Arriagada, C. R., Hernandez, M. A. A., and Lagos, N. (1962). *Neurocirugia*, 20, 95.
Banker, B. Q. (1961). *Journal of Neuropathology and Experimental Neurology*, 20, 127.
Beesley, R. A., and Daniel, P. M. (1956). *Journal of Clinical Pathology*, 9, 267.
Berlin, L., Tumarkin, B., and Martin, H. L. (1955). *New England Journal of Medicine*, 252, 162.
Bickerstaff, E. R., and Holmes, J. M. (1967). *British Medical Journal*, 1, 726.
Biemond, A. (1951). *Brain*, 74, 300.
Bradshaw, P., and McQuaid, P. (1963). *Quarterly Journal of Medicine*, 32, 279.
Cravioto, H., Rey-Bellet, J., Prose, P. H., and Feigin, I. (1958). *Neurology (Minneapolis)*, 8, 145.
Cross, J. N., Castro, P. O., and Jennett, W. B. (1968). *British Medical Journal*, 3, 214.
Dalal, P. M., Shah, P. M., Anar, R. R., and Kikani, B. J. (1968). *British Medical Journal*, 2, 769.
Davie, J. C., and Coxe, W. (1967). *Archives of Neurology*, 17, 313.
Denny-Brown, D. (1953). *Bulletin of the New England Medical Center*, 15, 53.
Dooley, J. M., and Smith, K. R. (1968). *Neurology (Minneapolis)*, 18, 1034.
Duffy, P. E., and Jacobs, G. B. (1958). *Neurology (Minneapolis)*, 8, 862.
Fang, H. C. H., and Palmer, J. J. (1956). *Neurology (Minneapolis)*, 6, 402.
Fisher, C. M. (1970). *Archives of Neurology*, 22, 13.

Fisher, C. M., Karnes, W. E., and Kubik, C. S. (1961). *Journal of Neuropathology and Experimental Neurology*, 20, 323.
Kubik, C. S., and Adams, R. D. (1946). *Brain*, 69, 73.
Louis, S., and McDowell, F. (1967). *Annals of Internal Medicine*, 66, 932.
Luhan, J. A., and Kaplan, L. A. (1951). *Archives of Neurology and Psychiatry*, 66, 389.
Meyer, J. S., Sheehan, S., and Bauer, R. B. (1960). *Archives of Neurology*, 2, 27.
Nagashima, C. (1970). *Journal of Neurosurgery*, 32, 512.
Prensky, A. L., and Davis, D. O. (1970). *Neurology (Minneapolis)*, 20, 945.
Salmon, M. L., Winkelman, J. Z., and Gay, A. J. (1968). *Journal of the American Medical Association*, 206, 85.
Schoenberg, B. S., Whisnant, J. P., Taylor, W. F., and Kempers, R. D. (1970). *Neurology (Minneapolis)*, 20, 181.
Silversides, J. L. (1954). *Proceedings of the Royal Society of Medicine*, 47, 290.
Sprofkin, B. E., and Blakey, H. H. (1956). *Archives of Internal Medicine*, 98, 617.
Stewart-Wallace, A. M. (1964). *British Medical Journal*, 2, 1528.
Trotsenburg, L. Van, and Vinken, P. J. (1966). *Journal of Neurology, Neurosurgery and Psychiatry*, 29, 241.
Wells, C. E., and Timberger, R. J. (1961). *Archives of Neurology*, 4, 268.
Williams, D., and Wilson, T. G. (1962). *Brain*, 85, 741.
Wood, M. W., and Murphey, F. (1969). *Journal of Neurosurgery*, 30, 260.

PRELIMINARY COMMUNICATIONS

Selective Proximal Vagotomy with and without Pyloroplasty

C. WASTELL,   J. F. COLIN,
J. I. MacNAUGHTON,   J. GLEESON

Summary

The early results of a prospective randomized clinical trial of selective proximal vagotomy with and without gastric drainage in a total of 36 patients are reported. In spite of adequate average acid reduction and little evidence of gastric retention two definite recurrent ulcers have occurred in the 16 patients who did not have a pyloroplasty. Further trials of the effect of selective proximal vagotomy without gastric drainage are necessary before the procedure is widely adopted.

Introduction

Truncal vagotomy was introduced on a firm scientific basis by Dragstedt and Owens (1943). Since then it has been constantly criticized because of the severe postoperative symptoms developed by a few patients. This has led to a series of refinements to the original operation, the latest of which is that of selective proximal vagotomy. Before this can be accepted as a procedure suitable for patients with a chronic duodenal ulcer, two questions require an answer: (1) is selective proximal vagotomy effective in curing duodenal ulcer? and (2) is a gastric drainage operation necessary?

Method

To answer these questions we prepared a consecutive series of 36 males suffering from uncomplicated chronic duodenal ulcer, prospectively randomized into two treatment groups. In

Westminster Medical School, Gordon Hospital, London S.W.1
C. WASTELL, M.S., F.R.C.S., Honorary Consultant and Senior Lecturer in Surgery
J. F. COLIN, F.R.C.S.ED., Surgical Registrar
J. I. MacNAUGHTON, B.SC., Biochemist
J. GLEESON, M.R.C.P., F.F.R., Consultant Radiologist

the first group of 16 patients selective proximal vagotomy alone was performed and in the second group of 20 patients selective proximal vagotomy plus a modified Finney pyloroplasty was the operation. All operations were carried out by the same person. The first patient was accepted into the series in August 1970 and the 36th in April 1971, providing a follow-up of between 3 and 12 months.

Before operation the peak acid output under pentagastrin stimulation was measured, and after operation, on approximately Days 5, 9, and 10, a barium swallow and meal, an insulin response, and a repeat peak acid output were measured. From the gastric function tests for acid secretion, the volume of fasting juice was also obtained. After operation this was taken as the mean of the values obtained when measuring the peak acid output and when measuring insulin response. The insulin responses were analysed by using Hollander's original criteria with the addition of a second hour's collection so that the result was negative, early positive (within 60 minutes after insulin), or late positive (60-120 minutes after insulin).

Operation

The technique used for selective proximal vagotomy is described briefly. Immediately after the induction of anaesthesia, which was carried out without atropine premedication, the patient was given 0·6 microgramme of pentagastrin per kilogramme body weight, intramuscularly.

After laparotomy the antrum was defined by using a pH probe passed orally into the stomach, enabling the pH of the surface of the mucosa to be sampled by manipulation of the probe through the anterior wall of the stomach. This procedure was facilitated by inflating the stomach slightly and so separating the anterior from the posterior wall. Generally, the boundary point between the antrum and the body, which was clearly definable in only half the cases, lay 5 to 7 cm from the pylorus. If the boundary could not be clearly defined it was taken as the point just proximal to the last clearly seen branch of the anterior nerve of the lesser curve (the nerve of Latarjet).

The vagotomy is performed by dividing the entire neurovascular inflow to the lesser curve from the boundary point, working proximally close to the stomach wall, to a position just short of the oesophagogastric junction. The dissection is then carried across the front of the stomach to its junction with the oesophagus on the left. Finally, the back of the oesophagus and stomach are cleared. The end result of this dissection is to denude the lesser curve of the stomach (see

Fig.) while preserving the anterior and posterior nerves of Latarjet, which supply the antrum, and the hepatic and coeliac nerves from the anterior and posterior main vagi.

When performed, the pyloroplasty was a modification of the Finney pyloroplasty resulting in a wide gastric-duodenal stoma.
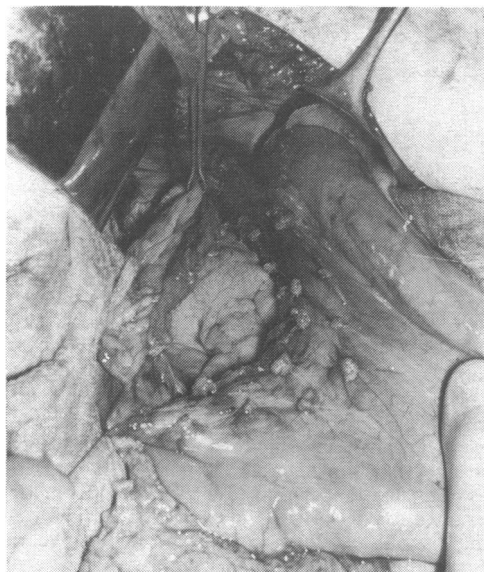
Lesser curve of stomach after the neurovascular inflow to the parietal-cell-bearing area has been removed.

Results

Insulin Tests.—Insulin stimulation in 16 patients who had undergone selective proximal vagotomy produced 11 negative, 3 early positive, and 2 late positive responses. After selective proximal vagotomy plus Finney pyloroplasty on 20 patients, 2 early positive, 3 late positive, and 14 negative responses were elicited. In one patient of the latter group the test was a technical failure.

Peak Acid Output. Pentagastrin.—In patients with a negative insulin response selective proximal vagotomy reduced the peak acid output by 58% from a mean of 35·4 to 14·5 mEq/hr and selective proximal vagotomy plus Finney pyloroplasty by 56% from 38·1 to 16·9 mEq/hr (Table I). It cannot be shown that the reduction in peak acid output by selective proximal vagotomy alone is significantly different from that due to selective proximal vagotomy plus Finney pyloroplasty.

Fasting Juice.—This was increased by selective proximal vagotomy from a mean of 51 to 139 ml and by selective

proximal vagotomy plus Finney pyloroplasty from 74 to 123 ml (Table II). These increases could not be shown to be significantly different from each other. In each group, after operation, two patients had mean values in excess of 250 ml.

Barium Meals.—These were assessed by inspection, and one patient after each operation was thought to have a significant degree of gastric retention.

Recurrence.—Two patients had a definite recurrence which required reoperation and both were in the selective proximal vagotomy group. At operation the recurrent duodenal ulcer was seen and the base was not covered with mucosa in either case. One other patient after selective proximal vagotomy had symptoms and a barium meal appearance strongly suggestive of recurrent ulceration.

Discussion

Selective proximal vagotomy results in the denervation of the body of the stomach—that is, the part containing parietal cells—while the antrum retains its nerve supply. It was investigated experimentally by Griffith and Harkins (1957) in the dog. Subsequently it was used, together with a form of segmental proximal gastric resection, in man by Ferguson *et al.* (1960).

Reports have now appeared in which selective proximal vagotomy has been used successfully without any form of gastric drainage for patients suffering from uncomplicated chronic duodenal ulcer (Amdrup and Jensen, 1970; Johnston and Wilkinson, 1970).

In our own cases the reduction in the peak acid output is comparable to that demonstrated by similar tests and stimuli by others (Amdrup and Jensen, 1970; Johnston and Wilkinson, 1970) following selective proximal vagotomy and total vagotomy (Multicentric Pilot Study, 1967) and greater than was obtained after selective vagotomy (Aylett *et al.*, 1969). The incidence of positive insulin responses is rather high but perhaps not more so than is obtained after total vagotomy. It is true that the gastric function tests have been carried out soon after operation and it is likely that the incidence of positive insulin responses will increase with the passage of time.

What is of exceptional interest is the fact that even in this short follow-up there have been two recurrences after selective proximal vagotomy without drainage. One of these two patients had an insulin test that was a late positive which reverted to an early positive on later retesting. His peak acid output was reduced by operation from 45·3 to 15·6 mEq/hr, a reduction of 66%. The fasting juice fell from 36 to 32 ml after operation. The second had a negative response to insulin which remained negative on retesting. His peak acid output was 70·1 mEq/hr before operation but, unfortunately, technical error spoiled his postoperative reading. The patient with a possible recurrence had a negative response to insulin and a 49% reduction in peak acid output from 34·0 to 17·9 mEq/hr after operation. His fasting juice rose from 26 to 48 ml.

It might be argued that the selective proximal vagotomy was inadequate in extent, but the operation we performed is, if anything, more radical than that performed by F. Holle (personal communication, 1971) and similar to the procedure reported by Johnston and Wilkinson (1970) and by Amdrup and Jensen (1970). Equally, it could be said that our procedure was too radical, resulting in antral stasis, but if this is so, at worst our operation results in no more than a conventional selective vagotomy which is claimed by Burge (1971) to be a satisfactory procedure when used without drainage. The volumes of fasting juice in the patients who have had a definite or possible recurrence do not indicate significant retention.

We hope to report a complete review of the patients who have been operated on in this trial. We feel, however, that the recurrence rate in the short follow-up is so high as to warrant

TABLE I—*Effect of Operation on Peak Acid Output in Group with Selective Proximal Vagotomy and Finney Pyloroplasty (S.P.V. + F.) and Selective Proximal Vagotomy without Drainage (S.P.V.).*

	S.P.V.		S.P.V. + F.	
	Before	After	Before	After
Mean (mEq/hr) ..	35·4	14·5	38·1	16·9
S.D.	15·7	10·2	14·0	8·1
No. of tests	11	11	14	12

TABLE II—*Effect of Operation on the Fasting Juice in the Group with Selective Proximal Vagotomy and Finney Pyloroplasty (S.P.V. + F.) and Selective Proximal Vagotomy without Drainage (S.P.V.).*

	S.P.V.		S.P.V. + F.	
	Before	After	Before	After
Mean (ml)	51	139	74	123
S.D.	23	147	46	95
No. of tests	11	22	13	26

an early report. The operation of selective proximal vagotomy is clearly of great interest and requires further careful assessment; it would be wise, however, for surgeons to await the results of further trials before carrying out many of these operations without gastric drainage.

We are grateful to Sister Sturrock, who has helped in the care of these patients, and to Professor H. Ellis, Mr. E. Harper, Mr. R. Fiddian, and Mr. D. H. C. Harland for referring some of the patients to us.

References

Amdrup, E., and Jensen, H. E. (1970). *Gastroenterology*, 59, 522.
Aylett, P., Wastell, C., and Wise, I. (1969). *American Journal of Digestive Diseases*, 14, 245.
Burge, H. (1971). *British Medical Journal*, 1, 172.
Dragstedt, L. R. and Owens, F. M. (1943). *Proceedings of the Society for Experimental Biology and Medicine*, 53, 152.
Ferguson, D. J., Billings, H., Swenson, D., and Hoover, G. (1960). *Surgery*, 47, 548.
Griffith, C. A., and Harkins, H. M. (1957). *Gastroenterology*, 32, 96.
Johnston, D., and Wilkinson, A. R. (1970). *British Journal of Surgery*, 57, 289.
Multicentric Pilot Study (1967). *Lancet*, 1, 291.

MEDICAL MEMORANDA

Spontaneous Spinal Subdural Haematoma

D. I. ANAGNOSTOPOULOS,   PETER GORTVAI

Spinal subdural haematoma is one of the rarer causes of acute spinal compression and is usually associated with injury or a bleeding diathesis (Stewart and Watkins, 1969).

A case is presented here in which the spinal subdural haematoma had no apparent cause. Operative removal of the haematoma alleviated the patient's serious plight.

Case Report

A woman aged 63 gave the history of violently severe sudden pain in the arms, back, and abdomen just before retiring to bed 18 days before her admission to this department. Next day she was aware of weakness and numbness of the lower limbs and the bladder had to be catheterized. After five days of nursing at home she was taken to her local hospital, where lumbar puncture yielded bloodstained fluid with xanthochromic supernatant containing 800 mg of protein and 20 mg of sugar per ml. C.S.F. pressure was 45
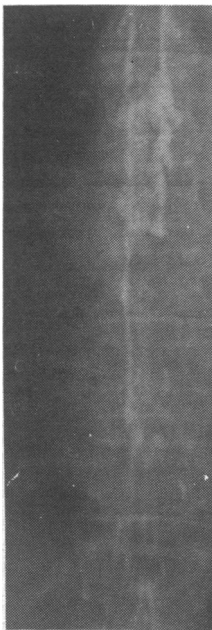
Myelogram showing partial block to the flow of contrast medium at D 8 and an irregular filling defect between D 8 and D 12.

Regional Centre for Neurology and Neurosurgery, Oldchurch Hospital, Romford, Essex

D. I. ANAGNOSTOPOULOS, M.D., Neurosurgical Registrar
PETER GORTVAI, M.CHIR., F.R.C.S., Consultant Neurosurgeon

mm of C.S.F., with no rise on jugular compression. Power and sensation in the legs improved for three days but the neurological condition of the legs deteriorated again, the abdomen was distended, and she was sent to the neurosurgical unit.

On admission there was severe flaccid paraparesis with virtually no hip or ankle movement. She was able only to wiggle her toes. The tendon reflexes were present and equal and the plantar responses extensor. There was a sensory level to pin-prick at the eighth dorsal level. Position sense and vibration sense were absent in the lower limbs. X-ray pictures of the spine showed slight arthritic changes but no other abnormality. The peripheral blood picture was normal. Lumbar puncture produced distinctly brownish fluid under a pressure of 100 mm of C.S.F., with no rise on jugular compression. Myelography showed a block to the flow of the contrast medium at D 8 and an irregular filling defect between D 8 and D 12 (see Fig.).

Laminectomy was performed on the day of admission, extending from D 8 to D 12 inclusive. The dura was tense and bluish, and when incised along the midline clotted old blood was extruded immediately. A soft rubber catheter passed freely upwards and downwards in the subdural space and no further clot was found. The spinal cord appeared normal and the subarachnoid space contained no blood within the limits of the exposure. No visible source of subdural haemorrhage could be seen.

Improvement in movement and sensation of the lower limbs began on the second postoperative day. She was discharged on the fourteenth day with voluntary control of micturition. Six months after operation she could walk with a walking frame. A year after operation her lower limbs became spastic and 17 months after operation bilateral obturator neurectomy was performed to correct spasm of the femoral adductors. At the time of writing she was able to get about with her walking frame.

Comment

Haematomas without apparent cause are uncommon. One of the cases described by Ainslie (1958) was similar to the present case. One can only surmise that the bleeding was caused by such minor trauma that it was not remembered by the patient. The remittent clinical course is reminiscent of intracranial subdural haematoma. The operative findings came as a surprise to us, but we now think that the history of sudden agonizing pain with bilateral radiation along several roots followed by paraparesis with a partial remission should suggest the clinical diagnosis of intraspinal haemorrhage. Such a diagnosis would be supported by the finding of altered blood on lumbar puncture and the myelographic finding of a block with an irregular extramedullary filling defect below it.

In spite of the long history of spinal compression and the very poor neurological condition on admission a worthwhile clinical result was obtained by laminectomy and evacuation of the haematoma.

References

Ainslie, J. P. (1958). *British Journal of Surgery*, 45, 565.
Stewart, D. H., jun., and Watkins, E. S. (1969). *Journal of Neurosurgery*, 31, 80.