these were aged over 60 years and two men were younger. Three women and three men died of circulatory failure at the intensive care ward. Two of the women were unconscious on admission, and three men died postoperatively. Of those one was aged 33 and the other 38 years, and they died of pancreatic abscess and haemorrhage respectively. The third man died of circulatory failure 20 days after recovering from his pancreatitis.

The mortality in this series was high in the first five years, but no patient died during 1969-70, probably owing to the fact that peritoneal dialysis was started earlier and the patients were not so ill on admission. During 1971 and 1972 we have so far treated 15 cases of acute haemorrhagic pancreatitis, of whom three have died. Since this regime was introduced there have been some cases of pancreatic cyst formation which have required surgical interference. A remarkable improvement in the conditions of the patients has always occurred and the pain has usually disappeared or decreased shortly after the start of the dialysis.

The mode of action of peritoneal dialysis in acute pancreatitis is not quite clear. Several factors are probably involved—a removal of oedema and pancreatic exudate with toxic substances; correction of electrolytes and blood gases; relieving the load on the kidneys by removing toxic substances from the blood; and supply of calories by absorption of glucose from the dialysing fluid.—I am, etc.

JON GJESSING

Department of Anaesthetics,
Sundsvall Hospital,
Sweden

1 Gjessing, J., and Nilsson, L., *Läkartidningen*, 1965, **62**, 3706.
2 Gjessing, J., and Dencker, H., *Acta Chirurgica Scandinavica*, 1968, **134**, 351.
3 Gjessing, J., and Jacobs, S., *Nordisk Medicin*, 1971, **86**, 927.
4 Gjessing, J., Oskarsson, B. M., Tomlin, P. J., and Brock-Utne, J., *British Medical Journal*, 1972, **1**, 617.

Foreign Exchange Visits

SIR,—While it is common for medical specialties to have travelling clubs we believe that visits to other countries under the auspices of postgraduate medical education centres are something new, and readers may be interested to know of two successful exchange visits we have carried out.

In 1969 we asked the Danish Medical Association whether doctors from a town or towns similar in population to ours would be interested in visiting us for four or five days. As a result a group of 26 doctors and their wives came from Esbjerg and Slagelse. These two Danish towns have neither medical schools nor universities but have well developed medical services. Each Danish couple stayed with an English family matched so far as possible for age, interest, or specialty.

The programme consisted of lectures on various subjects, an explanation of the evolution and functioning of the National Health Service, discussions on current world problems such as abortion and population control, on the development of coronary and intensive care units. There was, of course, an energetic social programme.

In 1970 we were invited to Denmark. So successful was this that this year we invited doctors from Holland, and 21 Dutch doctors with their wives came from Hengelo and stayed five days. Once again this was highly successful.

We are left with the impression that these visits are not only extremely pleasurable but promote international goodwill and understanding. Our visitors were impressed with the development of postgraduate medical education in Britain. For our part our overall impression is that our outlook on medical care is very similar to that of the Danes and the Dutch. If other area clinical tutors are thinking of doing something on these lines we will be happy to give further information on how we managed these exchange visits. —We are, etc.,

I. S. HODGSON-JONES
H. J. VOSS

Kettering and District Hospital,
Kettering, Northants

Respiratory Distress Syndrome

SIR,—Your leading article on the respiratory distress syndrome (7 October, p. 2) raises interesting possibilities. The close correlation of the results of prenatal examination of amniotic fluid with the probability of developing respiratory distress syndrome[1-3] would suggest that its onset in an infant should not come as a surprise to the medical attendants. Though it may often be possible to postpone the timing of elective interruption of pregnancy there will still be cases where the child is delivered before term, and even in infants of 35 weeks gestation or more the mortality from respiratory distress syndrome is 10%.[4]

Though surfactant deficiency is an important factor in the cause of respiratory distress syndrome[5] there is confusion about the reasons for the deficiency. As you point out, immaturity is one factor but failure of synthesis is unlikely to be the only cause, since most infants who subsequently die of respiratory distress syndrome are in good condition at birth[4 5] and infants who die of respiratory distress syndrome associated with maternal diabetes have no gross surfactant deficiency at the time of death.[7] Since deficiency of surfactant is also a feature of oxygen toxicity, and since the lung pathology of oxygen toxicity and respiratory distress syndrome are similar,[6] it has been suggested[8 9] that the development of respiratory distress syndrome is due to a pathological response to the oxygen in the normal atmosphere.

This problem could be approached from two routes—either by altering the inspired gases or by supporting the mechanisms which protect the lungs against the effects of oxygen. It has been shown that in established respiratory distress syndrome by applying continuous positive pressure to the airway the oxygen tension in the inspired gases can be reduced towards normal while maintaining the arterial oxygen level.[10] If this method was applied early it might allow the oxygen tension in the inspired gases to be reduced below the concentration in the atmosphere. Recent work[9] on the role of the second gas effect on the onset of pulmonary oxygen toxicity found that helium, the lightest gas, accentuated the oxygen toxicity while the heaviest gas, sulphur hexafluoride, showed the greatest amelioration. This work suggests an alternative possibility of modifying or preventing the disease by varying the non-oxygen component to a composition less likely to damage the abnormal lungs.

In exposure to hyperbaric oxygen ascorbic acid has a protective action against the toxic pulmonary effects of oxygen,[11] and indeed the ascorbic acid appears to be mobilized to the lungs and destroyed by oxygen.[12] Pentobarbital and other anaesthetic agents have a protective effect against lung lesions which develop in association with seizures induced by hyperbaric oxygen.[13] Sympatholytic agents also have a protective action against pulmonary lesions, but in contrast to the anaesthetic agents they have no effect on the seizures.[13] Since these agents protect a lung with normal protective mechanisms against the effects of a grossly raised inspired oxygen tension they may be applicable to the lung predisposed to the onset of the respiratory distress syndrome because of immature antioxidant mechanisms.—I am, etc.,

E. L. LLOYD

Royal Infirmary,
Edinburgh

1 Bhagwanani, S. G., Fahmy, D., and Turnbull, A. C., *Lancet*, 1972, **1**, 159.
2 Clements, J. A., *et al.*, *New England Journal of Medicine*, 1972, **286**, 1077.
3 Whitfield, C. R., Chan, W. H., Sproule, W. B., and Stewart, A. D., *British Medical Journal*, 1972, **2**, 85.
4 Scopes, J., *British Journal of Hospital Medicine*, 1970. **3**, 579.
5 Reynolds, E. O. R., Robertson, N. R. C., and Wigglesworth, J. S., *Pediatrics*, 1968. **42**. 758.
6 Reynolds, E. O. R., *American Journal of Obstetrics and Gynecology*, 1970, **106**. 780.
7 Boughton, K., Gandy, G., and Gairdner, D., *Archives of Disease in Childhood*, 1970, **45**, 311.
8 Prathap, K., *Lancet*, 1972, **1**, 745.
9 Shanklin, D. R., and Lester, E. P., *Biology of the Neonate*, 1972, **20**, 140.
10 Gregory, G. A., *et al.*, *New England Journal of Medicine*, 1971, **284**, 1333.
11 Jamieson, D., and Van der Brenk, H. A. S., *Biochemical Pharmacology*, 1964, **13**, 159.
12 Shanklin, D. R., Cunningham, J. J., Sotelo-Avila, C., and Crussi, F., *Archives of Pathology*, 1967, **84**, 451.
13 Wood, J. D., in *Physiology and Medicine of Diving and Compressed Air Work*. Ed. P. B. Bennett and D. H. Elliott. London, Bailliere, 1969.

Duodenogastric Reflux and Pyloric Surgery

SIR,—Much discussion goes on about whether pyloric reflux or gastric retention causes benign lesser curve gastric ulcer. I was interested to read that Dr. C. D. T. MacLean (14 October, p. 113) found pyloric reflux in 40% of patients with a normal barium meal but a higher incidence in those with gastric ulcer. Du Plessis,[1] who supported the reflux hypothesis, concluded that gastric resection must be done for gastric ulcer. So also did Mr. W. M. Capper (personal communication).

Those who believe that gastric ulceration is caused by retention secondary to duodenal ulceration or pyloric channel disease are led immediately to vagotomy—and indeed to highly selective vagotomy without drainage—for all benign gastric ulcers. I have treated 150 patients with benign lesser curve gastric ulcer by vagotomy since 1962. In 31 of these highly selective vagotomy without drainage has been used. Our results are the same as those of Johnston and his colleagues[2] in Leeds. The operation seems to be as curative for gastric ulcer as it is for duodenal.

Many of the lesser curve gastric ulcer specimens in the pathology museums in London show obvious evidence of pyloric channel disease. The important work of Liebermann-Meffert and Allgower[3] in Basle confirms this association. They describe all the known pathological features of pyloric channel disease in their specimens. No doubt