pupils; and in another, a woman of 38, the clinical picture closely resembled disseminated sclerosis. Nearly half (13 cases) were over the age of 60, and 6 of them were over the age of 70. It came as a surprise to find such a large number in my files. Comparable figures in the same period of other neurological diseases are subacute combined degeneration 32 cases, syringomyelia 27 cases. The figures confirm the view that the era of neurosyphilis is still very much with us.—I am, etc.,

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Gastrectomy and Vagotomy

SIR,—We read with interest the article by Mr. Alan G. Cox (3 February, p. 88), comparing symptoms after vagotomy and partial gastrectomy.

Perhaps it should be made clear that in the partial gastrectomy cases referred to the anastomosis has been made by bringing up a jejunal loop to reach the gastric stump, either in front of or behind the colon. We have shown that if this technique is avoided and a no loop gastrectomy is made¹ those sequelae in Mr. Cox's list which cause the real distress are virtually eliminated. The loss of pyloric function and control in this as in most operations for duodenal ulcer entails the need for meals to be taken slowly, reducing the sweet course, and taking drinks at other times, but this is not important: the crux of the matter is that those sequelae such as bile vomiting, recurrent ulcer, and insidious obstructive symptoms which make up the "gastric cripple" are avoided by the no loop technique.

The best surgical procedure for the intractable duodenal ulcer will remain debatable for many years, but it is misleading to compare gastrectomy without qualifications—loop or no loop—with other methods such as vagotomy.—We are, etc.,

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REFERENCE

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Aldosterone and Electrolyte Movements in the Colon

SIR,—May I reply to the letter of Dr. Oliver Wrong (10 February, p. 379) written in response to our paper on the altered colonic absorption and secretion of water and electrolytes in a patient with primary aldosteronism (13 January, p. 93)?

Firstly, it is not very surprising that in our patient sodium absorption by the colon was within normal limits. After all, sodium retention by the kidney is not a feature of primary aldosteronism, and, as we discussed in our paper, in this disease the colon may "escape" in a similar manner to the kidney from the sodium-retaining activity of aldosterone. Secondly, I do not think that Dr. Wrong is quite fair either to us or to Levitan and his colleagues¹ by implying that grossly dissimilar results can be obtained by small differences in experimental technique. The

composition of the solution perfused through the colon, the difference between our study and those of Levitan *et al.*, is critical, and the choice of perfusion solution requires considerable care. Thus Levitan¹ admitted that he was unable to evaluate the effect of 9- α -fluorohydrocortisone on potassium handling by the colon because the solution which he had used did not contain potassium.

Although I must congratulate Dr. Wrong and his colleagues for the excellent demonstration of the action of aldosterone on electrolyte handling by defunctioned colon, distal to a transverse colostomy, I cannot agree that this type of patient should be preferred for studies of colonic absorption. Firstly, to have normal colonic mucosa distal to a transverse colostomy is very rare.² Surely colon affected by diverticulitis to such an extent that a colostomy was required cannot be regarded as normal? Obviously it would be rarer still to encounter a patient with a colostomy and primary aldosteronism. Moreover, it would be impossible to study patients with ulcerative colitis³ or a villous papilloma,⁴ in whom colostomy is often unwise or unnecessary. Finally, by using patients with a transverse colostomy only the left side of the colon can be studied. We are more interested in the absorptive capability of the entire colon, especially because the right colon has a greater capacity for absorption than the left.⁵ For these reasons I feel that we shall have to continue with what Dr. Wrong regards as more complicated experiments.—I am, etc.,

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Haemophilus Epiglottitis

SIR,—Dr. L. Crome and others (24 February, p. 508) state, "In spite of stridor and croup in our two cases the fatal outcome was not due to upper respiratory obstruction, and intubation was not helpful. Death was probably caused by pulmonary collapse, peripheral circulatory failure, and rapidly developing cerebral oedema."

The three pathological findings to which they refer are all the end result of other processes, and can be explained by the presence of respiratory obstruction. Hypoxia and hypercarbia are well-known causes of cerebral oedema; respiratory and/or metabolic acidosis will cause "peripheral circulatory failure" and could explain the failure of intubation alone to produce clinical improvement; and respiratory obstruction will cause pulmonary collapse.

It is difficult to understand how they can consider that upper respiratory obstruction did not cause death (in the sense that it inexorably led up to the final collapse) when one child had stridor and was dyspnoeic and cyanosed, and the other had stridor, tachypnoea, and tachycardia.

Unless Dr. Crome and his colleagues can produce evidence of a normal acid-base state, and show that cerebral oedema was not due to hypercarbia and/or hypoxia, I do not think that their implication that *Haemophilus influenzae* infection of the epiglottis causes death other than from the sequelae of its local effect is valid. If they have drawn attention away from the importance of relief of respiratory obstruction, the correction of hypoxia and hypercarbia, and the treatment of metabolic acidosis and cerebral oedema, if present, they have not helped the management of this difficult disease.—I am, etc.,

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SIR,—If endotracheal intubation was "not helpful" in the fatal cases of haemophilus epiglottitis described by Dr. L. Crome and others (24 February, p. 508) might this have been because it was left too late?

Early tracheal intubation in infants with respiratory distress bypasses any upper respiratory obstruction, facilitates aspiration of exudate and secretions, reduces dead space, and enables intermittent positive pressure respiration to be carried out; once the child is adequately oxygenated elective tracheostomy can be performed. Because resistance to gas flow in the respiratory passages varies inversely as the fourth power of the diameter, the smallest degree of airway obstruction in a child can produce an intolerable increase in airway resistance. Thus if stridor occurs with signs of hypoxia (cyanosis, tachycardia, restlessness) tracheal intubation is needed urgently.

Dr. Crome concludes that death was not caused by upper respiratory obstruction but was probably due to pulmonary collapse, peripheral circulatory failure, and cerebral oedema. If oxygen therapy is given without relieving airway obstruction absorption collapse of the lung becomes very likely; and hypoxia is a very potent cause of circulatory failure and cerebral oedema.—I am, etc.,

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Deaths from Asthma

SIR,—I would like to make a few points on the management of acute and chronic bronchial asthma. You state in your leading article (10 February, p. 329) that practitioners should give patients in status asthmaticus intravenous or intramuscular hydrocortisone every 15 minutes. Oral prednisolone given in a dose of 20 mg. before the patient is sent to hospital, where he can be given a further 20 mg., will probably be of greater value. This is because the blood levels produced by parenteral hydrocortisone do not last very long, especially when given intravenously. The bronchodilator effect of corticosteroids takes from six to 10 hours to become established. Oral prednisolone is well absorbed and maintains adequate blood levels till this effect is produced. We usually find 40 mg. a day of prednisolone an adequate dose. The arterial PO_2 of patients in status