

Term	Number who Chose the Term as			Total Score: (1st Choice = 3; 2nd = 2; 3rd = 1)
	1st Choice	2nd Choice	3rd Choice	
Acid fundal selective vagotomy	1	0	1	4
Highly selective vagotomy	6	6	11	41
Parietal cell vagotomy	12	15	3	69
Proximal gastric vagotomy	17	7	6	71
Proximal selective vagotomy	2	2	1	11
Proximal vagotomy	0	4	3	11
Secretory selective vagotomy	1	0	0	3
Selective proximal vagotomy	5	5	8	33
Selective vagotomy of the parietal cell mass ..	0	2	1	5
Super selective vagotomy	0	0	0	0
Supra selective vagotomy	0	0	0	0

gastric operations and acid tests. Our list was circularized, modified, and recircularized, and the final version as approved by the society has been published.¹ No generally acceptable term could be agreed for the new vagotomy, and we were asked to try again to reach a consensus to clarify this confusing situation, in which at least 11 terms and abbreviations have already been used to describe this single procedure.

We sent a questionnaire to 46 workers in this subject, 36 in the United Kingdom and 10 abroad, and received 44 replies. The 11 terms that have been used were listed and each person was asked to select three terms in order of preference; they were also asked if the majority verdict would be acceptable should this not coincide with their first choice.

It will be seen from the table of results that "proximal gastric vagotomy" scored most points, followed by "parietal cell vagotomy," and "highly selective vagotomy" came third, whether by counting first choice only or by a scoring system. Three workers indicated that they would not accept a majority verdict. Although "proximal gastric vagotomy" was one of the first three choices of 30 of the workers asked, it was the first choice in only 17 of the 44 replies.

We are aware of the lack of precision of all the terms that are used and also that the originators of particular variations in the technique all wish to retain the term they themselves coined. It is for this very reason that so many terms have been used. It seems to us, however, that the best term to use is the one that most people would like to use. In the interests of clarity we are ourselves using the term "proximal gastric vagotomy" for the new operation and we are recommending this term to other workers.—We are, etc.,

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¹ Gut, 1972, 13, 673.

Gastric Ulcer after Highly Selective Vagotomy

SIR,—Mr. Robert Hall's interesting report (30 December, p. 789) of a patient who developed a gastric ulcer after highly selective vagotomy without a drainage procedure raises questions which are pertinent to the surgical treatment both of gastric ulceration and of duodenal ulceration. Does highly selective vagotomy often produce gastric stasis? What length of prepyloric stomach

should be left innervated? When must a drainage procedure be added? Finally, does the innervated gastric antrum release excessive amounts of gastrin, which in turn might lead to gastric ulceration (in accordance with Dragstedt's classic hypothesis) or to recurrent duodenal ulceration?

Gastric emptying was found by three groups of workers¹⁻³ to be normal after highly selective vagotomy in dogs, while a fourth group⁴ found that the operation produced gastric stasis. In rabbits truncal vagotomy without drainage led to gastric ulceration in 11 of 12 animals and selective vagotomy of the entire stomach to gastric ulceration in five of 10 animals, while none of 12 rabbits subjected to highly selective vagotomy developed gastric ulceration.⁵ In man gastric emptying of fluids,^{6,7} of food and barium,^{8,9} and of a radioactive "normal" meal¹⁰ was found to be satisfactory after highly selective vagotomy. There has been little clinical evidence of slow gastric emptying in the 230 patients who have undergone highly selective vagotomy in Leeds in the past four years, and the tests of gastric emptying which were performed more than one year after highly selective vagotomy the operation yielded no evidence of stasis.^{6,9} Two patients complained of foul flatulence and four of frequent vomiting in the early months after highly selective vagotomy, but in each case these symptoms disappeared with the passage of time. Only one patient has subsequently required the addition of a drainage procedure for the relief of gastric retention. Several hundred tests of gastric secretion performed three months to four years after highly selective vagotomy provided no evidence of "hidden" gastric stasis. Volumes of resting juice in the stomach after an overnight fast were low, and food residues were not found.

Another crucial question is how much of the stomach should be left innervated. In highly selective vagotomy¹¹ the main anterior and posterior nerves of Latarjet, with their terminal branches to the antral region, are preserved. The course of these nerves is constant, and they are visible in at least 90% of patients. The average length of stomach remaining innervated is 5-7 cm, but the point is that dissection should begin proximal to where the nerves cross on to the stomach. In a few patients with large stomachs this may be as much as 8 or 9 cm from the pylorus. Professor Amdrup, who routinely "maps" the extent of the antrum at the time of operation (which he terms "parietal-cell" vagotomy) finds that the antrum-corporis junction is, on average, 7-9 cm from the pylorus. Thus we would advise anyone who is thinking of trying the operation to start with slim patients, in whom the nerves are easy to see. Also, if he is in doubt,

it is probably better to leave too much stomach innervated than too little, because there is evidence³ that highly selective vagotomy may represent an unnecessarily extensive vagal denervation of the stomach. Professor Amdrup, for example, achieves similar clinical results to our own by means of a slightly less extensive denervation of the stomach.

What proportion of all patients with duodenal ulcer coming to operation require the addition of a drainage procedure? In the first year we "drained" approximately 10%, in the second and third years 5%, and in the past year none at all, although we would certainly "drain" the stomach of any patient who had gross, decompensated pyloric stenosis with an atonic "soup-plate" stomach on barium-meal examination. In the past 18 months we have treated 10 consecutive patients with clinically manifest pyloric stenosis by highly selective vagotomy plus simple digital dilatation of the stricture, without a drainage procedure. These patients are now in good health. The finding of a very scarred, distorted, and somewhat stenosed duodenum does not imply that a drainage procedure is necessary, *provided* that before operation the patient did not have clinical pyloric stenosis.

There is no evidence of "excessive" gastrin release after highly selective vagotomy. In dogs highly selective vagotomy leads to a 47% increase in acid output from a Heidenhain pouch,² but truncal vagotomy with¹² or without¹³ drainage leads to an even greater increase in acid output. In man truncal vagotomy has been shown to produce a significantly greater increase in fasting plasma gastrin concentration than does selective vagotomy.^{14,15} Preliminary data on gastrin levels in patients after highly selective vagotomy^{16,17} suggest that they are little different from preoperative levels. We have studied acid output in response to a liquid meal of meat extract in patients more than one year after highly selective vagotomy (antrum innervated) or truncal or selective vagotomy plus pyloroplasty (antrum denervated).^{18,19} The mean acid response was found to be no greater after highly selective vagotomy than after truncal vagotomy and pyloroplasty or selective vagotomy and pyloroplasty. We also vagally denervated the antral mucosa in dogs and found that the secretory response to various test meals was undiminished.²⁰ The best evidence, however, that gastrin release is not "excessive" after highly selective vagotomy is that not a single patient of our own or of Professor Amdrup's and Dr. H. E. Jensen's (300 in all) has developed recurrent duodenal ulceration in the past four years. Recurrent ulceration after vagotomy with pyloroplasty is characteristically early,²¹ usually within two years of the initial operation. Thus it now seems unlikely that the incidence of recurrent ulceration after highly selective vagotomy will be higher than the incidence after vagotomy and pyloroplasty. As with any type of vagotomy, it is essential that highly selective vagotomy should be a complete vagotomy of the parietal cell mass, as shown by the finding of a negative insulin test in the early postoperative period.—I am, etc.,

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"Twin" Intracranial Aneurysms

SIR,—We are prompted by Mr. B. Fairburn's memorandum (27 January, p. 210) on the occurrence of aneurysmal haemorrhage in monozygotic twin sisters to report two similar cases of our own.

Case 1.—A woman aged 42 years was referred to one of us (P.J.E.W.) after proved, coma-producing subarachnoid haemorrhage on 3 September 1969. The next day, though still drowsy, inert, and photophobic, she had no focal neurological deficits. Obesity and labile hypertension were noted. An electrocardiogram showed left bundle-branch block, sinus bradycardia, and T-wave depression in leads II, III, and aVF. Carotid angiography showed a small lobular aneurysm of the left middle cerebral artery (fig. 1) and an "infundibulum" (arguably

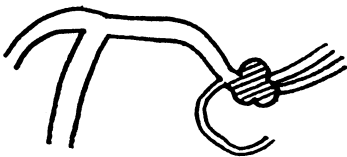


FIG. 1.—Case 1. Anteroposterior view, left carotid angiogram (tracing).

a second small aneurysm) at the origin of the right posterior communicating artery. There was no clot or vasospasm. Three days later, after direct-puncture vertebral angiography (with normal findings), she suddenly became aphasic with right hemiplegia, though retaining full consciousness. Recovery occurred during the next few days. An expectant regimen, with hypotensive medication, was followed and she has remained neurologically well.

Case 2.—This woman was referred to one of us (I.P.C.) at the age of 45 years after proved, non-coma-producing subarachnoid haemorrhage on 26 November 1972. She was the identical twin of the previous patient (but formal haematological substantiation of monozygosity was not

made). She was a known hypertensive and had had toxæmia of pregnancy. Two days after her haemorrhage she was alert and free of focal neurological signs, though both plantar responses were extensor. Carotid angiography showed a small saccular aneurysm of the left middle cerebral artery (fig. 2) and an "infundibular"

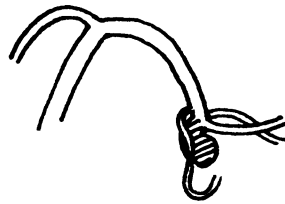


FIG. 2.—Case 2. Oblique view, left carotid angiogram (tracing).

origin of the right posterior communicating artery. Vertebral angiography and excretion pyelography were normal. Again an expectant regimen was advised. Her hypertension was somewhat resistant except to large doses of methyldopa and clonidine. On 23 December 1972 she had a second subarachnoid haemorrhage and four days later a further sudden, rapidly fatal haemorrhage. The diagnosis was confirmed at necropsy but detailed neuropathological studies are not yet complete.

The aneurysms were more closely congruous in our cases than in Mr Fairburn's and were at one of the commoner sites. Both our patients were hypertensive, as was their mother. Their father had died 15 years previously of "cerebral haemorrhage." Whether the hypertension was in any way linked with the haemorrhages, either as an immediate precipitant or as a factor in the development of the aneurysms, is conjectural. Whether, in the light of the fate of her sister, our first patient's expectant regimen should be abandoned for a more aggressive surgical approach now poses an unusual clinical dilemma.

The incidence of aneurysmal subarachnoid haemorrhage in a closed community of 400,000 was found by Pakarinen¹ to be 13 per 100,000 per annum. The incidence in the population at large cannot be computed with precision, but synthesis of the Registrar General's statistics² and published data of large series of proved cerebral aneurysms^{3,4} enables certain broad predictions to be made, assuming chance to be the sole operating factor. Thus an angiographic search of the population of England and Wales over the age of 30 years could be expected to yield eight pairs of male and 37 pairs of female monozygotic twins with at least one cerebral aneurysm. In any pair of female monozygotic twins of whom one has an aneurysm the chance of the other having an aneurysm at any site would be 1 in 50 (that is, an order of risk comparable with that of the female population at large). The chances of each twin having an uncommon (for example, "carotid-ophthalmic") aneurysm⁵, as in Mr. Fairburn's cases, would be of the order of 1 in 1,000; and of each twin having a common (for example, middle cerebral) aneurysm they would be of the order of 5 in 1,000. It can be predicted that in the U.K. as a whole, one case of twin aneurysmal subarachnoid haemorrhage in females should come to light every year, and one such case in males every four years (D. J. B. Ashley, 1973, personal communication).

It seems reasonable to postulate a transmissible genetic factor in some cases of familial subarachnoid haemorrhage, especially when kinship is close⁶ and the aneurysms congruous. Not only are the aetiological implications of twin aneurysms of obvious importance, but the possibility, in sibs "at risk," of elective investigation and surgical prophylaxis^{7,8} cannot be overlooked. These are further reasons for neurosurgeons to pool their case material in this field.—We are, etc.,

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SIR,—Mr. B. Fairburn (27 January, p. 210), reporting "twin" intracranial aneurysms causing subarachnoid haemorrhage in a pair of monozygous "twins," concludes that some common genetic factor was involved. I would suggest that this is a good example of "mirror-imaging," common in identical twins (in about 30%), which results from the early twinning-division of the zygote (fertilized ovum).—I am, etc.,

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Gonococcaemia in the Family

SIR,—The presentation of gonorrhoea in a family has previously been reported¹ and since then a more unusual family has come our way.

A healthy, 23-year-old West Indian woman gave birth to a baby boy. Four days later she developed a maculopapular eruption on the legs, arms, and buttocks; arthritis of the left knee and ankle; and a pyrexia of up to 102°F (38.9°C). Gram-negative intracellular diplococci were found in a smear taken from the urethra though they were not grown in culture. A clinical diagnosis of gonococcal septicaemia was made and she was treated with procaine penicillin intramuscularly and made a rapid recovery. At the same time her baby developed ophthalmia, the gonococcus being found in both smear and culture.

She denied intercourse with anyone but her husband, who, as it happened, was being investigated in the same hospital for hypertension. He had had no urinary symptoms or urethral discharge and swabs from the urethra were negative for the gonococcus. He was given no treatment. Three months later the husband was admitted to hospital with a three-day history of rash, joint pains, and shivering attacks. His temperature was 103°F (39.4°C), and there was a scanty erythematous and vesicular rash on his trunk, arms, and legs. The right knee joint and right wrist were hot, swollen, and very tender, the former containing a sizeable effusion. He denied any previous genitourinary symptoms and there was no clinical evidence of urethral discharge. He did not admit to any extramarital relationship. *Neisseria gonorrhoeae* were isolated from a blood culture though not from a specimen of the effusion in the knee joint. A swab