course to the duodenum and ascending to the stomach by way of the pylorus. Bilateral selective vagotomy must become the standard surgical operation; it gives the best results in the prevention of post-vagotomy diarrhoea. Our concern is not the possible existence of such hypothetical trunks, it is to find and deal with the main posterior trunk which supplies the coeliac division and to section all trunks, however small, lying on the oesophagus.

I believe the insulin test is of no practical value. I agree with Mr. Lythgoe¹ when he states: "Routine employment of the insulin test post-operatively will not help in the management of the individual patient . . ." and, "Clearly, it would be of great value to have a test which could detect an incomplete vagotomy at the time of operation and would allow the surgeon to make a further search for the remaining branches." This is exactly what the stimulation test does. He goes on: "There is no doubt that the electrical stimulation test can detect persisting vagal branches on the lower oesophagus, and that these account for some cases of incomplete vagotomy."

Clark and Murray² compared the two tests. They found the stimulation test "fallacious in indicating complete vagotomy when the insulin test was later found to be positive" in only one of 100 patients, and in this one case their pressure readings were very low. Clearly there was something wrong with the test in this case. I have used the stimulation test in some 400 patients since 1957; there has been only one recurrent ulcer needing operation. The first 200 were followed in great detail and the results published.³ Only one patient has come to further operation, and he for incomplete section. This, I believe, was due to a technical error in the test in its early history. He has remained well since his incomplete vagotomy was completed.

Without the electrical stimulation test 30% of our own cases would be incomplete according to its standards. Clark *et al.*⁴ studied two series, one with and one without the stimulation test, and wrote: "When the test is successfully applied the number of incomplete nerve sections is negligible. On the other hand, in the absence of the test and despite careful dissection at the lower end of the oesophagus it is estimated that the proportion of inadequate vagotomies is about 10%."—I am, etc.,

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Spontaneous Biliary Peritonitis

SIR,—I was most interested to read Mr. A. K. Dhar's account (7 October, p. 1198) of his three cases of spontaneous biliary peritonitis. I have always been interested in this condition, firstly because the title is a complete misnomer, the condition never being spontaneous, and secondly because the peritonitis, if it exists, is probably not due to bile. Uncomplicated bile in the peritoneal cavity causes minimal disturbance.

In the last four cases of peritoneal biliary effusion that have come my way I have in every one been able to demonstrate perforation of the lower part of the common bile-duct. In the presence of gross oedema and discoloration this is often difficult, and if the perforation is small failure to demonstrate it is probably of no consequence.

Two of these cases were traumatic, one associated with stones and one without obvious cause. May I suggest that in Mr. Dhar's cases it is much more likely that the initial pathology was in the lower part of the common bile-duct and that the pancreatitis was secondary, the result of oedematous obstruction of the closely associated duct of Wirsung?

Some time ago I was just completing an operation for a simple perforated duodenal ulcer when I received an urgent message from the Path. Lab. that the serum amylase, requested without my knowledge, was over 2,000 Somogyi units. I was pleased but not surprised, as it was in line with a view I have held for many years that the common acute oedematous, as opposed to the acute haemorrhagic, pancreatitis is probably always due to oedema from an extra pancreatic cause, the most likely lesion being in the biliary tract. Hence the universally good results no matter what form of treatment, or absence of treatment, is employed.—I am, etc.,

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Management of Respiratory Failure

SIR,—I wholeheartedly agree in principle with Dr. D. C. S. Hutchison and others (7 November, p. 1159) that oxygen therapy should be used in a controlled manner to produce "safe" levels of arterial oxygenation—that is, a P_{O_2} and saturation above those causing dangerous hypoxia but not necessarily in the normal range. I would, however, interpret their findings differently and contend that in fact they point to a simpler regimen than the authors suggest. They perhaps over-estimate the dangers of moderate hypoxaemia and acidaemia; I would accept as "safe" an arterial P_{O_2} above 40 mm. Hg, a saturation above 70%, and a pH above 7.22 (H^+ concentration below 60 nanonormal).¹ However, even if their "even safer" criteria (P_{O_2} above 50, pH above 7.25) are applied, their results still point to the following conclusions:

- (1) It is safe to assume that raising (as opposed to lowering) the inspired O_2 concentration to 30% will abolish dangerous hypoxaemia in these patients.
- (2) The pH and P_{O_2} only fell below safe levels when the inspired O_2 concentration had previously been raised above 33% (Cases 2, 3, 7, 9).
- (3) Measurements of pH and/or P_{O_2} were not necessary if the inspired O_2 concentration had not been raised above 33% and the PCO_2 was below 100 mm. Hg.

Our practice is to measure the PCO_2 on admission, and, if it is above 50 mm. Hg, to start O_2 therapy at an inspired concentration of 24½%, thereafter increasing to 28 and (sometimes) to 33% at approximately hourly intervals, provided the patient does not become drowsy or the PCO_2 rise excessively. If the patient does become drowsy, if coughing

becomes ineffectual, and if the PCO_2 rises excessively we employ the other therapeutic measures in the "crescendo" advocated by Dr. Hutchison and his colleagues—while keeping investigations piano. But the therapeutic fortissimo must be achieved within a few hours if severe underventilation, narcosis, and deterioration of the lungs are to be avoided. *We do not begin O_2 therapy with an inspired concentration over 30% and then, when in difficulties, reduce the inspired O_2 ; nor do we wait for clinical evidence of hypercapnia before measuring PCO_2 because, by the time such evidence is clear, the damage is done.*

None of over 50 patients in respiratory failure cared for in this way from admission has developed a PCO_2 over 100 mm. Hg.

If a patient has become unconscious owing to uncontrolled O_2 therapy Dr. Hutchison's results support our experience that the inspired O_2 concentration should not be reduced until steps have been taken to improve ventilation and until the hypercapnia has been brought under control—that is, until the PCO_2 has fallen below 100 mm. Hg. In such cases measurements of P_{O_2} and pH (or bicarbonate) are interesting; measurements of PCO_2 are almost essential.

I have no wish to be reactionary and will welcome the day when all hospitals have facilities for the continuous monitoring of the blood gases immediately available, 24 hours a day, seven days a week. But until that day comes most patients with respiratory failure can be satisfactorily managed with simple facilities already immediately available.—I am, etc.,

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REFERENCE

- ¹ Campbell, E. J. M., *Ann. N.Y. Acad. Sci.*, in press.

Histoplasmosis in Cyprus

SIR,—The occurrence of histoplasmosis in Cyprus reported by Wing Commander D. J. Stoker (26 September, p. 793) is interesting. It is difficult, however, to accept Dr. Stoker's suggestion that the encephalitis in Case 1 was due to histoplasmosis. Although Dr. Stoker does not give the result of a repeat lumbar puncture, he implies in his case history that the patient recovered from the illness spontaneously. This is contrary to the natural history of histoplasmosis of the nervous system, which is invariably progressive in nature unless the patient has received specific antifungal therapy. It is also generally accepted that histoplasmosis of the nervous system only occurs in the disseminated forms of the disease. Dr. Stoker reports no signs of dissemination of the disease except one gland in the cervical chain. If the disease was disseminated, one would expect a palpable spleen or liver, although this is not mandatory.

In any case, may I suggest that the patient be very carefully followed and his cerebrospinal fluid studied at regular intervals. Also that an intensive search for dissemination of the disease (including blood and bone-marrow culture, for instance) be made.—I am, etc.,

Florida, Transvaal,
South Africa.

P. J. CUSINS.