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.,

K. W. G. HEATHFIELD.

London W.1.

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.,

HERMON TAYLOR.

DOUGLAS EADIE.

London Hospital,
London E.1.

REFERENCE

- ¹ Taylor, H., and Eadie, D. G. A., *Brit. med. J.*, 1967, 1, 15.

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.,

R. SHIELDS.

Surgical Unit,
Royal Infirmary,
Cardiff.

REFERENCES

- ¹ Levitan, R., and Ingelfinger, F. J., *J. clin. Invest.*, 1965, 44, 801.
- ² Levitan, R., *J. Lab. clin. Med.*, 1967, 69, 558.
- ³ Annis, D., and Alexander, M. K., *Lancet*, 1952, 2, 603.
- ⁴ Harris, J., and Shields, R., *Gut*, 1967, 8, 633.
- ⁵ Shields, R., *Brit. J. Surg.*, 1966, 53, 893.
- ⁶ Levitan, R., Fordtran, J. S., Burrows, B. A., and Ingelfinger, F. J., *J. clin. Invest.*, 1962, 41, 1754.

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.,

H. G. PLEDGER.

Royal Aberdeen Hospital
for Sick Children,
Aberdeen.

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.,

Epping, Essex.

J. D. HILL.

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

asthmaticus is often very low (well below 40 mm. Hg), which makes them highly vulnerable to sudden death. The single most important early measure in preventing death in these patients is probably giving them oxygen. Their PCO_2 is usually satisfactory, and 100% oxygen can be given with safety. General practitioners should perhaps keep a portable supply of oxygen to be given to the patient while waiting for the ambulance, where the oxygen should be continued. Simple bronchodilators like aminophylline do not appear to influence the arterial PO_2 , but by producing some bronchodilatation they may produce subjective relief and reduce the work of breathing.

There is a prevalent fear among general practitioners of giving patients with chronic asthma corticosteroids. Depriving some chronic asthmatics, especially young ones, of long-term corticoids can have tragic repercussions. Apart from the social and educational consequences that uncontrolled chronic asthma can produce, it can result in irreversible pulmonary damage at an early age. The assessment of chronic asthmatics should be carried out only in hospital, where true responders can be selected for long-term corticoid therapy.¹ The important function of the general practitioner is to recognize the chronic asthmatic for hospital corticoid assessment.—I am, etc.,

A. J. AKHTAR.

University Department for
Respiratory Diseases,
City Hospital,
Edinburgh.

REFERENCE

- Walsh, S. D., and Grant, I. W. B., *Brit. med. J.*, 1966, 2, 796.

SIR,—Twenty years ago asthma was one of the commoner emergencies for which the general practitioner was called out. At the present day emergency calls for asthma seem to be much less common. Twenty years ago the general practitioner gave the patient an injection of adrenaline or aminophylline and waited until he was satisfied that the attack was under control. If he failed to control it he sent the patient into hospital. At the present day, however, instead of calling out his general practitioner the patient in an acute attack falls back on his pressurized aerosol.

There are thus two new factors present. First, treatment at the outset of the attack is by aerosol where formerly it was by injection. Secondly, the general practitioner is no longer on the scene at this comparatively early stage of the attack to take control of the situation and send the patient into hospital if he thinks it is necessary. Indeed, he may never see the patient at all. In 25 recent fatal cases of asthma studied by the Retrospective Study Group of the Royal College of Practitioners (unpublished) 15 patients died at home—nine before the general practitioner reached them.

Perhaps the absence of the general practitioner at an early stage of the attack is the crucial factor in the rising death rate (Dr. F. E. Speizer and others, 10 February, pp. 335 and 339). In his absence patients who would formerly have been sent to hospital may now remain on inadequate self-medication for too long. It would seem that what we need to know now is whether 10

years ago the majority of asthmatics died at home, as is happening now, or whether, as one suspects, they died in hospital.—I am, etc.,

PETER A. WALFORD,

Recorder, Retrospective Survey Group,
Royal College of General Practitioners,
Felsted, Essex.

Coal Gas and the Brain

SIR,—The points made in the letter from Drs. H. J. S. Matthew and A. T. Proudfoot (9 March, p. 638) are quite valid, but I think require further discussion. It is quite true that the overall prognosis for carbon monoxide poisoning is good provided that the patient is admitted to hospital at an early stage of the illness. In our paper¹ the late Dr. H. Garland and I suggested the use of hyperbaric oxygen for severe carbon monoxide poisoning. The main difficulty in suggesting this form of treatment is of course the very restricted availability of suitable pressure chambers. However, since it has been shown² that the clearance of carbon monoxide occurs twice as quickly with hyperbaric oxygen as it does with a mixture of carbon dioxide and oxygen, in theory, at any rate, this method should remain the treatment of choice for patients in deep coma with high levels of carboxyhaemoglobin. Although in general the prognosis is good, it is not uniformly so, as indicated by the patients we have described¹ and by the results obtained by Richardson *et al.*,³ who found a partial recovery in 10% and a fatal issue in 20% of their series of 85 patients admitted to a large general hospital.

It is not possible to draw conclusions by comparing two series of patients such as the one just quoted and the results obtained by Drs. Matthew and Proudfoot, because obviously there are many variable factors. These include the duration of exposure, the interval between exposure and the commencement of treatment, the age of the patient, and the level of carboxyhaemoglobin. Whether hyperbaric oxygen chambers will ever become a practical method of treatment available in all general hospitals is of course unknown, and until several large series of patients treated in this way with controls treated along conventional lines are available for assessment the indications and value of this form of treatment must remain a matter for conjecture.—I am, etc.,

JOHN PEARCE.

Department of Neurology,
Hull Royal Infirmary.

REFERENCES

- ¹ Garland, H., and Pearce, J., *Quart. J. Med.*, 1967, 36, 445.
² Norman, J. N., and Smith, G., *Postgrad. med. J.*, 1966, 42, 449.
³ Richardson, J. C., Chambers, R. A., and Heywood, P. M., *Arch. Neurol. (Chic.)*, 1959, 1, 178.

Pink Disease

SIR,—Following proof of the connexion between the administration of mercury to infants and pink disease, mercury was omitted from Steedman's teething powders late in 1953, and efforts were made at the time to withdraw powders containing mercury from the shops. We would like to draw attention

to two instances of pink disease due to the ingestion of mercury from other sources which have occurred in Dundee—one in May 1960 and another in August 1967.

The child presenting with pink disease in May 1960 was a girl aged 3 years and 10 months who was older than the usual infant with pink disease because the source of mercury in her case was that contained in two proprietary preparations of chocolate worm cake. The first of these is still manufactured and therefore available. It contains 60 mg. of mercurous chloride per cake. The second is no longer manufactured. According to her mother's history she was given a total dose of 180 mg. of mercury. The symptoms and signs were typical of pink disease, including loss of weight, hypotonia with disinclination to walk, listlessness, sweating, insomnia, photophobia, pinkness and peeling of the hands and feet, and hypertension. The excretion of mercury was estimated on four occasions with consecutive values of 91 $\mu\text{g.}/100\text{ ml.}$, 45 $\mu\text{g.}/100\text{ ml.}$, 103 $\mu\text{g.}/100\text{ ml.}$, and 66 $\mu\text{g.}/100\text{ ml.}$ In retrospect it might have been wise to repeat the mercury estimations on this child until a normal level was obtained. The noradrenaline excretion of 1.2 $\mu\text{g.}/24\text{ hr.}$ and the adrenaline excretion of 5.2 $\mu\text{g.}/24\text{ hr.}$ were considered within normal limits.

The second child, a boy aged 1 year, was given a variety of teething powders, totalling 34 altogether. The parents obtained three Steedman's powders in a yellow wrapper from a small shop in Dundee, which, according to the general manager of Steedman's firm, might have contained mercury. All the other Steedman's powders were in a white wrapper and did not contain mercury. He was also given Ashton and Parsons's teething powders, which do not contain mercury. However, in addition he received 12 powders from his general practitioner containing 15 mg. of pulv. hydrarg. cum cret. in each powder, that is, a total of 180 mg. of mercury. The clinical signs and symptoms were again typical of pink disease in his case, with loss of weight, hypotonia, listlessness, sweating, insomnia, photophobia, slight pinkness of the hands and feet, and hypertension. In addition he showed excessive salivation. The excretion of mercury was 20.6 $\mu\text{g.}/100\text{ ml.}$ six weeks after the onset of symptoms and 4 $\mu\text{g.}/100\text{ ml.}$ when he had recovered. The urinary vanil mandelic acid level of 1.4 mg./24 hr. was considered normal. It was of interest that the boy's cousin suffered from pink disease 10 years ago.

Pink disease is generally considered to be due to a hypersensitivity to hitherto accepted doses of mercury shown by certain individuals, and has to be distinguished from mercury-poisoning from excessive dosage. Our purpose is to draw attention once again to the danger of ingestion of mercury by infants and young children, a danger which may not be appreciated by every practitioner, as pink disease has become rare in recent years.—We are, etc.,

CONSTANCE C. FORSYTH.

D. C. L. SAVAGE.

Department of Child Health,
University of Dundee.

Pain in the Neck and Arm

SIR,—A television broadcast (27 February, complementing article 2 March, p. 563) demonstrated certain manipulations of the neck for the relief of pain due to pressure on cervical nerve roots. No reference seemed to be made to vertebro-basilar arteriosclerosis, a concomitant by no means unusual in cervical osteoarthritis.

Surely a patient with this condition in any