (μg/ml Tissue Fluid)

	Serum at preclotting graft (90–100 min)	Aorta (115–120 min)	Tissue, distal anastomosis (140–150 min)
Cefazolin, n = 16	43.3 ± 6.7	16.0 ± 3.6	15.2 ± 3.2
Cefamandole, n = 7	23.0 ± 4.9	8.7 ± 1.9	3.7 ± 0.8

Excluding isolates of *S. hemolyticus*, both CFZ and CFM were generally bactericidal for CNS. However, CFM was two- to fourfold more active on a weight basis with an MBC_{90} of 1.0 μg/ml whereas the MBC_{90} for CFZ was 4 μg/ml. Despite lower serum and tissue concentrations as compared to CFZ, CFM should be further evaluated as prophylaxis during vascular graft procedures since serum or tissue concentration/MBC_{90} ratios suggest that CFM is an equivalent or superior drug as compared to CFZ. The results of an ongoing randomized comparison, including quantitative and qualitative cultures of graft material will be presented.

R529

IS AORTOPROFUNDA BYPASS A SUCCESSFUL OPERATION FOR MULTILEVEL OCCLUSIVE DISEASE? P.G. Kalman, M. Rae, K.W. Johnston, P.M. Walker. Division of Vascular Surgery, Toronto General Hospital, Toronto, Ont.

Proximal reconstruction is performed first in patients with combined aortoiliac and femoropopliteal occlusive disease. Profundoplasty of variable length, however, may be required for providing adequate outflow. The purpose of this study was to determine whether aortoprofunda bypass is successful for multilevel occlusive disease. Aortoprofunda bypass was performed in 121 patients for disabling claudication (66%) and limb salvage (34%). The superficial femoral artery was occluded in 73% and diseased but patent in 27%. Fifty-seven (47%) had bilateral and 64 (53%) had unilateral profundoplasties — local in 16%, intermediate (past one perforator) in 40%, and extended (past three perforators) in 44%. The cumulative graft patency, clinical success (patent and clinically improved) and hemodynamic success (patent and improved ankle/brachial index) were determined by life-table analysis (see table).

	Cumulative (%) at 5 years	Standard Error (%)
Graft patency	97%*+	1.3%
Clinical success	77%*	5.2%
Hemodynamic success	67%+	5.9%

*, + $p < 0.01$, log rank test.

There was no significant difference in success when patients were stratified for sex, preoperative grade (disabling claudication versus limb salvage), type of proximal anastomosis and extent of profundoplasty. No patients underwent concomitant distal revascularization and only four subsequent infrainguinal bypasses were required.

In conclusion, aortoprofunda bypass is a successful operation for the management of multilevel peripheral occlusive disease with very few patients requiring subsequent distal revascularization. Despite continued patency, gradual subjective and objective deterioration occurs secondary to progression of disease.

R530

FACTORS INFLUENCING RESULTS OF 136 ARTERIAL RECONSTRUCTIONS FOR LOWER LIMB ISCHEMIA. F.M. Ameli, M. Stein, J.L. Provan. University of Toronto, Division of Vascular Surgery, The Wellesley Hospital, Toronto, Ont.

We present a 5-year follow-up study of 136 arterial reconstructions for lower limb ischemia. A statistical analysis was performed using the standard life-table analysis, the log rank test, the Krusbal–Wallis analysis

and the Spearman rank correlation coefficient. At 5 years of follow-up, cumulative patency rate (CPR), limb loss rate (LLR) and cumulative mortality were 52.3%, 15.8% and 30% respectively. The best predictors of long-term mortality were age ($p = 0.018$) and operative complications ($p < 0.001$). Over 5 years of follow-up CPRs were higher for claudicants than for limb salvage cases ($p = 0.037$). At 5 years, CPR of autogenous saphenous vein (ASV) grafts was 67.5% compared to 38.2% for prosthetic grafts ($p = 0.016$). Autogenous saphenous vein grafts had higher CPRs only for patients 64 years or younger ($p = 0.007$). For patients older than 64 years, ASV and prosthetic grafts had similar CPRs. In patients with ASV grafts, good runoff was associated with significantly higher CPRs compared to poor runoff ($p = 0.041$). Claudicants lost only 2 limbs (3.9%) over the 5-year follow-up, compared to 12 lost limbs (14.5%) for limb salvage patients ($p = 0.038$). Claudicants with ASV grafts had a 5-year LLR of 0% compared to 21.3% for limb salvage ($p = 0.039$). However, claudicants with prosthetic grafts had a 5-year LLR of 17.8% compared to 20.1% for limb salvage patients with prosthetic grafts ($p = 0.356$). Postoperative smoking increased the probability of limb loss and adversely affected CPRs.

In conclusion, LLRs and CPRs are affected by multiple interactions with variables such as preoperative symptoms, graft material, age, runoff and postoperative smoking.

R531

REOPERATION WITHIN TWO YEARS FOLLOWING AORTO-FEMORAL BYPASS. J.G. Sladen, A. Gerein, J.L. Gilmour, T. Maxwell, R. Wong. University of British Columbia, St. Paul's Hospital, Vancouver, BC

This study was undertaken to assess the factors leading to early reoperation following aortofemoral grafting. All data were recorded prospectively. We analysed 269 consecutive primary aortofemoral grafts (523 limbs) operated during the 5-year period prior to Dec. 31, 1983. Including the initial hospitalization, 36 limbs (7%) had another lower extremity vascular operation within 2 years. Hospital morbidity included thrombosis with revision (2.2%), amputation (0.7%), and death (0.7%) which compares favourably with the National Vascular Audit.

Precursors to reoperation were: occluded superficial femoral (12%), gangrene (23%) and acute severe ischemia (40%). Operations performed were: redo 1; thrombectomy 1; embolectomy 1; femoral crossover 5; profundoplasty 1; femoropopliteal or tibial bypass 27. The reason for secondary operation was classified as: technical 13; disease "progress" 5; insufficient improvement 6; "opposite" leg 2; questionable selection or candidate for dilatation 10.

Inadequate management of an isolated profundus outflow was the commonest technical problem, preceding nine reoperations, five of these for thrombosis. Reoperation varied from 9 to 33% by surgeon. Although occlusion of the superficial femoral artery (SFA) is of statistical significance with regard to reoperation ($p = 0.001$), 88% of the limbs with an occluded SFA did not require subsequent reconstruction within the first 2 years. Early reoperation can be reduced by identifying high-risk limbs and planning management and surgery more carefully in these patients.

R532

REVASCULARIZATION FOR RENAL FAILURE. S.J. Brister, J.R. Gutelius, P. Morrin, P.M. Brown. Department of Surgery, Kingston General Hospital, Kingston, Ont.

Between 1982 and 1987, 10 patients from our institution have undergone unilateral saphenous vein bypass (6), bilateral saphenous vein bypass (2), endarterectomy (1) or renal artery reimplantation (1) for renal failure. Six operations were done on an emergency basis for severe oliguria or anuria. Four patients were done on an elective basis for solitary kidneys with high-grade arterial stenoses. Nine of 10 patients had atherosclerotic renal artery stenosis or occlusion. One patient had bilateral renal artery obstruction after rupture of a high recurrent abdominal aortic aneurysm. Eleven of 12 kidneys were greater than 10 cm in length with the remaining kidney being 8.9 cm. Operations emphasized optimal exposure with rotation of viscera as required. Ischemic times at surgery varied from 20 minutes to 1½ hours with renal cooling used in only one patient.

Of nine patients with atherosclerotic renal disease, two required temporary dialysis postoperatively because of severe ATN. The patient with occluded renal arteries secondary to his aneurysm repair 48 hours previously did not recover renal function. Eight patients were discharged with one late death attributed to pneumonia. In those discharged, creatinine fell from a median of 720 μmol/L (range 160–1100) preoperatively to 290 μmol/L (180–470) postoperatively. No patients discharged required dialysis.

Renal artery surgery for salvage may be done in those with severe renal occlusive disease and normal size kidneys with a high degree of success. Although operative mortality and morbidity is significant, the possibility of relative normal renal function justifies the risks of renal revascularization.

R533

AORTIC PROSTHETIC–ENTERIC FISTULA. A.R. Downs, C.R. Lye, H. Fong. Department of Surgery, University of Manitoba, Health Sciences Centre, Winnipeg, Man.

Aortic prosthetic–enteric fistula (APEF) is an uncommon but devastating complication of abdominal aortic surgery. Over the past 10 years, 14 patients with APEF have been treated at our institution. There were 10 males and 4 females aged 50 to 82 years. The patients presented 3 weeks to 23 years after their initial graft placement. Four patients had upper GI bleeding, 12 patients had melena. One patient presented with anemia and unexplained fever. Massive bleeding in two patients required emergency operation. Upper endoscopy was performed in eight patients. In only one case was a diagnosis suspected. Aortography was done in 11 patients and was helpful in 3 patients, demonstrating two false aneurysms and a leak into small bowel. An upper GI barium study was done in four patients and demonstrated the lesion in two. The original operation was for aortic aneurysm in nine patients. The proximal anastomosis was end-to-end in 10 patients. One patient was operated upon for graft occlusion and the APEF was not discovered until postmortem. One patient had local repair of the duodenum and the aortic anastomosis. The remaining 12 patients had graft excision, omental patch and an extra anatomical bypass. Four patients died in the postoperative period and two patients died at 3 months and 4 months from aortic disruption. Eight patients have survived up to 4 years.

R534

IN-VIVO EXCIMER LASER ABLATION: ACUTE AND CHRONIC EFFECTS ON CANINE AORTA. D.L. Doyle, F. Litvack, W.S. Grundfest, A. Hickey, M. Fishbein, J. Forrester. Cedars-Sinai Medical Center, Los Angeles, CA

The lack of a fiberoptic delivery system has prevented in vivo excimer laser ablation. This is the first report of the acute and chronic effect of excimer laser on living arteries. We made 5 – 6 cm longitudinal anterior arteriotomies in the infrarenal aortas of 17 dogs. In 13, a 125 mm^2 rectangular segment of posterior intima was ablated by 85 ns pulsed, 308 nm excimer energy delivered via a 600 μm core 2 m optical waveguide. Four aortas were opened but not lased. Aortas were examined by light and scanning electron microscopy at 0, 1, 2, 24, 48, 72, 106 hours; 1, 2, 3, 4 weeks.

Gross luminal thrombus developed in two control and one lased aorta. No aneurysms or fistulae developed. Excimer produced no thermal injury. Acutely, lased aortas had craters penetrating into the media. At 24 – 72 hours there were red cells in the media, localized fibrin deposition and neutrophil infiltration. At 2 weeks the media had regenerated and the intima partially re-endothelialized. At 3 – 4 weeks the intima had completely healed and was indistinguishable from control.

In conclusion: (1) Excimer energy transmitted via fiberoptics precisely ablates in vivo without thermal injury. (2) Ablated arteries heal by 3 weeks. (3) Excimer may be the technology of choice for laser angioplasty, especially for small-calibre thin-walled vessels.

R535

IS FASCIOTOMY NECESSARY FOLLOWING REVASCULARIZATION FOR ACUTE ARTERIAL OCCLUSION? R. Corbisiero, A. Graham, B. de Varennes, R. Baffour, J. Symes. Cardiovascular Research Unit, McGill University, Montreal, PQ

Controversy exists as to the need for fasciotomy at the time of revascularization for acute arterial occlusion. To address this issue we developed a unique canine model of compartment syndrome. Both hind limbs

were completely devascularized at the popliteal level except for an isolated pedicle of anterior tibial artery and vein. The right limb served as control while the left had both tibial vessels occluded for 8 hours then released for 16. Anterior compartmental pressures (CMP) were continuously monitored and popliteal venous effluent was collected for CPK, K, lactate and pH. Transfascial Po_2($TFPo_2$) measurements were performed over the compartments. One hour after the onset of reperfusion 35 mCi of technetium-99m pyrophosphate (TcPYP) was injected systemically, and the uptake expressed as a ratio of the left limb to the right (L/R). Muscle necrosis was also graded histologically following the reperfusion period. In Group I (n = 7) no fasciotomy was performed while Group II (n = 7) underwent fasciotomy before reperfusion. *Results:* $TFPo_2$ after arterial occlusion confirmed severe ischemia in both groups (mean 4 mm Hg). Data 1 hour after reperfusion were:

	CMP (mm Hg)	$TFPo_2$ (mm Hg)	Lactate (mg/dl)	CPK (IU)	TcPYP (L/R)
Grp I	100	37 (±8.5)	22.85 (±5.5)	5072 (±3794)	8.14 (±2.7)
Grp II	15.7	84.3 (±24.5)	14.67 (±5.7)	1426 (±923)	2.16 (±1.3)
	*p < 0.0001	*p < 0.005	*p < 0.01	*p < 0.01	*p < 0.002

*Student's *t*-test.

Elevation of CMP due to reperfusion significantly increases the extent of muscle necrosis following acute arterial ischemia. This study strongly supports the use of prophylactic fasciotomy in this setting.

R536

EARLY EXPERIENCE WITH IN-SITU SAPHENOUS VEIN BYPASS FOR LIMBS WITH CRITICAL ISCHEMIA. H. Fong, A.R. Downs, C. Lye. Department of Surgery, University of Manitoba, Winnipeg, Man.

Fifty-seven lower extremities in 55 patients (41 males, 14 females, mean age 69 years) underwent in-situ saphenous vein bypass for critical ischemia between 1985 and 1986. Indications for distal arterial reconstruction were ischemic lesions 56% and rest pain 44%. Associated medical conditions included hypertension 58%, angina 40%, old myocardial infarction 37%, congestive heart failure 25%, diabetes 40% and chronic smoker 68%. Regional anesthesia was used in 3/4 of patient group. Status of tibial vessel run-off by angiography were one vessel 68%, two vessels 25% and three vessels 7%. Proximal saphenous veins were anastomosed to common femoral artery 54%, superficial femoral artery 35%, profunda femoral artery 4% and prosthetic graft 7%. Endarterectomies to common femoral and superficial arteries were performed in nine extremities of which three had prosthetic patch angioplasty. Distal anastomoses were performed below knee in 56 extremities of which 45% distal popliteal artery, 35% posterior tibial artery, 11% peroneal artery and anterior tibial artery 7%. There were four deaths, mortality rate 7%, of which three died of acute myocardial infarctions and one of respiratory failure. Other complications included leg edema 89%, wound complication 18%, acute graft occlusion 11% and acute hemorrhage 7%. There were six early and six late graft occlusions of which three extremities from each group required amputations respectively. Overall cumulative patency was 82% in 30 days, 81% in 3 months, 75% in 1 year and 72% in 2 years. We feel that for distal tibial arterial reconstruction, in-situ saphenous vein bypass is a good procedure with acceptable patency rate for limbs with critical ischemia.

R537

MANAGEMENT OF TRAUMATIC AORTIC DISRUPTION. C. Cina, R. Maggisano. University of Toronto, Sunnybrook Medical Centre, Toronto, Ont.

Fifteen patients have been managed for traumatic rupture of the aorta at Sunnybrook Medical Centre in the last 6 years.

While classic teaching is to operate immediately, a more deliberate plan of management was used. Cardiovascular hemodynamics were carefully monitored and pharmacologic hypotension was maintained in an intensive-care environment. Priorities for associated injuries were established and often their treatment preceded that of surgical repair of the aorta. This was delayed until cardiorespiratory and CNS injuries were improved. Neither pump nor shunt was used in the operative repair.

Six patients were not operated upon: two were dead on arrival; one died of MI after 13 days; two refused operation (and are still alive); one died waiting for operation. The average cross-clamping time was 36 ± 12 minutes. Tube-graft interposition was used in seven patients and direct repair in two. The overall mortality rate of this management approach is 23%, and the operative mortality 22%. One patient became paraplegic and one had a left vocal cord paralysis.

Prior to this experience a comparable group of patients were managed with urgent operative repair: three with direct cross-clamping and three with cardiopulmonary bypass. The mortality rate was 85%.

We conclude that delaying surgical repair until major cardiopulmonary and CNS associated injuries resolve, will improve mortality and possible morbidity.

R538

CATHETER INJURIES OF PERIPHERAL ARTERIES: MANAGEMENT AND PREVENTION. T. Kieser, T. Wesley-James, G. Barber, N. McPhail, T.K. Scobie. Division of Vascular Surgery, Ottawa Civic Hospital, Ottawa, Ont.

Fifty angiographic catheter injuries required surgical repair between 1977 and 1985, 0.3% of 12 244 cardiac and 0.25% of 2858 peripheral angiograms. Arteries involved included 21 common femoral, 10 superficial femoral and 4 profunda femoral. Injuries were thrombosis in 25, laceration-hematoma in 12, false aneurysms in 6, emboli in 5 and arteriovenous fistula in 2. Major pathological lesions were laceration-perforation in 24, intimal damage in 3, significant atherosclerosis in 9, hypoplastic arteries in 6 and no gross wall change in 8. Operative repair was thrombectomy and patch angioplasty in 18, primary repair in 9, thrombectomy in 8, formal bypass grafting and other procedures in 16. Rethrombosis requiring reoperation occurred in five patients, all of whom were successfully repaired. Four of 47 patients died.

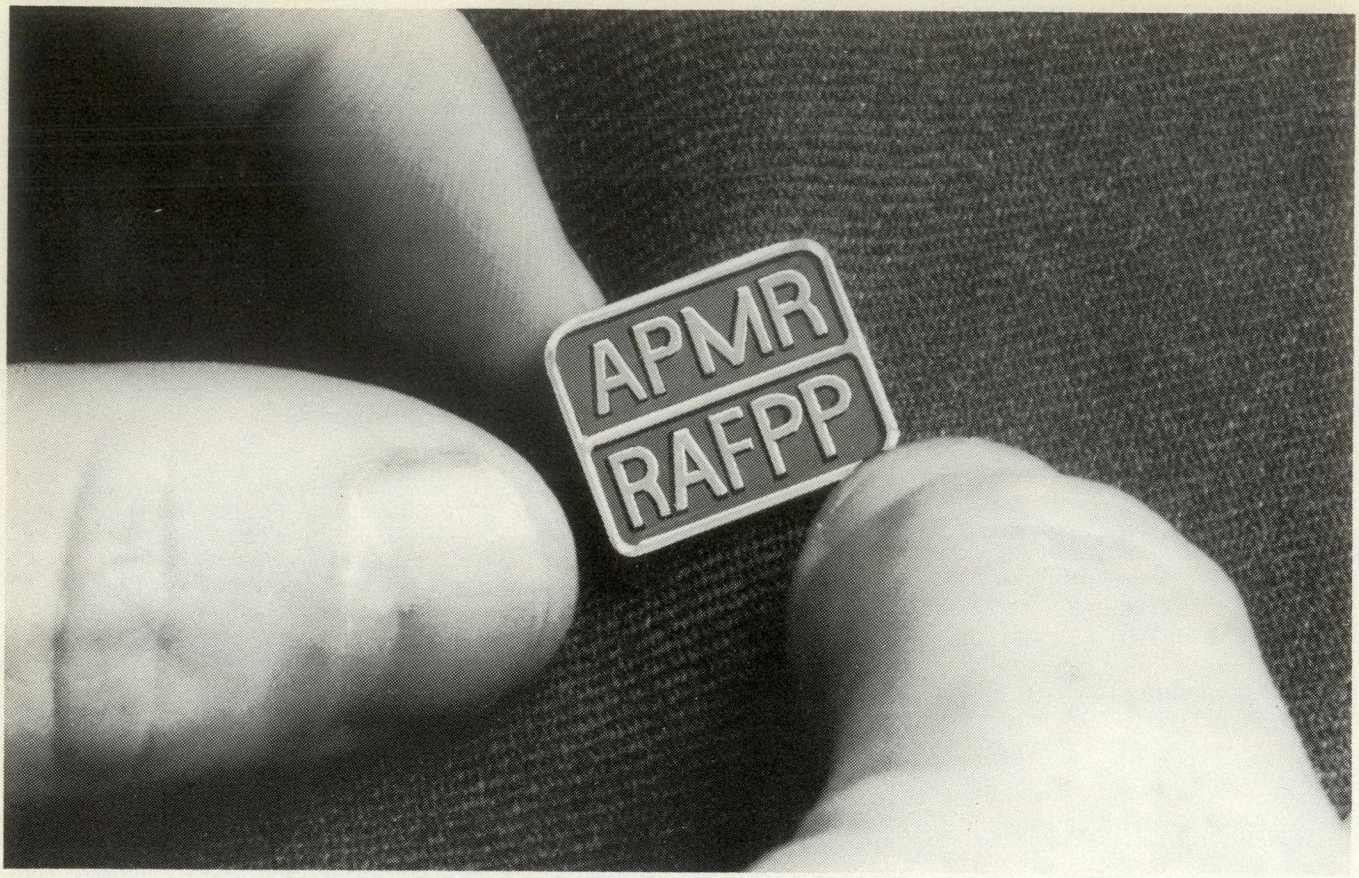
Since common femoral artery catheterization is preferable to catheterization of major branches, 100 patients were assessed at the time of elective femoral artery surgery to demonstrate the level of common femoral bifurcation. The area was marked by a transverse line laterally from the pubic tubercle and in the groin skin crease. Ninety-one percent of femoral bifurcations were below the pubic tubercle confirming the value of this landmark for safe femoral artery catheterization.

The incidence of catheter injuries can be reduced by precise common femoral artery puncture and the avoidance of atherosclerotic and hypoplastic arteries. Results depend on prompt diagnosis and immediate surgery, thrombectomy with patch angioplasty providing optimal operative repair.

R539

RADIONUCLIDE ANGIOGRAPHY AT REST AND EXERCISE IN PREDICTING CARDIAC OUTCOME IN PATIENTS UNDERGOING ABDOMINAL AORTIC RECONSTRUCTIVE SURGERY. W.P. Joyce, J.L. Provan, F.M. Ameli, P. McEwan. University of Toronto, Toronto, Ont.

Coronary artery disease (CAD) is the major cause of morbidity and mortality in patients undergoing elective abdominal aortic surgery. To identify this high-risk group of patients preoperatively, we have selected resting and exercise gated blood pool studies in 40 unselected and consecutive patients undergoing infrarenal abdominal aortic reconstructive surgery. Preoperatively, patients were allocated into two risk groups depending upon their left ventricular ejection fraction at rest (EF_R). Patients with $EF_R < 50\%$ (n = 11) were considered high risk and patients with $EF_R \geq 50\%$ (n = 29) were considered low risk. Evidence of CAD was based on a history of myocardial infarction (MI), congestive heart failure (CHF), or an abnormal preoperative ECG. Outcome was defined only in terms of cardiac complications and cardiac end points were taken as death, MI, CHF and pulmonary edema. Only four patients suffered a postoperative cardiac complication with one death (mortality 2.0%). These four patients had aneurysmal disease and a mean ejection fraction (EF_R ± SD) of 27 ± 5. Complications included pulmonary edema in three patients and cardiorespiratory failure and death in one patient. The clinical presence of CAD was significant in predicting outcome ($p = 0.01$) while information derived from stress electrocardiograms and exercise gated blood pool studies were not. Our preoperative assessment of risk based on $EF_R < 50\%$ was highly significant in predicting outcome ($p = 0.0004$) while EF_R alone was the best overall predictor of outcome ($p = 0.0001$).



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continued from page 232

that the timing would never be suitable for an intervention. The debate in the appeals court is expected by the QMA to establish some criteria for the body that represents physicians and for the timing for changes.

The QMA recently released "A New Contract for Health Care in Quebec", which says among other things, "...the greater part of [health-related] expenses is blamed on physicians; only in appearances can such an erroneous assumption be explained, not with facts and figures.... If we deduct the amount paid in 1984 by the RAMQ [Régie de l'Assurance Maladie du Québec] for medical services, that is $882 772 551, or 12% of total expenses, we then obtain the total amount of $6 337 665 449 for all other expenses.

"The Quebec physician is the one earning the lowest gross average income in Canada. In 1984, the gross average income was 40% lower than the Canadian average. Between 1971 and 1980, the consumer price index increased by 108.4%, while the physician's gross income was raised by a mere 48%. His purchasing power decreased by 30%; that of the salaried worker increased by 15%.

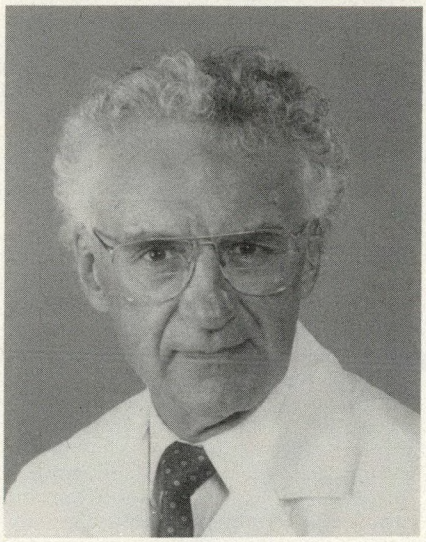
"...our Quebec physicians should be entitled to rates that compare to the Canadian average, to an updating of fees considering the technical difficulty, the rising costs of insurance, etc. They should be entitled to premiums for services rendered evenings, nights, weekends and statutory holidays."

Canada-wide statistics compiled by the federal government lend some support to the QMA's contentions that the physicians in that province are the lowest paid in the country. Criticized by medical associations as being a reflection of utilization as much as differences in fees, the indices are based on a sample of the most highly utilized items in the schedules for each specialty, the fees paid for consultations and an average price per service weighted by use patterns and total services rendered. Using these statistics, the government establishes a "median" fee for all services and gives it the value of 100, valuing accordingly the "all-services fee" for each province.

The indices for 1986 put Quebec's general surgeons, urologists, ophthalmologists, and thoracic and cardiovascular surgeons as the lowest paid for their specialty in Canada.

New Chairman for General Surgery, U of Ottawa

Mark Dover, FRCSC, was recently appointed chairman for the division of general surgery at the University of Ottawa and chief of general surgery at the Ottawa Civic Hospital. Dover received his medical degree from the University of Ottawa in 1955 and took postgraduate training in Ottawa and New York.



M. Dover, MD, FRSCS

Casualties of War Treated in Canada

Simon Wren, FRCSC, a general surgeon in Kingston is chairman of the medical committee of the local chapter of the Afghan Medical Relief Organization, and for the past few months has been coordinating efforts in Canada to bring medical help to casualties of the freedom fighting. He recently spoke to CJS about his work: "We have had two objectives: one to transfer medical equipment to the refugee camps in Pakistan and to bring to Canada people who require reconstructive surgery for injuries sustained during the war.

"At present at Hotel Dieu we have taken four patients — the first was a man who had chronic osteomyelitis caused by a gunshot wound to the tibia; the second had a malunion of the distal femur with consequent problems in the knee joint (he ended up with a fused knee). These patients were handled here by two orthopedic surgeons, Michael Ashworth and Douglas Hedden. The third patient was a young girl who had injuries, I believe, from stepping on a mine. Her foot and ankle had healed but were malaligned and she couldn't walk well. She has been treated with, in essence, an ankle and foot fusion and her foot has been straightened. The last one we treated here is a young girl who sustained shrapnel injuries to the head; she has retained some shrapnel in the brain tissue and has a partial right hemiparesis and partial aphasia. She's here for some of the problems associated with the hemiparesis — problems to the right arm and leg."

Wren has arranged for treatment of some of the patients elsewhere — in Toronto at the Toronto General, Western and St. Joseph's hospitals.

"All were patients in the refugee camps in Pakistan," he said. "I am coordinating the medical end of it; I get lists of patients from Pakistan with explanations of the problem; I relay the information to physicians who have elected to care for such patients. All the care is voluntary. The AMRO chapter has set up translation and home care services so these patients can be treated as outpatients.

"The patients have adjusted well to a life that is very foreign to them; all will be going back after their care is complete. The intergovernmental committee for migration in Geneva coordinates the travel of these people from the camps to the various countries that are providing assistance. The committee arranges exit visas with the government in Pakistan."

AMY CHOUINARD

continued from page 271

work of a related symposium. Most of the authors are well-respected, experienced physicians, renowned for their clinical expertise. The text is divided into eight sections that include historical considerations, surveillance and detection, pathologic features, special diagnostic techniques, management, adjuvant therapy, complications and follow-up procedures. The text is supplemented by beautiful illustrations and tables. The print is clear and easy to read.

Most of the book is well written. However, the multiauthor format has resulted in a weak and uneven style. Many of the chapters, particularly on surgical technique and radiation therapy, are of the "how I do it" type. There is frequently too little analysis of important recent trials or too much analysis of less-important issues (e.g., the no-touch technique). The chapters on radiotherapy are particularly poor in that regard. The results of major adjuvant radiation studies are buried in the chemotherapy section. In particular, the chapter dealing with preoperative radiotherapy and sphincter preservation contributes nothing to our knowledge. Similarly, there are two chapters, dealing with urologic and vascular complications, that are neither particularly common nor unique to colorectal cancer surgery. They seem, at best, to be filler. There are three separate chapters on polyps by four authors that, although not contradictory, are repetitive and still leave the reader confused.

The descriptive chapters on surgical techniques are the strongest in the book. The chapters on dysplasia in ulcerative colitis, proliferative defects and follow-up procedures are also excellent.

Steele and Osteen have selected fewer (14) contributors and used a much different format for *Colorectal Cancer*. They have established the controversial and key issues in each area of colorectal cancer and have asked the contributors to provide all of the important relevant data, analyses of them and their conclusions. To a great extent they have been successful.

The chapter on screening is particularly good at eliminating the confusion created by a multitude of studies. The chapters on surgical treatment concentrate on the survival advantages for particular techniques in a variety of situations. The conclusions on management of anal cancer are somewhat controversial but are well supported. The section on polyps is a reasonable analysis of the literature but does omit recent British literature which is at variance in some respects with the author's conclusions. The chapters on radiation by Gunderson and adjuvant therapy by Mayer are much clearer in their interpretation of the literature than in *Colorectal Tumors* and provide more of an opportunity for the reader to evaluate their conclusions.

The major weaknesses of the book lie in the poor quality of editing and printing. The quality of reproductions in the surgical treatment section is particularly poor, and the tables are at times too cluttered to be interpreted easily. There are a number of grammatical and typographic errors throughout the book.

In conclusion, both texts are comprehensive studies of colorectal cancer. Beahrs and colleagues have produced a generally well-written text that would be of some use to the practising general surgeon or resident. However, it has little to offer over and above standard British and American textbooks of colorectal surgery. For the serious students of colorectal cancer, particularly those involved in conducting clinical trials, Steele and Osteen have produced the better book.

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DIAGNOSTIC PATIENT STUDIES IN SURGERY. Bernard Sigel. 449 pp. Illust. Lea & Febiger, Philadelphia, 1986. $79.75. ISBN 0-8121-0996-1.

This excellent, concise textbook of commonly used diagnostic techniques provides rapid access to and selection of the most appropriate test for a specific clinical situation. Many specialists in the fields of surgery, radiology and medicine have provided detailed reviews of patient diagnostic studies, which are defined as those "requiring presence of the patient, not only specimen sent to the laboratory".

Diagnostic investigations are initially divided into three groups: imaging, endoscopy and function studies, but the book is divided into two parts: part I — imaging and part II — endoscopy, function studies and magnetic resonance. This is somewhat confusing but fortunately is the only weak point in this book.

Part I contains nine chapters, giving detailed reviews of radiologic techniques, beginning with an interesting introduction to the basics and technique. This is followed by plain roentgenography of the skeleton, chest and the abdomen. Then ultrasonography, computerized tomography and radionuclide scanning are described. Finally, the more invasive luminal contrast studies of hepatobiliary and gastrointestinal systems and angiography are presented. Whenever indicated, emphasis is placed on the application of a particular study in trauma or some other emergency situation. Each chapter is designed to present the background of the technique, indications, principles of diagnosis, complications and limitations of the test related to technique as well as to the examiner. Each overview is followed by a specific organ study, again with emphasis on indications, complications and limitations. In the chapter on plain-film roentgenography, the controversy regarding the use of preoperative chest x-rays is discussed. The chapters on ultrasonography and computerized tomography describe the indications for synchronized percutaneous biopsy. Percutaneous fine-needle biopsy is elaborated upon in more detail in part II.

Part II consists of 10 chapters dealing with the principles of urologic, respiratory, upper and lower gastrointestinal endoscopies. Two chapters are devoted to the applications of diagnostic laparoscopy and paracentesis, with emphasis on trauma. Pulmonary function tests are outlined with detailed discussions on the indications, interpretation and even cost-effectiveness. In the chapter on cardiac assessment, outlines of electrocardiography, cardiac catheterization and invasive bedside monitoring techniques are presented. Continuous esophageal pH monitoring, esophageal motility studies and gastroduodenal function studies are described. The efficacy of noninvasive vascular techniques and technical problems associated with them in relation to both the venous and arterial systems are well presented. In the last chapter, applications for magnetic resonance imaging are dealt with.

In all chapters, numerous well-selected studies including controversial issues are quoted. Each topic is supported with well-selected illustrations of normal or pathologic states, sometimes with emphasis on diagnostic errors. The endoscopy chapters lack illustrations, but these can be found in many textbooks and atlases.

Each chapter lists references from the current literature as well as general reading on the subject.

This textbook is not a manual and does not pretend to teach one how to perform or interpret a particular study. However, it gives very reliable reviews of most of the commonly used techniques with their rationale, indications and limitations. This book can be strongly recommended to all medical and surgical residents. Surgeons, medical specialists and general practitioners may use it as a reference guide to help them choose the proper study or to avoid inappropriate and costly tests in a particular clinical situation.

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FINE NEEDLE ASPIRATION FOR THE CLINICIAN. Joseph A. Linsk and Sixten Franzen. 337 pp. Illust. J.B. Lippincott Company, Philadelphia, 1986. Price not stated. ISBN 0-397-50678-3.

This book is based on the large experience of a consultant clinical oncologist who is an expert in fine-needle aspiration and works with a highly experienced cytopathologist. The authors rightly emphasize the lack of familiarity of many physicians with the use of fine-needle aspiration to solve clinical problems.

In the introductory chapter, the pictures and text on technique are excellent and the hazards of the procedure are well documented. The remainder of the book is divided into sections that catalogue practically all visible and palpable tumours in the human body, as well as those that can be imaged by various techniques. A short description of the clinical and pathological findings of each is given, and the potential role of fine-needle aspiration in establishing the diagnosis is considered. The section on the fine-needle aspiration of intrathoracic lesions, intra-abdominal lesions, the transrectal and transvaginal approach to pelvic lesions, and lesions of the skeletal system are of interest only to specialists in these areas and usually require the cooperation of an expert in diagnostic imaging.

The chapter on the breast includes a section on how masses may be discovered by a patient and an extensive section on the technique of clinical examination, both of which are not really germane to the central theme.

The authors continually mention the importance of experience in the use of the technique by the clinican and the interpretation of the slides by the pathologist. However, they fail to give any indication of the percentages of false-negative and false-positive results in their own experience or in that from other clinics, which have been well documented in the literature. In the primary surgical treatment of breast cancer, they imply that positive cytologic findings may preclude the need for frozen-section confirmation of the diagnosis of cancer before embarking on a primary surgical procedure. Most surgeons would forcefully disagree with this because false-positive findings have been reported.

This book contains a lot of extraneous information which clouds the clarity of its message. It is not recommended for the average physician. It is a good reference for any person who is interested in developing his or her expertise in the technique of fine-needle cytology to help elucidate the diagnosis of palpable lumps in any area of the body.

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UPPER EXTREMITY REPLANTATION. Basic Principles, Surgical Technique, and Strategy. Viktor E. Meyer. 121 pp. Illust. Churchill Livingstone, Edinburgh; Academic Press Canada, Don Mills, Ont., 1985. $58. ISBN 0-443-08448-3.

The author of this book on upper-extremity replantation has had extensive experience in the last decade in the management of patients with amputations of the finger, hand and forearm. The book presents a balanced, logical, thoughtful approach to the subject, which is one of the most complex in hand surgery. There are many problems in the management of amputations. Patient selection, operative technique and postoperative care are among the most important considerations and one chapter is devoted to each of these. Of particular note is the chapter on personnel and organizational requirements for a replantation centre. This chapter should be seriously considered by anyone thinking of doing solo replantation. Dr. Meyer's team consists of four surgeons who can provide not only 24-hour service, but can back each other up for long procedures and assist in taking patients back to the operating room for revisions.

The chapter on indications is a most important one and presents a balanced conservative approach with which I am in nearly complete agreement.

Surgical technique is well covered, with strong emphasis on skeletal fixation, which it rightly deserves. This chapter emphasizes technical principles and provides us with technical details that the author favours. Most replantation centres would agree with the majority of the techniques used, but might favour a free-tissue transfer rather than a groin flap for major soft-tissue defects associated with upper limb replantation. Early mobilization and edema control by elevation is considerably easier with a free flap.

SESAP V Critique

ITEM 315

This patient has an imperforate anus, better termed anorectal atresia with a rectourethral fistula. Patients with high-lying defects located above the levator sling mechanism are prone to urinary tract infection, primarily because of associated genitourinary malformations. In such patients the risk of infection prior to definitive pull-through operation and division of the rectourethral fistula is about 60%. Some patients have only sporadic difficulty, but others, particularly those with neurogenic bladders, develop chronic problems. In one series of 200 patients with various anatomic types of imperforate anus, 40% had some form of anomaly of the urinary tract, neurogenic bladder, significant urinary tract infection, or other forms of urinary complications. Metabolic acidosis may result from poorly functioning kidneys, further compromised by urinary tract infection. The reflux of urine through the fistula into the distal limb of the colostomy also causes acidosis, Chloride ions from the urine are reabsorbed through the bowel muscosa. Severe degrees of recurrent acidosis would necessitate division of the fistula with or without completion of the rectoplasty.

E

References

315/1. Stephens FD, Smith ED (eds): Anorectal Malformations in Children. Chicago, Year Book Medical Publishers Inc, 1971, pp 293-303

315/2. Wiener ES, Kiesewetter WB: Urologic abnormalities associated with imperforate anus. J Pediatr Surg 8:151-157, 1973

The section on postoperative care could be stronger. The author stresses the importance of postoperative therapy but allows only one page for discussion of postoperative therapy for the hand.

The reporting of results is a difficult area and the author is to be congratulated for his efforts. He has used the classification of Chen; unfortunately, this includes the patient's return to work, a factor that depends upon motivation, social support systems and the pre-injury job and does not really give us the information we would like — grip and pinch strength, manual dexterity, tactile gnosis and psychological adaptation to the injury. Nevertheless, the results section is useful, as it gives some idea of the levels of amputations and types of amputation injuries that are most likely to result in successful replantation.

The book is written in a lucid style and the illustrations are excellent. Overall, it is a major addition to the microvascular literature and is necessary reading for all surgeons who are practising in this field, and residents who are training in hand surgery.

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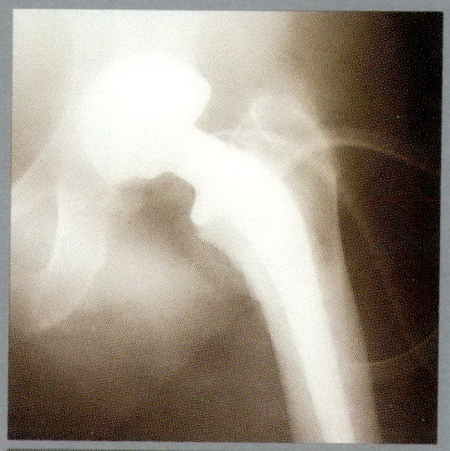
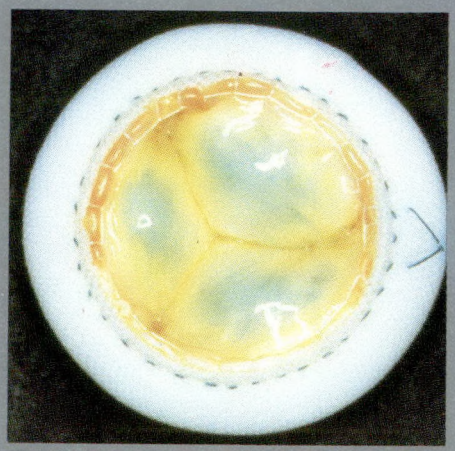
MICROSURGICAL COURSE: — The Department of Surgery, McMaster University is now offering one week, on-going courses in microsurgery, using animal facilities and videotapes. These courses are for anyone interested in small vessel anastomosis, nerve repairs, vas and fallopian tube reconstructions. The laboratory is organized to guide you through the basic microsurgical techniques to more advanced tasks with the help of an assistant on a one-to-one basis. Please call: **Dr. A. Thoma at (416) 523-0019 for further information.** —S86-23

MORE CONFIDENCE IN ANTITHROMBOTIC THERAPY

New Guidelines for Antithrombotic Therapy In Surgical Cases



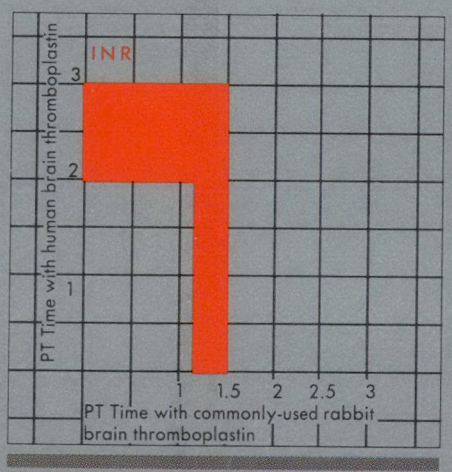
The American College of Chest Physicians and the National Heart, Lung and Blood Institute recently issued new guidelines for antithrombotic therapy.[1] There were four Grade A recommendations for the use of warfarin (Coumadin®) in surgical cases. In each of these four (and other Grade A and B recommendations for Coumadin®) a PT ratio of 1.2-1.5 (rabbit brain) was emphasized for clinical efficacy and reduced side-effects risk.



INDICATIONS: **Elective hip surgery / surgery for fractured hips.**

". . . . patients undergoing elective hip surgery should be pretreated prophylactically with adjusted-dose heparin or moderate-dose warfarin sodium . . ."

". . . . patients undergoing surgery for fractured hips should be treated prophylactically with moderate-dose warfarin.

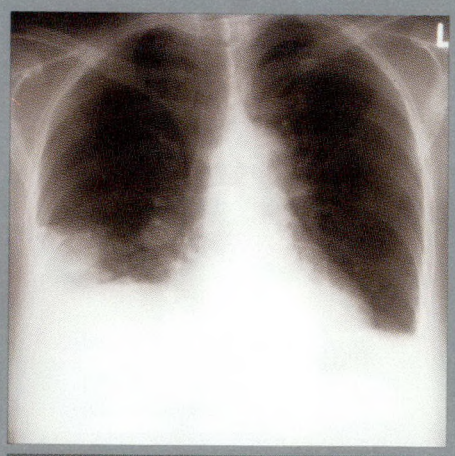


CONDITION: **Bioprosthetic heart valves.**

". . . all patients with bioprosthetic heart valves in the mitral position be treated for the first three months after valve insertion with less intense warfarin . . ."

INDICATION: **Prophylaxis of venous thromboembolism.**

"It is strongly recommended that the therapeutic range for prophylaxis . . . in high-risk medical or surgical patients should be equivalent to an INR of 2.0-3.0 (corresponding rabbit brain thromboplastin ratio 1.2-1.5)."



"We believe these recommendations will be of value to practicing physicians."

James E. Dalen, M.D., F.C.C.P., Jack Hirsh, M.D., F.R.C.P.C., Co-Chairmen: ACCP-NHLBI Conference on Antithrombotic Therapy

CONDITION: **Prophylaxis of venous thromboembolism.**

"It is recommended that anticoagulant therapy should be continued for three months using oral anticoagulants to prolong prothrombin time . . ."

1. **Chest** 1986;89(2);1S-106S

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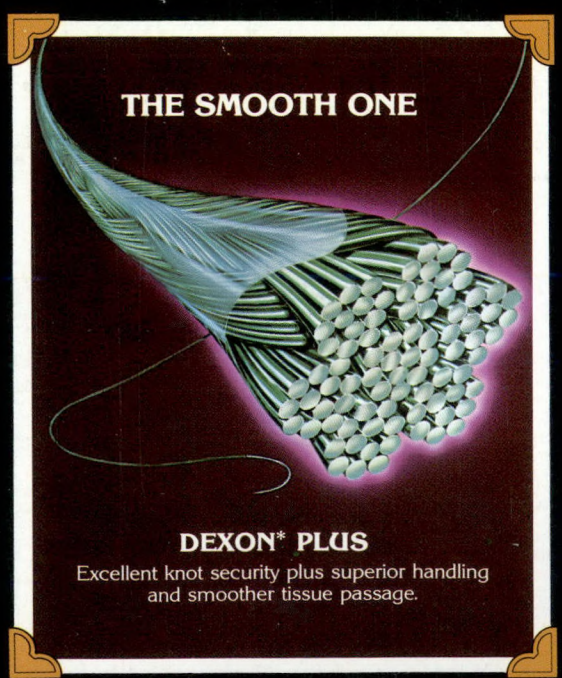

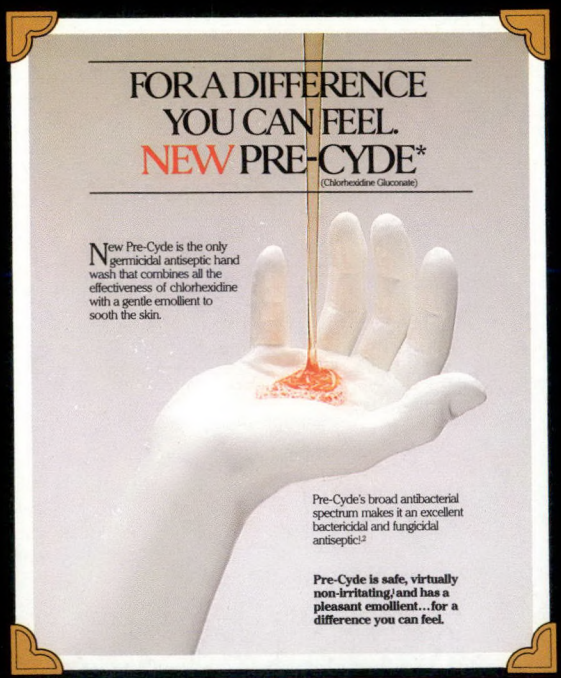

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