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Hepatitis Hazard in Clinical Laboratories

SIR,—Further to your leading article on hepatitis hazards in regular haemodialysis (2 December, p. 501) it may be useful to report some preliminary findings from the survey of laboratory staff being carried out under the auspices of the Association of Clinical Pathologists. During the period 1964-70 biochemical technicians had the highest number of cases of hepatitis and the highest attack rate, contrasting sharply with 1971, when no case was reported in this group—conceivably because of their increased awareness and improved technique and facilities. The 31 cases among staff of all types reported in 1970 and 1971 were scattered through the country and showed an even sex distribution, with 58% below the age of 30 years. Encouragingly, none was fatal and only eight required hospital ad-

mission—five of 11 Australia-antigen-positive, one of 10 Australia-antigen-negative, and two of nine untested cases.

A more detailed summary was recently presented at the Royal Society of Medicine, and in due course a more complete report of this continuing survey will be published. Only with the co-operation of numerous members of the A.C.P. has this study been made possible, and I am also grateful for the assistance given by Mrs. M. E. Martin of the Medical Records Department of Glasgow Northern Hospitals and by my secretary, Miss E. H. Simpson.—I am, etc.,

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Changes in Lung Capillary Permeability in Renal Failure

SIR,—Dr. W. A. Crosbie and others (18 November, p. 388) have shown that there is increased pulmonary capillary permeability to sodium in the pulmonary oedema of renal failure. In their discussion, and in the title of their paper, they imply that this change in permeability is in some way a particular feature of uraemia, albeit a feature that is seen only when the uraemic lung becomes oedematous. In doing so they ignore the work of Bauman *et al.*,¹ who showed an increase in lung capillary sodium permeability in the pulmonary oedema of heart failure, and of Lilienfield *et al.*,² who demonstrated an increase in pulmonary capillary thiocyanate permeability in "pulmonary congestion" of cardiac origin. It would seem from these studies that pulmonary capillary permeability increases in pulmonary oedema

associated with conditions other than renal failure. It is very surprising that the authors have not studied patients in pulmonary oedema with normal renal function before attributing permeability changes to uraemia.

Chinard³ has discussed the pulmonary capillary barrier to diffusion of small molecules (such as urea or thiocyanate) and ions. He presents evidence that the permeability of the barrier (which he believes to be the basement membrane and interstitial space) depends upon its hydration. In pulmonary oedema the gel matrix of the basement membrane and interstitium would imbibe water, swell, and become more permeable. Although this would mainly affect the passage of small particles, an increased protein permeability might be expected if the change of hydration was extreme.

If raised pulmonary capillary sodium permeability were a feature of uraemia *per se*, then one would expect that the lung capillaries of some non-oedematous dialysis patients would show this excessive permeability. However, non-oedematous patients with normal permeability were not found in Dr. Crosbie's study. One patient in whom three separate estimations were made showed a fall of "permeability fraction" as his oedema cleared. If the increased sodium permeability was a consequence of renal failure, then one would not expect to see the permeability return to normal in this way.

In their discussion Dr. Crosbie and his colleagues state that the capillary pressure in the lung and the plasma protein osmotic pressure may be normal in pulmonary oedema of renal failure. They opine that "a change in the permeability of the capillary wall would provide an alternative explanation" for the oedema. The importance of the permeability changes must not be exaggerated, particularly when one remembers that the permeability change could not be demonstrated in the non-oedematous state. Moreover, an increased permeability to ions and small molecules cannot of itself cause oedema in the face of normal capillary hydrostatic and osmotic pressures. Increased capillary permeability is important in oedema formation when it allows plasma proteins to cross the capillary wall. This study has shown no increase in protein permeability and therefore has added little to our knowledge of the aetiology of the pulmonary oedema of renal failure.—I am, etc.,

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¹ Bauman, A., Rothschild, M. A., Yalow, R. S., and Berson, S. A., *Journal of Applied Physiology*, 1957, 11, 353-361.

- 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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- 1 Amdrup, E., and Jensen, H. E., *Gastroenterology*, 1970, 59, 522.
 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