

question of how continence was maintained. A technique similar to that adopted in urodynamics was used. Rectal and anal canal pressures were measured together with puborectalis and external anal sphincter electromyogram whilst the subject carried out a Valsalva manoeuvre. The rectum was filled with a dilute barium solution until a sensation of fullness was perceived. During the Valsalva manoeuvre the rectum was visualised radiologically whilst measurements were made. We have now carried out over 50 such examinations and never observed a flap valve occlusion of the upper anal canal. Indeed the anterior rectal wall was always clearly separated from the top of the anal canal. Moreover rectal pressures never exceeded anal canal pressures. The conclusions of our study reported in the *British Journal of Surgery*<sup>3</sup> were similar to those of Bannister *et al*<sup>1</sup>—namely, that continence is maintained by sphincteric means.

D C C BARTOLO

Consultant Senior Lecturer in Surgery,  
University Department of Surgery,  
Bristol Royal Infirmary,  
Bristol

#### References

- 1 Bannister JJ, Gibbons C, Read NW. Preservation of faecal continence during rises in intra-abdominal pressure: is there a role for the flap valve? *Gut* 1987; **28**: 1242–5.
- 2 Parks AG, Porter NH, Hardcastle JD. The syndrome of the descending perineum. *Proc R Soc Med* 1966; **59**: 477–82.
- 3 Bartolo DCC, Roe AM, Locke-Edmunds JC, Virjee J, Mortensen NJ McC. Flap-valve theory of anorectal continence. *Br J Surg* 1986; **73**: 1012–14.

#### Extrahepatic portal venous obstruction

SIR,—We thank Dr Triger for commenting on our paper in his leading article.<sup>1,2</sup> We would agree with him on most of the points except on the following issues. Our recent analysis of 521 patients with portal hypertension revealed cirrhosis of the liver in 215 (41%), EHPO in 213 (40%) and non-cirrhotic portal fibrosis (NCPF) in 93 (18%) patients. Only 17% of our patients with EHPO had their first bleed after the age of 20 years, compared with the series quoted by Webb and Sherlock where almost 50% were adults.<sup>3</sup> Because patients with NCPF have a similar presentation as EHPO, namely, well tolerated bleeds with splenomegaly, the discriminant analysis of the two conditions (EHPO and NCPF) from our centre has shown NCPF to present mostly in 2nd or 3rd decade with gross splenomegaly usually more than 11 cm below the costal margin, while EHPO presents in the

first or second decade with the spleen size usually less than 7 cm below the costal margin.<sup>4</sup> Our analysis of the size of spleen in EHPO has shown mild splenomegaly (5 cm) to be present in 42% of patients, moderate (6–10 cm) in 40% and gross splenomegaly (>11 cm) in only 18%. Thus gross splenomegaly, as mentioned in the leading article,<sup>1</sup> is not a very common feature in EHPO. Because of the size of the spleen, hypersplenism is more of a feature in NCPF than in EHPO.

As far as aetiology of EHPO is concerned, we have been unable to find any cause or association in 92% of our patients. Umbilical sepsis was responsible in only 13 (6%) of 213 patients. Congenital anomalies in the form of VSD, and mitral valve prolapse syndrome was noticed in only four patients (2%). We have never observed EHPO in a patient with cirrhosis, unlike the series quoted from Italy in the article.

In our series of 213 patients, obstruction in right and left portal vein branches was seen in 14.2% of patients, main portal vein obstruction in 51.7% and a total block of portal and splenic vein was seen in 28.9%. Isolated splenic vein thrombosis was seen in only one patient, unlike the series quoted by the leading article. Hence splenectomy alone is almost never done in a patient with EHPO.

With respect to the management of patients with EHPO, we agree with the feelings of Dr Triger that the first line of treatment should be sclerotherapy in this relatively benign condition with the hope that they will grow out of their bleed as the time passes. Indeed we have recently shown (under publication) that 40% of the patients developed large spontaneous/natural shunts after obliteration of oesophageal varices by endoscopic sclerotherapy. To date they have not rebled with a follow up of two years. These sclerotherapy induced spontaneous/natural shunts are significantly more common, than observed otherwise in EHPO without sclerotherapy<sup>2</sup>.

J B DILAWARI AND Y K CHAWLA

Postgraduate Institute of Medical Education and  
Research,  
Chandigarh 160012,  
India

#### References

- 1 Triger DR. Extrahepatic portal venous obstruction. *Gut* 1987; **28**: 1193–7.
- 2 Dilawari JB, Chawla YK. Spontaneous (natural) splenoarenorenal shunts in extrahepatic portal venous obstruction: a series of 20 cases. *Gut* 1987; **28**: 1198–200.
- 3 Webb LJ, Sherlock S. The aetiology, presentation and natural history of extra-hepatic obstruction. *Q J Med* 1979; **48**: 627–39.

- 4 Koshy A, Deshpande JV, Bahl CM. Computed aided clinical discrimination between portal vein thrombosis and non-cirrhotic portal fibrosis. *Bull PGI* 1983; 17: 106-9.

### Reply

SIR,—Thank you for letting me see the letter from Drs Dilawari and Chawla in response to my recent editorial. The only comment that I would wish to make is that my editorial was concerned with the whole problem of extra hepatic portal venous obstruction rather than the specific experience in India. I have indeed suggested in the paper (midway down page 1194) that there may indeed be features which distinguish EHPO in different parts of the world. The picture described in Chandigarh is not typical or representative of that found in the West.

D R TRIGER

*Dept of Medicine,  
Royal Hallamshire Hospital,  
Glossop Road, Sheffield S10 2JF*

### Dilatation of 'impossible' malignant oesophageal strictures using angiographic techniques

SIR,—Bown *et al* recently reported in *Gut* that in seven of the 37 patients in their series with advanced oesophageal carcinoma, it was impossible to insert a guide wire through the lumen.<sup>1</sup> They suggest that this finding is a good indication for laser therapy because it renders dilatation and intubation impossible. Prograde laser therapy, however, is more hazardous in the situation where there is no visible lumen to guide application. We have successfully used a new method to pass a guide wire and dilate such carcinomas by using angiographic techniques, where it was impossible or unsafe to pass a standard endoscopic guide wire.<sup>2</sup> This has now been done under sedation in nine patients on 12 occasions.

The technique used was as follows: a 16 French nasogastric (NG) tube was placed in the proximal oesophagus *via* the oral route. Dilute barium was then injected into the NG tube to confirm proximal oesophageal location. With the patient in the left lateral position, a Cook 15 mm J (TSCF) 0.96 mm guide wire was then placed through the NG tube into the proximal oesophagus with the tip of the guide wire exiting through the distal side port of the tube. The guide wire with its floppy end was then coiled in the proximal oesophagus. The NG tube was removed and a 5.3 French multipurpose torque control catheter was exchanged over the guide wire. The guide wire was then removed. Small amounts of contrast (water soluble may now be used) were injected to outline the lumen if present. One of a

variety of wires was then used in an attempt to cross the stenotic lesion. For a moderate to severe stenosis a very floppy straight wire (TSFNB 0.965 mm) was used. Through the torque control catheter, the TSFNB wire was advanced at small increments into the patent lumen of the stricture. The catheter then followed the TSFNB at small intervals, providing stiffness and mild steerability. This procedure continued until the lesion has been completely crossed by both wire and catheter.

If unsuccessful or for higher grade stenosis, a steerable wire such as a Lunderquist torque guide (Cook THG 0.965 mm) or a Waltman (Cook SMG 0.63 mm) wire was used to cross the lesion. These wires offer maximal steering capacity and safety.

When the wire and catheter were safely in the stomach, the steerable wire was removed and water soluble contrast was once again injected to confirm the intraluminal position. A heavy duty exchange wire (Cook Teflon-coated Lunderquist exchange wire 0.965 mm) was then passed through the catheter and the distal tip coiled safely in the stomach. The stiffening catheter was then removed.

At this point we used either Savary-Guillard dilators, a high pressure balloon angioplasty catheter (USCI PE Plus II) or Olbert fascial balloon dilator to dilate the stricture. All of these devices easily pass over the exchange wire. After dilatation, either the balloon catheter itself or the torque control catheter originally used was passed into the gastric lumen. The stiff exchange wire was then removed and a softer wire (Cook 15 mm J TSCF 0.965 mm) was placed through the catheter with its tip coiled in the stomach. This wire facilitates passage of the endoscope through the stricture.<sup>3</sup> Laser cautery or endoscopic intubation could then be carried out.

We have seen no complications as a result of this dilatation technique apart from one patient who developed a transient tachyarrhythmia without hypotension during the procedure.

We describe a simple technique to dilate difficult oesophageal carcinomas. It can readily be done in the radiology department without major equipment purchases. We suggest that angiographic techniques have a role in the dilatation of such 'impossible' malignant oesophageal strictures prior to treatment with laser or intubation.

F E MURRAY, J GUBEN, D H BIRKETT, AND D R CAVE

*Departments of Medicine, Surgery and  
Vascular Radiology,  
Boston University Medical Centre,  
88E Newton Street,  
Boston,  
Ma 02118,  
USA.*