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.,

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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.
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4 Barger, J. E., *Thresholds of Acoustic Cavitation*, Acoustics Research Laboratories, Harvard University, Technical Memorandum No. 57, 1964.
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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 1959[1] 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.

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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 Shwachman[1] 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 intussusception 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.,

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1 Kulcyzcki, 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 postoperatively. 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.,

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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.,

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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 difference[1] 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 paper[7] 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,