cases, although in two female patients this was not obtained until necropsy. There were 19 female and two male patients, the mean age of presentation was 57.9 years, and seven patients (33%) were over the age of 65.

These data reveal that in Tayside the annual incidence of primary biliary cirrhosis is 10.6 per million. On 31 December 1979 16 patients with the diagnosis were still alive, revealing the point prevalence as 40.2 per million. The incidence figure is almost twice that found by Dr Triger in Sheffield (5.8 per million), and by other workers in NE England (6.0 per million—as reported at the September meeting of the British Society of Gastroenterology by Hamlyn *et al.*). The lower value for point prevalence may indicate higher mortality in Tayside, although it is similar to the figure in the Newcastle upon Tyne study (47 per million).

The weakness in the Tayside data is the paucity of information on the number of non-biopsied cases during the study period, and on the number of biopsied cases alive prior to 1975. An attempt is being made to rectify this problem at present, but it is unlikely that these gaps in information have distorted the annual incidence figures greatly or significantly altered the point prevalence as late as the end of 1979.

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¹ Sherlock S, Scheuer PJ. *N Engl J Med* 1973;289: 674-8.

Finger wrinkling after immersion in water

SIR,—Dr G Alvarez and others question the usefulness of the finger wrinkling test, as one-third of the patients investigated by them did not show any skin wrinkling (30 August, p 586).

I recall Mr Wilfred Hynes, at a British Association of Plastic Surgeons meeting in Bristol some years ago, pointing out that when one describes a new operation or test your colleagues do two things—first they modify the procedure and then they come back and tell you that it does not work. The authors kindly refer to my description of the test, but I must point out that they did not carry out the test as described by me.¹ I clearly stated that the hand and forearm should be immersed in water at 40°C for 30 minutes. They did not immerse the forearm, nor did they keep it in water for 30 minutes.

I carry out this test as a routine on patients attending my clinic and find it most useful. A small proportion, perhaps 10-15%, do not show skin wrinkling and in these the test is of no use, but neither is it misleading. Prospective users of this test should also be warned that it does not become positive for a few days after peripheral nerve injury.

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¹ O'Riain S. *Br Med J* 1973;iii:615-6.

SIR,—Dr G Alvarez and others (30 August, p 586) state that it is safe to say that sympathetic function is preserved if finger wrinkling after immersion in water is observed. However, Bull *et al.*¹ showed that a sympathectomised hand deprived of its blood supply will wrinkle normally. The corollary may well be true—that a hand with an intact sympathetic supply but a high blood flow might not wrinkle. This would explain the large percentage of false-positive results that Dr Alvarez and his colleagues found. It is known that an individual may have different flow rates in the two hands. It has been suggested that the dominant hand has a higher flow rate.² They tested only one hand: was it the patient's dominant hand?

I feel that they have usefully pointed out the possibility of a false-positive result occurring with this test. It would have been even more useful to have tested both hands to ascertain whether the variance in their results was symmetrical. If they had done so we would now know whether the opposite hand could be used as a control in cases of unilateral disease such as in peripheral nerve injury.

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¹ Bull C, Henry JA. *Br Med J* 1977;ii:551-2.

² Kumar PP, Kovi J. *JAMA* 1978;70:837-40.

*We sent this letter to the authors, and Dr Alvarez replies below.—ED, *BMJ*.

SIR,—Testing for finger wrinkling was carried out under ordinary bedside conditions. Our habit was to use the right hand as a rule, but this was not invariable and dominance was not ascertained. Mr Williams's theoretical objection therefore stands, although we feel that if in normal individuals wrinkling in only one hand after prolonged wetting were a habitual occurrence attention would have been drawn to it.

A number of our subjects were manual workers. It could thus be argued that mild thickening of the skin over finger pulps and hypothetical trauma to peripheral nerves could account for some of our false positives, but not for as many as we found.

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Porphyria cutanea tarda and beta-thalassaemia minor with iron overload

SIR,—I would like to answer the points raised by Dr R S Day and his colleagues (20 September, p 809) concerning my case report of familial porphyria cutanea tarda and β -thalassaemia minor in a mother and daughter (24 May, p 1255).

(1) They state that the urinary uroporphyrin levels quoted are low for overt porphyria cutanea tarda in their experience. It is clearly stated in the report that porphyria cutanea tarda was not clinically overt in 1968 when the urinary uroporphyrins were measured. I would also refer them to the extensive experience of Harber and Bickers,¹ who state that the levels of urinary uroporphyrin vary widely and cannot be correlated with the degree of clinical involvement in any absolute manner.

(2) Dr Day and his colleagues state that a full porphyrin analysis is necessary to confirm the diagnosis of porphyria cutanea tarda, and raise the possibility of variegate porphyria in the mother. A full biochemical porphyrin analysis is, of course, necessary for accurate diagnosis and it was performed in both mother and daughter, but for

reasons of brevity details of the full biochemical porphyrin analysis were not included in the short report. However, raised urinary uroporphyrins and low (as reported in both patients) or slightly elevated urinary coproporphyrins are characteristic of porphyria cutanea tarda unlike variegate porphyria, where raised urinary coproporphyrins predominate.^{1,2} Urinary porphobilinogen levels (usually raised in variegate porphyria²) were repeatedly normal in both mother and daughter, (characteristic of porphyria cutanea tarda). Elevated faecal coproporphyrins were detected in the mother in 1968 but have not been found since. Faecal protoporphyrin excretion has been repeatedly normal in both mother and daughter (characteristically elevated in variegate porphyria and normal in porphyria cutanea tarda).²

(3) Clinically, despite a long follow-up of 16 years the mother has not shown any acute abdominal crises or neuropsychiatric episodes to suggest a diagnosis of variegate porphyria. Both patients exhibited hepatic siderosis and urinary uroporphyrin excretion decreased on iron removal, a characteristic feature of porphyria cutanea tarda.^{1,2}

I would like to thank Dr Day and his colleagues for drawing attention to the typographical error resulting in the omission of an important line in the published text (see correction in this issue, p 1040). However, I strongly submit that on the evidence of the clinical and biochemical data presented the diagnosis in mother and daughter is porphyria cutanea tarda.

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¹ Harber LC, Bickers DR. In: Malkinson FD, Pearson RW, eds. *Yearbook of dermatology*. Chicago: Yearbook Medical Publications, 1975:9-47.

² Meyer UA. In: Isselbacher KJ, ed. *Principles of internal medicine*. 9th ed. New York: McGraw Hill Inc, 1980:494-500.

Laboratory features of pleural effusions

SIR,—Following your leading article on laboratory features of pleural effusions (20 September, p 763), we would like to present a recent case in which an additional simple laboratory test was of value in the diagnosis of a pleural effusion of uncertain origin.

A 22-year-old Iranian design student was admitted for elective splenectomy. Idiopathic thrombocytopenic purpura had been diagnosed some six years previously, and she had been maintained on corticosteroid therapy. The surgical resection was straightforward with minimal trauma, and the immediate postoperative course was quite satisfactory. Two days postoperatively she developed a tachycardia, fever, leucocytosis, and left pleuritic chest pain, although no haemoptysis and no sputum. Chest radiography showed some collapse and a small effusion at the left base. A ventilation and perfusion lung scan did not show any features of pulmonary embolism, and this was thought to be postpneumonic in origin. There was no clinical improvement with physiotherapy and broad-spectrum antibiotics, and repeated chest x-ray examinations over the next week confirmed the auscultatory findings of a rapidly enlarging left pleural effusion. This was repeatedly aspirated, and 1150 ml of heavily bloodstained fluid was removed. This was sterile on culture, and gas-layer chromatography showed no metabolites of anaerobic infection. Although there had been little clinically to suggest pancreatitis, the amylase content of the pleural fluid was assayed and found to be markedly elevated at 2352 and 2325 U/l. The serum amylase concentration at this time was 310 U/l (normal 70-300 U/l). Following complete aspiration the clinical features returned to normal, and the patient was discharged.

Pleuropulmonary complications are well documented in pancreatitis, with an incidence

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