ance of the effect in whole blood makes it unlikely that the damage arose from the direct interaction of the sound with the cell.

This work was supported by a grant from the M.R.C.

—We are, etc.,

W. T. COAKLEY
D. E. HUGHES

Microbiology Department,
University College,
Cardiff

J. S. SLADE
K. M. LAURENCE

Department of Child Health,
Welsh National School of Medicine,
Penarth, Glam

¹ Slade, J. S., and Coakley, W. T., in preparation.

² Heddle, J. A., Evans, H. J., and Scott David, in *Human Radiation Cytogenetics*. Ed. Evans, H. J., Court Brown, W. M., and McLean, A. S., p. 6, Amsterdam, North Holland, 1967.

³ United Nations General Assembly, *Report of the United Nations Scientific Committee on the Effects of Atomic Radiation*, 24th Session, Suppl. No. 13 (A/7613) New York, U.N., 1969.

⁴ Barber, J. E., *Thresholds of Acoustic Cavitation*, Acoustics Research Laboratories, Harvard University, Technical Memorandum No. 57, 1964.

⁵ MacLeod, R. M., Dunn, F., *Journal of the Acoustical Society of America*, 1966, **40**, 1202.

Problems of Rectal Prolapse

SIR,—Your leader on this (14 November, p. 381) sadly fails to lead. May I respectfully suggest, firstly, that all the many reports indicate that the Ivalon wrap that I presented on a film on numerous occasions before publishing in 1959¹ is simple, safe, and successful? Recurrences of prolapse are in the low single figures per cent. and no foreign body remains, since the material disintegrates within about a year. Secondly, all patients with complete prolapse are, of necessity, incontinent before operation. The operation I described does nothing to make things worse but permits whatever sphincteric action exists to operate to the best advantage.

Your leader rightly concludes that the aetiology and treatment of anal incontinence remain "important challenges" but the blanket statement that "incontinence had improved in about 60% of cases" does nothing to help meet that challenge, since it makes no analysis of either the preoperative condition or the type of operation employed.

CHARLES WELLS

Hoylake, Cheshire

¹ Wells, C., *Proceedings of the Royal Society of Medicine*, 1959, **52**, 602.

SIR,—Your leading article (14 November, p. 381) states that this condition occurs most commonly in infancy and old age. However, no mention was made of cystic fibrosis as a cause of rectal prolapse in infancy. Kulczycki and Shwachman¹ reported that this symptom was the initial complaint in 16 patients with cystic fibrosis and that 22.6% of 386 patients with cystic fibrosis gave a history of rectal prolapse. The National Cystic Fibrosis Research Foundation's guide to the diagnosis and management of cystic fibrosis states that cystic fibrosis is the commonest cause of prolapse of the rectum in the paediatric age group in the United States. The recent observations you refer to showing that rectal prolapse is an intussusception is of considerable interest, as an increased incidence of other forms of intus-

susception in patients with cystic fibrosis has also been noted in this clinic.

The diagnosis of cystic fibrosis should always be considered in infants and children with rectal prolapse and a sweat test is advisable if there is no obvious cause. Treatment is with a low-fat diet, pancreatic enzyme supplements, and measures to improve the pulmonary condition. Surgery is rarely required.—We are, etc.,

JOHN LLOYD-STILL
KON-TAIK KHAW
HARRY SHWACHMAN

Children's Hospital Medical Center,
Boston, Massachusetts, U.S.A.

¹ Kulczycki, L. L., and Shwachman, H., *New England Journal of Medicine*, 1958, **259**, 409.

Haemoptysis in Cystic Fibrosis

SIR,—Your recent leader "Haemoptysis in Cystic Fibrosis" (19 December, p. 702) is timely, for there is no doubt that children suffering from this disease are living longer and are presenting with previously rarely seen complications. However, this is a rare disease and the experience of any one surgeon in dealing with severe haemoptysis in these patients is likely to be limited, and for this reason a brief note on the treatment of such a patient may be of interest.

A boy of 17, known to suffer from fibrocystic disease of the lungs since early life, was referred with severe haemoptysis in February this year. At that time he was expectorating up to 300 ml of bright red blood daily. An intelligent boy, he was sure that this was coming from the base of his right lung, for prior to each episode of haemoptysis he felt the blood bubbling at the back of his right lower chest. He was treated conservatively, and fortunately after two weeks, during which time he received 8 units of blood, this symptom subsided. However, he was readmitted in October 1970 with further severe haemoptysis, and on this occasion he was producing up to 800 ml daily. He was now very anaemic and demoralized, and for this reason surgery was considered but while awaiting an elective operation he had massive haemoptysis of 1,500 ml of bright red blood. It was assumed that the bleeding was originating from a bronchiectatic right lower lobe and that the blood was coming from bronchial arteries rather than pulmonary arteries. Emergency right thoracotomy was undertaken. The lung was boggy and pale but the striking feature was the hypertrophied bronchial artery, coursing along the lower edge of the right main bronchus. This was almost the size of a normal brachial artery, 0.5 cm in diameter, giving off a small ascending and a large descending tortuous artery to the right lower lobe. The bronchial artery was divided between ligatures. Postoperatively the haemoptysis ceased dramatically although there was a further small episode one week post-operatively. He developed a severe pulmonary infection, which responded to appropriate antibiotics, and was discharged well four weeks after operation. This appears to have been a successful operative procedure, although it is likely that adventitious and equally large bronchial arteries will in due course develop and may produce further haemoptysis.

Contrary to the gloomy attitude of some writers, these patients are not necessarily disabled. This patient leads an otherwise fairly normal life.

I thank Dr. Beryl Corner for asking me to treat this patient.—I am, etc.,

G. KEEN

Bristol Royal Infirmary,
Bristol

Resuscitation Too Late

SIR,—This week I saw with my own eyes for the first time the relatively recent syndrome of a patient who was resuscitated too late—that is, after prolonged cerebral anoxia. The general practitioner is in a great state of isolation compared to those who work in hospitals. Many general practitioners who, like myself, qualified a couple of decades back have never even seen these new man-made syndromes unless they stray on them by chance, as I did. Nevertheless, unless certain criteria are established and made known, we ourselves may be guilty of resuscitating people too late, with all the anguish that results.

I feel it is long overdue that the profession was given the chance to create a new platform on which new developments may be debated up and down the country together with church leaders and others concerned with our presentday ethics. Even the question of euthanasia must constantly, by implication, be reassessed so that we may have the consolation that there exists the widest possible consensus of opinion that is constantly under review. We can no longer escape these issues and it is cowardly to do so.—I am, etc.,

ELEONORE M. ETLINGER

Little Cornard,
Near Sudbury, Suffolk

Skin Disease and the Gut

SIR,—Several important points are raised by Dr. E. J. Moynahan's interesting letter (28 November p. 559).

The first concerns the apparent difference in the incidence of rashes in coeliac disease of children and adults. We ourselves have also noticed this unexplained difference¹ and the general feeling that rashes are rare in childhood coeliacs has been supported by a recent detailed study of children with coeliac disease.²

The second point concerns the incidence of the more specific rash of dermatitis herpetiformis in these patients. From replies to a recent national questionnaire sent out through the Coeliac Society it is interesting that of the coeliacs who had a rash in about 10% this was in fact dermatitis herpetiformis. We have in our care three such patients with proved gluten-sensitive coeliac disease who have been referred to us with a rash which has turned out to be dermatitis herpetiformis and several similar cases have been reported in Britain.³⁻⁶ So far, however, no study of the bowel in juvenile dermatitis herpetiformis has been published.

Important though the association of dermatitis herpetiformis with clinical coeliac disease is, this is not really our message.^{1,7-13} In our original paper⁷ we reported that two-thirds of patients with dermatitis herpetiformis had on biopsy a jejunal mucosal appearance indistinguishable from that found in patients with coeliac disease or "idiopathic steatorrhoea." This is a separate issue from the occurrence of clinical coeliac disease and indeed most of our patients, including those with the most severe changes on mucosal biopsy, have no symptoms and some of them have no biochemical evidence of malabsorption either. It is therefore irrelevant to say, as Dr. Moynahan and many others have said, that patients with dermatitis herpetiformis have no symptoms or signs and therefore do not have an enteropathy—only jejunal biopsy in these patients will show these severe mucosal changes. Moreover,

our findings are not peculiar to Newcastle for the same high incidence of jejunal mucosal abnormalities has been found in large series from London^{14, 15} and Glasgow¹⁶ and several cases have been reported from the Netherlands,¹⁷ Canada,³ and the U.S.A.¹⁸

This means that all patients with dermatitis herpetiformis should be investigated for coeliac disease solely on the grounds that they have dermatitis herpetiformis. The evidence is now so unequivocal that the day may not be far off when any of us may be called to answer a charge of negligence if we omit this essential investigation.—We are, etc.,

JANET MARKS
SAM SHUSTER

Department of Dermatology,
Royal Victoria Infirmary,
Newcastle upon Tyne

- 1 Shuster, S., and Marks, J., *Systemic Effects of Skin Disease*, p. 81, Heinemann, London, 1970.
- 2 Robinson, D. C., Personal Communication, 1970.
- 3 Bendt, B. J., and Williams, P. B., *Canadian Medical Association Journal*, 1968, 98, 575.
- 4 Fraser, N. G., Ferguson, A., and Murray, D., *British Medical Journal*, 1968, 4, 30.
- 5 Inman, P., Personal Communication, 1970.
- 6 Wilkinson, D. F., Personal Communication, 1970.
- 7 Marks, J., Shuster, S., and Watson, A. J., *Lancet*, 1966, 2, 1280.
- 8 Shuster, S., Watson, A. J., and Marks, J., *Lancet*, 1968, 1, 1101.
- 9 Shuster, S., and Marks, J., *Proceedings of the Royal Society of Medicine*, 1969, 62, 985.
- 10 Marks, J., and Shuster, S., *Proceedings of the International Conference on Coeliac Disease*, 1969, in press.
- 11 Watson, A. J., Shuster, S., and Marks, J., *Modern Gastroenterology. Proceedings of VIIIth International Congress of Gastroenterology*, p. 798, 1969.
- 12 Marks, J., and Shuster, S., *Gut*, 1970, 11, 281.
- 13 Marks, J., and Shuster, S., *Archives of Dermatology*, 1970, 101, 452.
- 14 Fry, L., Kier, P., McMinn, R. M. H., Cowan, J. D., and Hoffbrand, A. V., *Lancet*, 1967, 2, 729.
- 15 Marks, R., Whittle, M. W., Beard, R. J., Robertson, W. B., and Gold, S. C., *British Medical Journal*, 1968, 1, 552.
- 16 Fraser, N. G., Murray, D., and Alexander, J. O'D., *British Journal of Dermatology*, 1967, 79, 509.
- 17 Van Tongeren, J. H. M., Van der Staak, W. J. B. M., and Schillings, P. H. M., *Lancet*, 1967, 1, 218.
- 18 Brow, J. R., Parker, F., and Rubin, C. E., *Gastroenterology*, 1968, 54, 1223.

Depression and Oral Contraception

SIR,—One factor that has not been mentioned in the discussion on oral contraceptives and depression is the high incidence of depression occurring in women following childbirth. These no doubt form a sizeable proportion of women attending family planning clinics and the majority of those receiving birth control advice in my own practice.

Pitt¹ found that 10% of women delivered at a London teaching hospital suffered from postpartum depression. More recently, Miss Sylvia Lutkins and I² used the Beck Depression Inventory to assess the incidence of depression associated with childbirth. At a cutting score of 17, the incidence of moderately severe depression was 10%, which is similar to the finding of Pitt. We also found that throughout the 12-month period following childbirth the incidence of moderately severe depression was constant at 10%, with about 3% of mothers severely depressed. (Incidentally, during the antenatal period 9% of mothers are moderately depressed and 3% severely depressed, while the before and after rate for moderate depression among fathers remains constant at 3%.)

The finding that particularly interested me in the paper by Dr. Brenda N. Herz-

berg and her colleagues (17 October, p. 142) was that "no difference was found between the average depression scores of the oral contraceptive and the control groups." I would have liked to have seen as much emphasis given to this finding as given to the fact that 6.6% of those taking oral contraceptives were severely depressed. The latter finding surprises me perhaps less than the former as, from my own use of the Beck Depression Inventory, I expect about 3% of women receiving contraceptive advice from me to be severely depressed at the first interview.—I am, etc.,

DEWI REES

Llanidloes, Mon

- 1 Pitt, B., *British Journal of Psychiatry*, 1968, 114, 1325.
- 2 Rees, W. D., and Lutkins, S. G., *Journal of the Royal College of General Practitioners*, in press.

Cystic Degeneration of the Popliteal Artery

SIR,—Your leading article on this subject (19 December, p. 699) is a good review of this uncommon cause of intermittent claudication in young adults. Only one point of practical importance should perhaps be added. While it is likely that most cases will go unrecognized until arteriography or operation show the typical lesion, which at once distinguishes itself from the commoner obstructions due to arteriosclerosis or Buerger's disease—that is, in the 70% or so of this age group of the population who smoke cigarettes—yet when the cystic lesion (or popliteal entrapment for that matter) brings about claudication in one of the non-smoking 30% a non-arteriosclerotic cause should be suspected. Such a case was diagnosed clinically, even before arteriography, in my report some years ago.¹ The incidence of non-smoking in male claudicators in my experience remains, as it was then, about 1%.—I am, etc.,

H. H. G. EASTCOTT

London N.W.1

- 1 Eastcott, H. H. G., *Lancet*, 1962, 2, 1117.

Spontaneous Rupture of Spleen

SIR,—I wish to report a case of spontaneous rupture of a normal spleen. Traumatic rupture of the spleen is very common while spontaneous rupture of a pathological spleen, although rare, is well documented especially in tropical diseases such as malaria and kala-azar where the spleen may be markedly enlarged. Spontaneous rupture of a normal spleen, however, is extremely rare.

A 55-year-old housewife was admitted to hospital as an emergency with abdominal pain of one week's duration. The latter was a constant dull ache in the left hypochondrium markedly accentuated by deep breathing and by coughing. There was no shoulder pain. She was certain that she had not sustained any injury. On examination she was very pale but not in pain. The relevant abnormal findings were a pulse of 120 per min.; an abdomen which was soft but with slight tenderness on deep palpation in the left hypochondrium; a haemoglobin of 5 g/100 ml; and a reticulocyte count of 7.2%. A barium meal a few days after admission showed a large filling defect in the fundus of the stomach very suggestive of a carcinoma.

The patient was transfused with packed cells and 12 days later a laparotomy was performed. A very large perisplenic haematoma was found

pushing the fundus of the stomach medially and forwards but otherwise the stomach was normal. There was about 300 ml of dark altered blood in the peritoneal cavity. The haematoma was separated easily from the stomach, left dome of the diaphragm, and abdominal wall, and in its centre was a normal-sized spleen showing a laceration extending into the splenic parenchyma. The spleen and the surrounding haematoma were removed and a liver biopsy was performed although the liver was not enlarged. No other abnormality was found in the peritoneal cavity. Histology showed a normal spleen and liver.

To exclude the diseases with which spontaneous rupture of the spleen has been associated we did repeated blood counts, bone marrow examination, plasma proteins and electrophoretic pattern, and Paul-Bunnell test, and these were all normal. She has now been followed up for over a year and remains well with a normal blood count. During the post-operative period the patient was questioned closely by different people and she was absolutely certain that she had not sustained any injury, however slight, in the preceding few weeks.

This case differs from the other reported cases of spontaneous rupture of the normal spleen in that our patient did not present with the clinical picture of bleeding into the peritoneal cavity, as is normal, but instead presented as anaemia due to carcinoma of the stomach—a very misleading presentation of an extremely rare condition.—I am, etc.,

A. GRECH

Redhill, Surrey
Redhill General Hospital,

Home Haemodialysis

SIR,—Much has been written about the difficulties of initiating haemodialysis in the home and the time-consuming frustrations that take place between the hospital and the local authority in setting up this urgent life-saving service. It is both disturbing and frightening to read that these frustrations exist in view of the detailed guidance given to local authorities by Ministry of Health Circular issued in August 1968.¹

In the district for which I am responsible there is a very simple and immediate working relationship between my department and the hospital dialysis team. As soon as it is found necessary for a patient to have home dialysis the administrator of the hospital dialysis team immediately contacts the owner of the property for permission to install equipment and adapt to the necessary requirements. Once this permission is given I am consulted in order that the adaptation work can be finalized within the provisions of Section 28 of the National Health Service Act 1946. My council is conscious of the necessity for urgent provision of all types of service covered by Section 28 and has had the foresight to delegate to its medical officer of health the giving of approval subject to availability of finance within the annual estimate. There is no valid reason for delay while awaiting committee consent.

It has been found advantageous to employ where possible the same local building contractor, who is fully aware of the building regulations and who will advise if any planning consent is required. Any such problem is then quickly resolved at local authority officer level. My records show that the time taken from first request to