- ² Lilienfeld, L. S., Freis, E. D., Partenope, E. A., and Morowitz, H. J., *Journal of Clinical Investigation*, 1955, 34, 1.
³ Chinard, F. P., *Advances in Respiratory Physiology*, ed. C. G. Caro, ch. 4. London, Edward Arnold, 1966.

Gastric Ulcer after Highly Selective Vagotomy

SIR,—Because of recurrent episodes of epigastric pain a 34-year-old building labourer had lost seven jobs in seven years and had worked for only two weeks in the seven months before undergoing highly selective vagotomy on 5 July 1971. Stippling of the anterior wall of the first part of the duodenum together with minimal thickening of the lesser curve side of the pylorus were the only abnormalities noted. At this operation 5.5 cm measured on the lesser curve side of the antrum were left innervated and the oesophagus was thoroughly bared for 6 cm. When his chest was cleared he did well, so that within six weeks he was pain-free and eating larger meals than for years. He did, however, admit to foul eructations.

One year later he was unable to work for six weeks because of recurrence of pain similar to that for which his operation had been performed. He complained of poor appetite, epigastric fullness, and, again, flatulence. He had lost 5 kg in weight but was otherwise well. Acid tests were performed in the professorial surgical unit at the General Infirmary at Leeds and showed: resting juice 110 ml; spontaneous acid output nil; peak acid output (intravenous insulin) 0.90 mEq/hr free acid; maximum acid output (pentagastrin) 7.77 mEq/hr total acid; and insulin test late positive. Dr. R. F. Ashwin (who had carried out barium-meal studies on this patient in 1966 and 1971) reported that there was a small lesser-curve gastric ulcer (see Fig) where the stomach had been normal before, whereas the pyloric channel and duodenal cap now filled normally.

Fortunately the patient has been able to return to work as his pain has gone. Although his flatulence has abated he still cannot eat more than half-sized meals and he has not yet regained his lost weight.

Highly selective vagotomy^{1,2} is becoming increasingly popular as the operation of choice in cases of uncomplicated duodenal ulcer. There are cogent reasons for its popularity which do not need to be enumerated here. Latterly the operation has been extended to cases of benign gastric ulcer, apparently with equal success^{3,4}. This case has been reported because it is believed to be the first case of benign lesser-curve gastric ulcer caused by highly selective vagotomy. No similar case has arisen in more than 300 highly selective vagotomies for duodenal ulcer in Copenhagen and Leeds⁵. It could easily be argued that this patient inadvertently underwent total gastric vagotomy without drainage—a situation more akin to truncal vagotomy alone and liable to give rise to secondary gastric ulceration from stasis. Perhaps a 5.5 cm lesser-curve measurement for remaining antral innervation was too mean in this individual—certainly no other method was used to distinguish between antrum and fundus.

Highly selective vagotomy alone promises to be better than truncal vagotomy with drainage, but it is slightly more difficult. Thus cases in which the vagotomy falls short of its aims may be expected. These should be recognized rather than condemning the aim of preserving an undrained innervated antrum.

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 2 Johnston, D., and Wilkinson, A. R., *British Journal of Surgery*, 1970, 57, 289.
 3 Johnston, D., Humphrey, C. S., Smith, R. B., and Wilkinson, A. R., *British Journal of Surgery*, 1972, 59, 787.
 4 Burge, H., *British Medical Journal*, 1972, 4, 360.
 5 Johnston, D., personal communication 1972.

AHF-Related Protein and Precipitation Reactions

SIR,—Caution must be exercised before interpretations of the mode of inheritance of von Willebrand's disease are made from the studies reported by Dr. L. Holmberg and Professor I. M. Nilsson¹ (5 August, p. 317), as the authors do not report on the steps they undertook to ensure that their antiserum was truly specific. The antibody was prepared by injection of AHF purified by Sepharose 6B chromatography into rabbits. It is well known that during the purification of AHF lipoproteins and IgM cannot be excluded from the purified AHF.² To ensure that the antiserum is specific to "AHF-related antigen" the antiserum should be absorbed with a plasma known to contain minimal amounts of AHF-related protein but which is otherwise normal.

We have produced antiserum to AHF by a method similar to that used by the authors. The serum had a high specific activity against AHF in a coagulation system and produced a single precipitin line against purified AHF and cryoprecipitate. It has been used by us to detect AHF-related protein by means of a coagulation system. When this antiserum was absorbed with the plasma of a patient with von Willebrand's disease with low AHF activity (1%) and low AHF-related protein anti-AHF activity persisted but the precipitation lines were lost. We concluded that the precipitation reaction was due to antibodies to proteins contaminating the purified AHF.

Using this antiserum and a coagulation system we have found AHF-related protein in 20 children with haemophilia and in normal adults. In eight patients with von Willebrand's disease we found that AHF activity and AHF-related protein were reduced. There was a strong correlation between AHF activity and AHF-related protein. We have not found any patients with von Willebrand's disease who have low activity and normal AHF protein.

It would be of great interest to know whether the authors have used a coagulation system to test the levels of AHF-related protein in their patients and whether they were able to confirm the results of precipitation methods.—We are, etc.,

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- ¹ Hershgold, E. J., Davison, A. M., and Janszen, M. E., *Journal of Laboratory and Clinical Medicine*, 1971, 77, 185.

Trapped Nerves

SIR,—In support of Mr. J. D. Sidey and Dr. M. D. Mehta (9 September, p. 647) I offer the experience gathered in the treatment of well over 1,000 non-impact cranio-cervical injuries with nerve entrapment symptomatology. Most of these cases were due to rear-end auto collisions and had treatment for six weeks to six months, with resolution in 90% by 12 weeks if treatment was actively pursued. I found recognition of these painful lesions to be very poor in the medical community in general, and 20% of the cases were referred as a "last resort" when chronicity was established. Fortunately, conservative treatment is usually effective even in cases of several months' duration. The other cases were acute.

Three main distributions of pain were defined: (1) Pain in the occipital region of the neck radiating to the vertex, temples, and behind the eyes. (2) Pain arising at the level of the transverse process of the fifth and/or sixth cervical vertebrae and radiating up the neck to the occiput and/or down to the point of the shoulder and/or along the medial border of the scapula. (3) Low back pain (with muscle spasm) radiating along the fifth lumbar and first sacral distribution. Neurological signs were present in 75% of cases, involving the triceps, ankle, and knee jerks (as depression of activity) in that order. Some of the other symptoms noted were changes in visual acuity (90% in patients over 45) and alterations in libido of varying severity, from impotence in males to serious aversion in the female not related to the degree of pain involved. Horner's syndrome was seen in 1% of cases and emotional disturbances related to frontal and temporal lobe damage (aggressiveness and hostility mostly) in 2%. Mydriasis was seen twice.

Diagnosis was established by injection of 2% lignocaine, which usually gave immediate relief of pain. This response was so dramatic that the 10 patients not responding were classed as having non-organic disease.

Treatment was by: (1) Injection of 2% or 1% alcohol in 3-5 ml 2% lignocaine with 2 ml of Depo-Medrone (containing 80 mg methylprednisolone acetate) in cases of six

weeks' duration or more or where fibrotic reactions appeared to be marked. (2) Diazepam 5 mg three times daily. (3) Anti-inflammatory drugs (phenylbutazone). The effect of the diagnostic injection of lignocaine lasted a few hours to a few days and was rarely permanent. Early return of pain was a poor prognostic sign. Injections of lignocaine and 1% alcohol once a week with medication cleared 60% of the symptoms in six weeks and 90% in 12 weeks. Headaches were often devastating initially but resolved more rapidly than the neck and back symptoms.

In 26 patients serious bone and brain injuries were identified; two of these died and six were operated on. Twenty patients showed objective signs of lumbar intervertebral disc rupture with nerve root compression, but only three were operated on, as their symptoms resolved over a period with conservative management by injection.—I am, etc.,

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Mixed Connective Tissue Disease

SIR,—Your leading article (11 November, p. 315) shows that the delineation of "mixed connective tissue disease"¹ as a distinct entity appears at present to be rather more than an exercise in nosology. It remains to be seen, however, whether or not further experience will blur the edges of the currently fairly clear-cut situation.

Among the classical connective tissue diseases, progressive systemic sclerosis showing features of systemic lupus erythematosus (S.L.E.)² can provide diagnostic problems. In 1965 we³ drew attention to the occurrence in S.L.E. of small stiff lungs giving rise to severe dyspnoea. It is now apparent that "lupus lung" is not specific for S.L.E. but can occur in systemic sclerosis, as the following case report shows.

A 55-year-old Italian housewife presented in October 1968 with a two-month history of bilateral Raynaud's phenomenon and severe ischaemia of a finger-tip. She was known to have a hiatus hernia and had taken amphetamines (Durophet) for many years as an anorectic. In December 1968 she was admitted to another hospital with pleurisy, fever, and consolidation of the right lower lobe of the lung, which cleared after 10 days. In January 1969 she complained of severe breathlessness on slight exertion. Her left diaphragm was now elevated and later a linear shadow appeared at the left base. Her respiratory function was impaired: FEV₁ 1.0 l., FVC 1.5 l. A lung scan was suggestive of multiple pulmonary emboli.

In May 1969 she was severely dyspnoeic at rest and unable to climb stairs. There was pain in the lumbar and shoulder regions, especially on deep breathing, together with severe Raynaud's phenomenon. She later brought up undigested food and gave a history of dysphagia for some months. On examination her fingers looked sclerodermatous. Heart and lungs were clinically normal, blood pressure 130/80 mm Hg. There was no evidence of pulmonary hypertension. Investigations: Hb 14.9 g/100 ml, W.B.C. 6,800 mm³, differential normal, E.S.R. 10–20 mm in 1 hr. Tests for antinuclear factor, L.E. cells, and rheumatoid factor (latex) negative. Serum flocculation tests strongly positive. Chest x-ray showed bilateral high diaphragms with linear basal shadows. E.C.G., right heart catheterization, and pulmonary angiogram normal. FEV₁ 0.5 l., FVC 0.9 l. Skin biopsy not diagnostic. Barium swallow suggestive of systemic

sclerosis. Blood urea and electrolytes and urine analysis normal.

She was treated initially with warfarin and later with prednisone, on which she felt less breathless. Her respiratory function tests improved a little. She had several episodes of nocturnal pulmonary aspiration, in one of which she had a cardiac arrest and died in August 1969.

Postmortem showed the classical histological changes of systemic sclerosis in the skin and oesophagus. The lungs showed changes of chronic bronchitis but no evidence of pulmonary fibrosis, embolism, infarction, or arteritis. The heart, pericardium, and pleural spaces were normal.

This patient had a fulminating disease, dying of progressive systemic sclerosis less than 12 months after her initial symptoms. The chest picture, with severe breathlessness, gross restrictive impairment of ventilation, a radiological picture resembling pulmonary infarction, and the absence of specific lung pathology was identical to that found in S.L.E.³ There are thus at least two causes for dyspnoea in progressive systemic sclerosis in addition to the well-recognized pulmonary fibrosis. One is pulmonary arteritis,⁴ the other "lupus lung."—I am, etc.,

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- 1 Sharp, G. C., Irvin, W. S., Tan, E. M., Gould, R. G., and Holman, H. R., *American Journal of Medicine*, 1972, 52, 148.
- 2 Dubois, E. L., Chandor, S., Friou, G. J., and Bischoff, M., *Medicine*, 1971, 50, 199.
- 3 Hoffbrand B. I., and Beck, E. R., *British Medical Journal*, 1965, 1, 1273.
- 4 Case Records of the Massachusetts General Hospital, *New England Journal of Medicine*, 1972, 286, 91.

Poisoning Treatment Centres

SIR,—We were interested to read the article by Dr. Peter Kennedy, (4 November, p. 255) comparing the prognosis in paracetamides admitted to a poisoning treatment centre with that in those treated by a psychiatrist as an outpatient or by a general practitioner.

In a recent paper¹ we described the medical aspects of acute poisoning as seen in a general medical unit and showed that the results compared favourably with those obtained in more sophisticated centres. The social and environmental aspects of acute poisoning in the same area have also been described.² Of 131 patients admitted to our unit during 1970 after attempted suicide, only 11 (8.4%) had a further episode during 1970–1. Our recurrence rate is thus lower than Dr. Kennedy's figure of 13% despite the fact that we have included all our patients admitted in 1970 (not merely those admitted for the first time), and we have included recurrences in these patients until the end of 1971 rather than 1970. This low recurrence rate in a general medical unit without immediate psychiatric cover suggests that Dr. Kennedy is justified in being cautious about his conclusion that special units like the one in Edinburgh should be set up in other regions.

Dr. Kennedy compared only the results of management in a regional poisoning treatment centre with outpatient psychiatric care and general practitioner care. It would have been more reasonable to compare treatment in general hospitals (where most patients with self-poisoning are still treated) with treatment at home or in a sophisticated centre. It is possible that admission to hos-

pital, by temporarily withdrawing the patient from a hostile environment, may be therapeutic quite apart from psychiatric intervention. In contrast to Dr. Kennedy, we concluded¹ that it was "possible to treat successfully the vast majority of acutely poisoned patients in any general medical unit." This statement was concerned primarily with medical treatment, but we feel it is also applicable to psychiatric intervention.

We would continue to support the concept of a limited number of Regional Poisoning Treatment Centres as areas where sophisticated toxicological and epidemiological studies can be performed, but there seems little justification for the full implementation of the Hill Report.³—We are, etc.,

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- 1 Lawson, A. A. H., and Mitchell, I., *British Medical Journal*, 1972, 4, 153.
- 2 Mitchell, I., and Lawson, A. A. H., *Community Medicine*, in press.
- 3 Department of Health and Social Security, Central and Scottish Health Services Councils, *Hospital Treatment of Acute Poisoning*, London, H.M.S.O., 1968.

Herpes Simplex Encephalitis

SIR,—In the interesting and important paper by Dr. A. D. Dayan and others (18 November, p. 405) there is a reference to our work¹ which is flattering but inaccurate. In our series of electroencephalographic findings in herpes simplex encephalitis (which is the largest series in the literature) we found that the electroencephalogram reflects the severity of the illness and the site of maximum damage rather than the pathology. We also pointed out that similar changes may be seen in cerebrovascular disease, raised intracranial pressure, and other conditions. The electroencephalogram in herpes simplex encephalitis is only "diagnostic" in the sense that it may help to make the diagnosis, but we would like to stress that the electroencephalographic findings are in no way pathognomonic.—We are, etc.,

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- 1 Illis, L. S., and Taylor, F. M., *Lancet*, 1972, 1, 718.

Causes of Failure in Antibiotic Treatment

SIR,—Professor L. P. Garrod (25 November, p. 473) points out that Mr. D. S. Tindal and I¹ did not say how many wound infections in a controlled series of patients given a single parenteral dose of ampicillin were due to penicillin-resistant staphylococci.

We were concerned only with the clinical control of wound infections, and in many cases pus swabs were not taken. A few swabs grew more than one organism. Altogether, 164 patients were given ampicillin and there were 22 infections. In 12 cases no pus swab was taken and in four it was sterile. Five patients were infected with staphylococci insensitive to ampicillin and two had coliform organisms, also insensitive to ampicillin. In the control series of 185 patients not given ampicillin, 25 developed a wound infection. In 12 cases no