1 Pelican G, Henteges D, Butt J, Haag T, Rolfe R, Hutcheson D. Bacteraemia during colonoscopy. *Gastrointest Endosc* 1975; **23**: 33–5.
2 Coughlin GP, Butler RN, Alp MH, Grant AK. Colonoscopy and bacteraemia. *Gut* 1977; **18(8)**: 678–9.
3 Kiss A, Ferenci P, Graniger W, Pamperl H, Potzi R, Meryn S. Endotoxaemia following colonoscopy. *Endoscopy* 1983; **15(1)**: 24–6.
4 London MT, Chapman BA, Faoagali JL, Cook HB. Colonoscopy and bacteraemia: an experience in 50 patients. *N Z Med J* 1986; **99(800)**: 269–71.

Authors' reply

Sir
We thank Mr Drew and associates for their interest and comments on our case report. The incidence of bacteraemia following lower GI endoscopy has been reported as ranging from 1–27 per cent in a recent review[1]. Bacteria entering the portal blood during large bowel endoscopy are filtered out by the liver, hence the low reported incidence as they are not detected so readily in the systemic circulation[2]. As described in the report, the patient presented with pneumaturia; this constitutes a 'high risk' in our opinion, because there is an appreciable 'septic' complication rate in such patients. There have been no 'sporadic' cases of portal pyaemia reported in the literature following flexible sigmoidoscopy, and we believe routine antibiotic prophylaxis in such cases is reasonable.

R. T. Patel
K. I. Deen
J. W. Fielding

Department of Surgery
Queen Elizabeth Hospital
Edgbaston
Birmingham B15 2TH
UK

1 Moayyedi P, Lynch D, Axon A. Pseudomonas and endoscopy (Review). *Endoscopy* 1994; **26**: 554–8.
2 Patel RT, Jalleh RP, Glencross EJ, Vellacott KD. Should we treat bacteraemia following colonoscopic polypectomy in healthy patients? *Gut* 1992; **33(2)**: 815.

Genetic susceptibility to colorectal cancer in patients under 45 years of age

Sir
In the paper by Mr Hall *et al.* (*Br J Surg* 1994; **81**: 1485–9) the authors state that pernicious anaemia is an inherited condition not associated with the colorectum. I would like to draw your readers attention to the paper by Talley *et al*[1] in which, on the basis of a population-based cohort study, it was suggested that pernicious anaemia imposed a four-fold increased risk for colorectal cancer in the first 5 years after diagnosis of the anaemia.

A. D. Spigelman

Academic Surgical Unit
St Mary's Hospital Medical School
10th Floor
Queen Elizabeth the Queen Mother Wing
London W2 1NY
UK

1 Talley NJ, Chute CG, Larson DE *et al.* Risk of colorectal adenocarcinoma in pernicious anaemia. A population-based study. *Ann Intern Med* 1989; **111**: 738–42.

Diabetes impairs the development of early strength, but not the accumulation, of collagen during intestinal anastomotic healing in the rat

Sir
We read with interest the article by Drs Verhofstad and Hendriks (*Br J Surg* 1994; **81**: 1040–5), who examined the effects of streptozotocin-induced diabetes on intestinal anastomotic repair in the rat. They found that diabetic rats have a lower bursting pressure of the sutures and a greater incidence of 'anastomotic abscesses with essentially unchanged postoperative collagen synthetic capacity? They concluded that diabetes impairs elderly anastomotic strength in the rat intestine, but not through deficient accumulation of collagen.

In the text (*Table 2*), in full contradiction with the abstract, the authors show that abscess formation was not increased in diabetic rats and that the lower bursting pressure was due to abdominal sepsis. As eleven diabetic rats, killed 3 days after operation, had both ileal and colonic anastomoses, the rate of 'abdominal' infections must have been between seven and 11 (seven ileal abscesses and four colonic abscesses). It is well known that sepsis impairs wound healing[1] even at distant sites[2], and the authors should have considered the effects of colonic abscesses on ileal wound healing and vice versa.

Wound healing does not of course mean only collagen synthesis (certainly not at 3 days after operation), but it cannot be denied that there is a good correlation between the impairment of wound bursting strength and the inhibition of collagen synthesis, as the authors found.

Seven days after operation the only difference in wound bursting pressure was in the ileum. No significant changes were found in the colon, and the collagen synthesis was the same in diabetic and control rats irrespective of the site of the anastomosis. However, at least four of nine rats had intraabdominal sepsis (three ileal and one colonic) and four rats had zero bursting pressures in the ileal anastomosis. In other words, excluding the rats with sepsis, the bursting pressures were the same in both diabetic and control rats.

From these data, we conclude that the impairment of intestinal wound healing in diabetic rats is not related to glucose-metabolism abnormalities but to the induction of abdominal sepsis. This might be a consequence of treatment with streptozotocin, which has immunoinhibitory effects in rodents[3,4].

If the data for septic rats had been excluded from the experiment, no effect of diabetes on wound healing and collagen synthesis would have been found. On reading the papers cited by the authors (references 6, 20, 21), we found that there is no contradiction with previous experimental works on skin wound healing in diabetic rats, as significant differences in wound breaking strength are found only 10–14 days after the operation.

In conclusion, we feel that the results of Verhofstad and Hendriks were influenced by the time of observations (too early) and the rate of abdominal sepsis (too high), and do not demonstrate any true differences between intestinal and cutaneous wound healing in diabetic rats.

D. Foschi
F. Corsi
P. Cellerino
P. Cassini
E. Trabucchi

First Department of Surgery
Institute of General Surgery
and Thoracic Surgery
via F. Sforza 35
20122 Milan
Italy

1 Dehaan BB, Ellis H, Wilks M. The role of infection on wound healing. *Surg Gynecol Obstet* 1974; **138**: 693–700.
2 Carrel A. Effet d'un abcès a distance sur la cicatrisation d'une plaie aseptique. *C R Soc Biol* 1924; **90**: 333–5.

3 Ishibashi T, Kitahara Y, Harada Y, Harada S, Takamoto M, Ishibashi T. Immunologic features of mice with streptozotocin-induced diabetes. *Diabetes* 1980; **29**: 516–23.
4 Nichols WK, Spellmann JB, Vann LL, Dagnes RA. Immune responses of diabetic animals. Direct immunosuppressant effects of streptozotocin in mice. *Diabetologia* 1979; **16**: 51–7.

Authors' reply

Sir

Dr Foschi and his colleagues suggest that the results presented in the abstract contradict those in the text, i.e., *Table 2*. However, *Table 2* presents only the results of experiment 1. Abscess formation was significantly increased in diabetic rats when the results of both experiments 1 and 2 were taken together, as clearly stated in the *Results* section under experiment 2. Moreover, we have certainly not stated that the lower bursting pressure was due to abdominal sepsis. In the Discussion we raise – but cannot yet answer – the question of whether abscess formation is a cause or a consequence of deficient repair. It remains to be proven if and how sepsis affects anastomotic healing: a detrimental effect of infection on anastomotic repair has not been conclusively established[1].

It is well known that poorly controlled hereditary or chemically induced diabetes may impair certain functions of the immune system. This phenomenon is an essential part of any experimental model for diabetes; to ignore it, and exclude the rats with abdominal abscesses from the experiment as suggested, would be incorrect. Streptozotocin itself cannot be responsible for any negative effects on wound healing since loss of strength and anastomotic abscess formation can be prevented by restoring normoglycaemia with exogenous insulin (as stated in the Discussion).

The aim of our experiment was to establish the effect of experimental diabetes on early anastomotic healing, assayed at a time when strength is low and chances for leakage relatively high. Anastomotic collagen synthesis is maximally stimulated 4 days after operation[2]. As no differences in anastomotic hydroxyproline content were observed between control and diabetic groups after 3 or 7 days, and the collagen synthetic capacity was similar in both groups after 7 days, it seems highly unlikely that examination at a later time point will show any differences in this respect.

M. H. J. Verhofstad
Th. Hendriks

Department of Surgery
University Hospital Nijmegen
Nijmegen
The Netherlands

1 Gutman M, Klausner JM, Lelcuk T. Fecal peritonitis – the effect on anastomotic healing. *Eur Surg Res* 1993; **25**: 366–9.
2 Martens MFWC, Hendriks T. Postoperative changes in collagen synthesis in rat intestinal anastomoses: differences between small and large bowel. *Gut* 1991; **32**: 1482–7.

Kidney retrieval from asystolic donors: a valuable and viable source of additional organs

Sir

We read with interest the recent publication by Mr Varty *et al.* regarding the successful asystolic programme for kidney donation in Leicester[1]. We have followed the evolution of this pioneering programme and were aware of previous publications from that centre dealing with the problems of the early immunosuppressive protocol in recipients of kidneys from asystolic donors[2]. In their most recent publication[1] the group state that a 14-day course of OKT3 is given to avoid cyclosporin toxicity in these recipients, who invariably suffer acute tubular necrosis. We understand that this protocol is not now followed and that OKT3 has been abandoned.

Many transplant units initiating a programme of asystolic donors will follow the Leicester recommendations and may therefore follow the immunosuppressive protocol which is described. Consequently we feel it is important that the Leicester group clarify which immunosuppressive protocol they now recommend for recipients of kidneys from asystolic donors, and why the protocol was changed.

D. Rix
D. Talbot
E. Minford
H. Pleass
G. Proud
J. L. R. Forsythe
R. M. R. Taylor

Renal Transplant Unit
Royal Victoria Infirmary
Queen Victoria Road
Newcastle upon Tyne NE1 4LP
UK

1 Varty K, Veitch PS, Morgan JDT, Bell PRF. Kidney retrieval from asystolic donors: a valuable and viable source of additional organs. *Br J Surg* 1994; **81**: 1459–60.
2 Keluride EO, Veitch PS, Scriven SD *et al.* OKT3 as an immunosuppressive induction agent for recipients of kidneys from non-heart beating donors? No. *International Conference on New Trials in Clinical and Experimental Immunosuppression.* Geneva 1994. Abstract 43.

Authors' reply

Sir

We thank the Newcastle group for their interest in our article. In the format of a Short Note article we were unable to give in-depth details of our protocol but, as they rightly point out, we have experienced and reported adverse toxicity using OKT3 in the recipients of non-heart-beating kidneys. We have now moved to a low-dose cyclosporin regime (4 mg/kg) combined with prednisolone and azathioprine until recovery from acute tubular necrosis is evident. A standard cyclosporin and prednisolone regime is then adopted monitored by cyclosporin levels.

K. Varty

Department of Surgery
Clinical Sciences Building
Leicester Royal Infirmary
Leicester LE2 7LX

Patterns of reflux in recurrent varicose veins assessed by duplex scanning

Sir

We read with interest the paper by Messrs Redwood and Lambert (*Br J Surg* 1994; **81**: 1450–1) which reports the results of duplex assessment of recurrent varicose veins. We have a number of comments to make regarding the conclusions they draw from their work.

We recently reported the results of our assessment of 118 consecutive patients presenting with recurrent varicose veins and found a positive association between mid-thigh perforator reflux giving rise to recurrent varices, and the presence of an intact