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Bleeding oesophageal varices

Bleeding from oesophageal varices is one of the most dramatic, urgent, frightening, and life threatening events for a patient. Its swift and accurate diagnosis and confident and effective management remain one of the most challenging clinical exercises in medicine. The diagnosis having been made, what is the method of choice for arresting the bleeding—and minimising the chances of recurrence?

Firstly, we have available two temporary measures to control immediate bleeding—intravenous administration of vasopressin and the Sengstaken-Blakemore tube. Either may help to bring the escape of blood under control—buying time for a more definitive attack on the varices with the patient more stable.

Secondly, we have five procedures with more lasting effect: one or other form of portasystemic shunt; transection of the oesophagogastric junction with reanastomosis; direct ligation of the varices through an abdominal or thoracic approach; porta-azygous disconnection using one of the circular stapling devices; or endoscopic variceal sclerotherapy.

The order of listing of these procedures more or less reflects the chronological order in which they were introduced and found favour. Currently most debate seems to centre on the relative merits of the last two.

The main reason for the considerable reduction in the number of portacaval shunts for bleeding varices has been the high risk of immediate mortality and serious morbidity, notably encephalopathy. Furthermore, the theoretical advantage of permanent reduction in the pressure in the veins at risk is more than offset by the price in other sequelae, particularly in patients with evidence of severe liver damage. Shunts may still be seriously considered in those with reasonable results of liver function—and the more proximal selective patterns of shunt (for example, that introduced by Warren) are more likely to have reasonable results.

The other direct surgical approaches to the varices have the simple objective of interrupting the link between the portal system, with its raised pressure, and the low pressure systemic tributaries around the lower oesophagus. None of these procedures do anything to reduce portal pressure permanently, and the anxiety is, therefore, that further anastomotic connections will develop and that bleeding will recur. The disillusionment with shunting is such, however, that surgeons are now concentrating on critical evaluation of these simpler direct procedures on the varices. We learnt with the shunts how much the outcome depended on the state of the liver damage; so it is no great surprise for us now to discover that again with the operations directly on the veins the same factors relating to the severity of liver upset are the principal determinants of good and bad outcomes.

Now that we can categorise patients with liver disease into prognostic groups with a fair degree of confidence it is surely right that when faced with variceal bleeding in patients with a poor prognosis our objective is to use the simplest procedure that will prevent death from haemorrhage. The two least disruptive options seems to be injection sclerotherapy and oesophageal division and stapling. Indeed, if endoscopic injection is shown to be as effective as stapling, or better, then open operation for this condition may become a thing of the past.

Not surprisingly, therefore, a recent spate of reports has assessed the early and medium term results of endoscopic sclerotherapy of varices.[1-4] As with all other procedures the thoroughness and competence of the technique matter: adequate protection against rebleeding depends on obliterating all the relevant varices at risk. Debate continues on issues including the number of injections which may safely be undertaken at each session, whether to inject directly into the vein or submucosally near the "neck" of the varix, and how frequently to repeat the endoscopic assessment or the injection or both. There is every indication that attention to detail and careful regular follow up to ensure satisfactory eradication of the actual varices are leading to better control and lower incidences of recurrent bleeding. If this picture is further reinforced there will be fewer open operations performed for this condition—and that implies the need for at least one competent and experienced endoscopist in each major centre.

No method, however, is going to be universally available or applicable—and surgeons will still have to be prepared to offer a satisfactory alternative to control the bleeding when endoscopic sclerotherapy is either not available or has failed. In this emergency setting in ill patients the ideal procedure must be one that can be completed expeditiously without too much dissection in very vascular planes and avoids excessive manipulation of other organs and tissues. These requirements point to the advantages of the circular stapling devices. All that is required is a small proximal gastrostomy to introduce the instrument, freeing a short segment of the lower oesophagus, the passage of an encircling ligature, firing the stapling "gun," removal, and closure of the wound. Several encouraging reports have appeared of the immediate results of this procedure[5] but I am not aware of any direct controlled trials of this method compared with injection sclerotherapy

—or direct comparisons between stapler transection and the various other manual procedures on or around the lower oesophagus. The evidence accumulating from uncontrolled reports of stapling/transection are at present sufficiently good, however, to suggest that this technique may now be the first choice when a direct surgical attack on the varices is indicated. If the surgeon either has no access to a stapler or no experience with the technique, then he is left with the choice between transection and reanastomosis or multiple ligation of the varices under direct vision—not always an easy procedure.

Finally, are shunts obsolete? Not yet, but if they are to continue to have a place in the overall management of cirrhosis with portal hypertension—then probably they will be selective rather than total, performed only on patients with reasonably good liver function (not as emergency procedures) and undertaken in selected centres by teams with special skills in both the operations and, as important, the management of the problems of liver failure.

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Staying cool with a hot test: gastroenterologists and ^{75}SeHCAT

Clinical tests of small intestinal function are few and crude. In part this is due to overlap in the absorptive functions of the jejunum and ileum; but the ileum has two functions unique to itself—active absorption of vitamin B_{12} and active absorption of bile salts. Furthermore, the active transport of bile salts is limited to the terminal ileum[1]; all we need to assess the function of the terminal ileum is a good test of bile salt absorption (on the reasonable assumption that most of this absorption occurs by active transport).

In the past, tests of bile salt absorption were too complicated and tedious for routine clinical use—but then ^{75}SeHCAT became available in January 1985. This selenium-75 labelled artificial bile salt, a homologue of taurocholate, has transformed matters. Now with nothing more complicated than a gammacamera any nuclear medicine department can measure how efficiently the circulating pool of bile salts is retrieved from the ileum and returned to the biliary tract.

This test seems like the answer to the gastroenterologist's prayer. After all, one of the biggest challenges in gastroenterological practice is Crohn's disease, which affects the terminal ileum more often than any other part of the gut. Yet at a recent conference on new radioactive tests in gastroenterology —at which most attention was given to the ^{75}SeHCAT test—the audience stayed calm, even cool, during discussion of this test.[2] Later I sent a postal questionnaire to 43 randomly chosen British gastroenterologists about their use of and views on the test and had replies from 38 of them (88%). Only six had the test available in their hospital. Only five of the other 32 admitted to being unhappy without it.

Why are gastroenterologists showing so little interest? Firstly, in Crohn's disease the function of the terminal ileum is usually undisturbed until structural changes are obvious on a good barium study. Secondly, simple tests for inflammation like the plasma concentration of C reactive protein and seromucoids and the platelet count are quite sensitive, if non-specific, pointers to the disease. Thirdly, new scanning tests, especially the indium-111 leucocyte scan, are coming into use[2-4] (they are used by 12 of the 38 gastroenterologists in my survey).

If ^{75}SeHCAT has a future it is not with Crohn's disease but with an even more elusive condition, idiopathic bile salt malabsorption. Unabsorbed bile salts cause watery diarrhoea and some gastroenterologists claim that they are responsible for between one quarter and one half of their cases of obscure diarrhoea—patients who would otherwise be labelled as having the irritable bowel syndrome.[2 5 6] Possibly these patients malabsorb bile salts because chyle moves too fast through the terminal ileum, but nobody really knows why the ileum fails in its duty of reabsorbing bile salts—structurally it appears normal.[5] This disorder is important because specific and effective treatment is available in the form of cholestyramine and other bile salt binding resins; and to diagnose it we now have a simple, safe, and specific test—the ^{75}SeHCAT test.

The case for ^{75}SeHCAT seems clear cut—the gastroenterologist without it cannot diagnose a disabling condition. Yet this is not really so. The diagnosis may be made in another way, which is simpler and cheaper: try the treatment and see if it works. By definition diarrhoea induced by bile acid is cured by cholestyramine. The pragmatic doctor sees nothing to gain from ^{75}SeHCAT except intellectual satisfaction—an expensive luxury, perhaps, at £50 a dose plus labour costs.

Might it be argued that since cholestyramine is a tedious if not unpleasant powder to take doctor and patient alike will not be motivated to stick to the treatment unless they have the result of a test to back their subjective impressions? That does not seem to me a convincing argument. If symptoms are bad and a treatment works the patient takes the treatment.

Furthermore, antidiarrhoeal agents which are easier to take may be just as effective, even if they are less specific. Resistance to loperamide is not a reported feature of idiopathic bile acid diarrhoea. Loperamide slows down small bowel transit and would be expected to improve bile acid absorption when impaired by rapid ileal transit.[7]

^{75}SeHCAT is an elegant and powerful research tool, allowing the enterohepatic circulation of bile salts to be studied more easily and in greater detail than ever before. With current financial restraints, however, the case for including it in the techniques of evey nuclear medicine department has yet to be proved.

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