Hypernephroma Presenting with Hypertension

SIR,—The report by Mr. M. D. Ram and Mr. D. G. Chisholm (11 October, p. 87) of a case of hypernephroma with the "very rare" presenting feature of hypertension prompts me to put on record a similar case. This patient was observed over a period of eight years with steadily rising blood pressure, finally revealed to be a consequence of a hypernephroma. The following is a brief report.

The patient was in the Army Medical Corps with the rank of matron. Her blood pressure in May 1948 (when she was 42 years of age) was discovered to be 150/110 mm. Hg. It was on record that it had been normal six months earlier. On each occasion the examination was purely routine and not related to any symptoms. As she was an important person in the Service, she was seen by a number of notable doctors during the ensuing eight years, while her blood pressure rose steadily each year, reaching 220/140 mm. Hg in October 1955. Treatment with Serpasil (reserpine) and other such drugs was begun at this time, though symptoms from the high blood pressure were not conspicuous. There were no urinary symptoms at any time and no abnormal findings on examination of the urine. An intravenous pyelogram in August 1952 was declared to be normal, and again in December 1955, even though some enlargement of the right kidney was recognized and the patient actually complained on one occasion of a feeling of "fullness in the right loin." A further intravenous pyelogram in October 1956 (and a subsequent right retrograde pyelogram) did indeed reveal changes, which even then were regarded as "suggestive of old pyelonephritis with possible cystic disease in the kidney," though it was declared that "malignancy cannot be entirely excluded on the films."

In any case, right nephrectomy was performed in November 1956, revealing a sizeable hypernephroma of the lower pole. The blood pressure fluctuated violently during recovery and convalescence, but eventually settled to between 150/90 mm. Hg and 170/110 mm. Hg (various readings).

She returned to service in the Army Medical Corps for the next four and a half years. Rather naively, she believed that the nephrectomy had been done for high blood pressure (which indeed was true, though not the whole truth), fortunately remaining blissfully ignorant of the true nature of her complaint. Metastases in the bones and thorax finally proved fatal in October 1961, only a month short of the five-year survival.

In retrospect it was considered almost certain that this lady had had a kidney tumour as far back as 1948, and that its only manifestation during the following eight years was hypertension. It seems worth while recording this case, alongside that of Mr. Ram and Mr. Chisholm, to alert physicians treating hypertension to such a possibility.—I am, etc.,

ROBERT S. LAWSON.

Melbourne, Victoria,
Australia.

Trigeminal Sensory Neuropathy

SIR,—Your very interesting leading article (10 January, p. 64) on the above subject, with brief reference to early neuroma of the Gasserian ganglion, brings back to my memory the year I spent at the Mayo Clinic in 1929, working in the neurosurgical department there under the directorship of Dr. A. W. Adson and, incidentally, having as one of its consultant staff at that time J. R. Learmonth, later to become the eminent Sir James Learmonth of Edinburgh.

During that year three cases of this type were dealt with by the department, and I was allowed to go through the records of all previous ones dealt with at the Clinic. This brought the total up to nine, of which seven were of tumour origin involving the ganglion and two were due to chronic inflammation. After studying the literature of the previous 25 years, which covered about 60 further cases, I produced a paper entitled "A Study of Tumours and Inflammation of the Gasserian Ganglion."¹ Though this was 40 years ago, it may still be of interest to those dealing with this problem.—I am, etc.,

F. A. R. STAMMERS.

Birmingham 29.

REFERENCE

- 1 Stammers, F. A. R. *British Journal of Surgery*, 1930, 18, 125.

The Influenza Epidemic

SIR,—Observers are noting that the 1969/70 epidemic of A2(1968 Hong Kong) strain of influenza is unusual in a number of ways and does not compare exactly with previous experience. This is not altogether surprising, since the virus has changed its characteristics slightly, and the community which it is infecting has changed its social habits dramatically.

It is important that as much research as possible should take place into the behaviour of this virus. Most of the research may have to be retrospective, but it is possible that a third wave of infection may yet occur in 1970. It seems also important that researchers should follow the example of Edward Jenner (1749-1823) and study carefully those individuals and communities who, though exposed to the virus, have not been ill.

I agree with Dr. J. Fry (17 January, p. 171) in his hope that the Department of Health and Social Security will amass as much information as possible about the epidemic and publish it.—I am, etc.,

MICHAEL A. WELLER.

Thaxted,
Essex.

Vagotomy for Peptic Ulceration

SIR,—When the anterior and posterior vagus nerves are stimulated beneath the diaphragm in man there is a rise of intra-gastric pressure of some 12-14 cm. of water. We now know that the vagal trunks on the lower oesophagus are multiple. Some of these small nerves are almost hair-like in size. We now know too that these small

nerves do matter in vagotomy. In some patients a grossly incomplete vagotomy never gives rise to recurrent ulceration; in others a very small nerve left undivided at operation may be responsible for severe recurrent disease. If, using the electrical stimulation test, we regard a rise of gastric pressure of as little as 0.2-0.4 mm. water as indicative of incomplete vagotomy (as I think we should), then probably at least 90% of all vagotomies done without the test are incomplete.

Because of the high recurrent ulcer rate following vagotomy and simple drainage, many surgeons may before long reject vagotomy and turn once more to gastric resection. Some I think have already done this, while others are seriously considering the same step. Yet in all patients with recurrent duodenal ulceration after vagotomy nerve section is incomplete.

The following case is therefore of some importance. After twelve attempts to make selective vagal section complete at the stomach, the electrical stimulation test showed incomplete nerve section by a rise of gastric pressure of 5 mm. of water (Fig. 1).

FIG. 1.—The electrical stimulation test. A rise of pressure of approximately 5 mm. of water on stimulation, after 12 unsuccessful attempts to achieve complete section. (One large square = 3 mm. of water).

It was then decided to make no more attempts to find this small nerve for fear of damage to the oesophageal wall. Atropine was therefore given, after which the stimulation test became negative (Fig. 2) con-

FIG. 2.—The response abolished by atropine.

firming that the tracing was indeed indicative of incomplete vagotomy. Ten days later the insulin test was positive. This is just one of a number of cases in which the importance of a very small rise of gastric pressure, using the electrical stimulation test, has been proved.

In a series of cases the electrical stimulation test has been applied after the leucomethylene blue dye test has been used. Details of this study will be published later. It seems that leucomethylene blue used as described by Lee¹ does not stain even the smallest nerves.—I am, etc.,

HAROLD BURGE.

West London Hospital,
London W.6.

REFERENCE

- 1 Lee, M., *British Journal of Surgery*, 1969, 56, 10.