varying from 14% to 53%.¹ Pleural effusion occurs in 3-17% of patients. These are often haemorrhagic, usually painless, without pleural features, and three times more likely than other effusions to be left sided.² Other forms of intrathoracic involvement are elevation of a hemidiaphragm, atelectasis, pulmonary infarction, and pneumonia with various infiltrates present.³ It is generally agreed that pulmonary complications are more frequent in fatal cases, especially if true adult respiratory distress syndrome occurs when the mortality is high.² Bronchopleural fistula is also reported, and the possibility that this complication will develop was the rationale for the removal of the enzyme-rich pleural fluid. The possible routes of pulmonary involvement have been well reviewed.¹ There is also agreement that an amylase level in the pleural fluid elevated above that of the serum amylase assayed simultaneously is diagnostic, minor elevations occurring only infrequently.¹

Acute pancreatitis can occur following trauma or upper abdominal surgery and also occurs with increased frequency in patients taking corticosteroids. It is, however, unusual for pleuropulmonary involvement to be the presenting feature, as was the case with our patient. This case illustrates the value of a simple laboratory investigation and gives some indication of the circumstances in which pancreatic involvement may be the primary pathology.

We wish to thank Mr A Lewis and Professor A V Hoffbrand for enabling us to study this patient.

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¹ Kaye MD. *Thorax* 1968;23:297-306.

² McKenna JM, et al. *Chest* 1977;71:197-204.

³ McWilliams A, Cross R. *Am Surg* 1974;40:448-52.

Comparison of continuous and intermittent papaveretum after cholecystectomy

SIR,—There is nothing revolutionary about the conclusions that Dr J A Catling and others draw from their comparison of continuous and intermittent narcotic analgesia for the treatment of postoperative pain (16 August, p 478). The effects of narcotics on the respiratory centre have been known for many years and the low therapeutic index is well documented. This paper, like all those assessing the efficacy of analgesics, suffers from the wide variability in the subjective response to pain, the total dose in each group varying more than sevenfold.

Table II records the change in breathing pattern of the seven patients in the intramuscular group with that of seven patients in the intravenous group. No conclusions can be drawn from this table without knowing the normal respiratory pattern during sleep of each patient. Case 13, a woman of 73, appears to have not only periods of prolonged apnoea but also a persistently low respiratory rate. However, over the 48-hour period she received a total dose of only 37 mg papaveretum, about 0.01 mg/kg/h, an unlikely dose to depress respiration to that extent.

The authors were fortunate in having the approval of their hospital ethical committee

for the insertion of arterial cannulas into each patient to assess the effect on blood gases of the two forms of analgesia. It was here that concrete evidence could have been obtained; unfortunately it was not. The arterial PCO₂ levels were marginally raised in the group with the better analgesia but levels around 6.2 kPa are of doubtful clinical significance. However, no conclusions can be drawn from Po₂ results owing to the enormous difference in the two groups after anaesthesia but before analgesia had commenced.

Finally, when the analgesic action of drugs in the treatment of postoperative pain is studied, the findings must be influenced by the peroperative analgesic used, particularly if given as required in supplementary doses. Would it not have been tidier to have given each patient a standard dose of analgesic at the onset of anaesthesia?

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*.*We sent this letter to the authors, who reply below.—ED, *BMJ*.

SIR,—We agree with Dr Church that there is nothing revolutionary about the idea put forward in our paper (16 August, p 478)—that apnoeic periods in the postoperative period, whatever their cause, will lead to potentially hazardous fall in PaO₂. However, Dr Church's comments about respiratory depression are difficult to follow. On the one hand, he is already convinced of the respiratory depressant effects of narcotics. On the other hand, he searches for "concrete evidence" of respiratory depression in our blood gas data yet fails to recognise it. If he doubts the significance of a mean increase in PaCO₂ to 6.2 kPa what does he make of PaCO₂ values of 2 SD above that—that is, 8.0 kPa? There must be few traditional clinicians who fail to understand the significance of a PCO₂ of 60 mm Hg.

But this is not the real point of our paper. Our attention in this study was drawn to the respiratory patterns in our postoperative patients. Lugaresi *et al*¹ have reported that some normal subjects show apnoeic periods of not more than 5-15 seconds during light and rapid-eye movement (REM) sleep and Guilleminault *et al*² record a maximum number of 10-second apnoeas of four per hour (mean 0.5/h) in normal subjects. Because of this we chose a greater than 20-second apnoeic period to indicate abnormal apnoea and we found that these apnoeic periods were invariably more frequent than four an hour. It is remotely possible that, by chance, three out of seven subjects in our intravenous group had the very rare pathological sleep apnoea syndrome; otherwise we conclude that this was related to the drug regimens used in these patients.

We pointed out in our paper that the intravenous infusion was discontinued within 48 hours if pain had ceased. Case 13 was such a patient; the 37 mg of papaveretum was given in 24 hours, so the infusion rate was 0.02 mg/kg/h. Dr Church, while accepting that there is a wide variability in response to pain, finds the wide variability in respiratory responses unbelievable. Yet in his own paper, of three patients who had respiratory depression requiring reversal with naloxone, two were receiving an opiate dose only a factor of 1.5 greater than our patient—in whom, he claimed, respiratory depression was unlikely.

We share with Dr Church an interest in

providing adequate postoperative pain relief. Apnoeic periods, no matter how produced, result in a fall in PaO₂ no matter what the initial PaO₂. To investigate the severity of these changes we are currently making continuous measurements of breathing pattern, oxygen saturation, and sleep staging in the postoperative period. The episodes which are life threatening last for a minute or so and cannot be recognised by intermittent measurements of respiratory rate or arterial blood gas tensions.

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¹ Lugaresi E, Coccagna G, Cirignotta F, et al. *Adv Exp Med Biol* 1978;99:35-45.

² Guilleminault C, Van den Hoed J, Mitler MM. In: Guilleminault C, Dement WC, eds. *Sleep apnea syndromes*. KROC Foundation Series, vol 2. New York: Alan R Liss Inc, 1978:1-12.

Anthropometric and dynamometric variables and serious postoperative complications

SIR,—I was most interested in the article by Mr A M Klidjian and others (4 October, p 899). May I make one comment? It does not detract in any way from the possible value of their screening test, but I ask myself whether it is really detecting malnutrition, and if so to what extent.

It is interesting that among the measurements taken weight loss, which is probably consequent on the illness, showed no significant correlation with complications; whereas measurements of muscularity and body shape do so. Among these forearm girth, which is least affected by subcutaneous fat, is the most significant, together with strength of handgrip. The relationship between body type and strength as measured by lumbar pull dynamometer was reported for healthy subjects in 1958.¹ I suggest that body type with its genetic element is a more important contributory factor than malnutrition. More muscular people have a relatively greater reserve store of protein on which to draw in time of adversity.

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¹ Parnell RW. *Behaviour and physique: an introduction to practical and applied somatometry*. London: Edward Arnold, 1958: 54-8.

Antibiotics in surgical treatment of acute abscesses

SIR,—Your correspondent Mr Roger Grace states that primary suture of an abscess after incision and curettage under antibiotic cover is not appropriate for anorectal sepsis (20 September, p 813). He seeks to establish this point from the information contained in our paper (12 July, p 111). This paper was not written to give specific guidance on the treatment of acute anorectal infection. Its purpose was to report that there is no delay in the healing time of an abscess if the customary postoperative course of antibiotic therapy is omitted, provided that the surgery has been performed under an effective antibiotic cover.

Apparently Mr Grace does not appreciate that we make a clear distinction between an