

PAPERS AND ORIGINALS

Operative mortality and postoperative morbidity of highly selective vagotomy

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Summary

In a world-wide survey of the results of 5539 highly selective vagotomies (HSVs) performed electively for duodenal ulcer the operative mortality was found to be 0.3%. This was lower than that found in collected series after either vagotomy with drainage (0.8%) or gastric resection with or without vagotomy (over 1%). Necrosis of the lesser curvature occurred in 10 patients (0.2%) after HSV and caused death in 5 (0.1%). Such necrosis is probably ischaemic in origin. Hence reperi-tonealisation of the raw area on the lesser curvature and prompt laparotomy if the patient develops signs of peritonitis might lower the mortality still further. Three deaths were due to pulmonary embolism, one to mesenteric vascular occlusion, and four to myocardial infarction; such deaths might be reduced by the prophylactic use of low-dose heparin. Persisting gastric stasis requiring drainage occurred in only 0.1% of the patients in the early postoperative period and in 0.6% of the patients later. Hence drainage procedures, which produce side effects such as early dumping, bilious vomiting, and diarrhoea, could be abandoned if the mean incidence of recurrent ulceration after HSV remains close to its present level. HSV is probably the safest operation for duodenal ulcer because the alimentary tract is not opened and there is no anastomosis, suture line, or stoma.

Introduction

"Death is the worst thing that can happen to a patient with a peptic ulcer"—L R Dragstedt.

Modern surgery for duodenal ulceration is relatively safe. In large collected series the "average" operative mortality for

vagotomy with drainage was 0.8%,¹ and for vagotomy with antrectomy 1.0 to 1.2%^{1 2}; the operative mortality for Polya partial gastrectomy is probably between 1 and 2%. Since death after operation is so unusual the differences in mortality rates are never statistically significant. But they are by no means "insignificant" to the patient. The phrase "1% mortality" means nothing less than 100% mortality for the man who dies. In the case of peptic ulcer that man is apt to be young, with a wife and young family.

When highly selective vagotomy without drainage (HSV) was introduced³ it seemed that the operative mortality of surgery for duodenal ulcer might decrease still further, because HSV entails no breach of the integrity of the alimentary tract and there is no suture line or anastomosis to leak or bleed. Early experience with HSV in Leeds and Copenhagen tended to confirm this view,³⁻⁵ with no deaths occurring in several-hundred cases. Isolated case reports then began to appear, however, describing necrosis of the lesser curvature, peritonitis, and sometimes death after HSV.⁶⁻⁹ My aim, therefore, was to attempt to define the operative mortality of HSV in large numbers of patients and to try to discover how often lesser-curve necrosis and gastric stasis occurred.

Method

A questionnaire was sent to all surgeons (table I) whom I knew to be using HSV for the elective treatment of duodenal ulcer (DU). Replies were received from all of them. The surgeons listed in table I include those who reported the first four cases of lesser-curve necrosis after HSV. The surgeons were asked the following questions:

- (1) How many elective HSV operations have you done for DU?
- (2) Have you had any deaths after HSV?
- (3) What percentage of all DU patients coming to operation do you treat by HSV?
- (4) Have you had any cases of gastric or oesophageal necrosis?
- (5) How many patients have had gastric stasis or ileus in the early post-operative period?
- (6) Have you found any cases of "late" stasis, months or years after operation?

Results

Operative mortality—Out of all 5539 patients who had undergone elective HSV 17 (0.3%) had died (table I).

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TABLE 1—Mortality and morbidity from highly selective vagotomy in different centres (replies to questionnaire)

No	Names of surgeons	Centre	No of HSVs	No of postoperative deaths	Percentage of DU patients treated by elective HSV	No of cases of necrosis of stomach	No of cases of early gastric stasis	No of cases of late gastric stasis
<i>Great Britain</i>								
1	H L Duthie	Royal Infirmary, Sheffield	100	1	50	1	0	1
2	H Burge	Charing Cross Hospital, London	350	0	100	0	0	2
3	C Wastell	Westminster Hospital, London	77	0	95	0	0	0
4	R M Kirk	Hampstead General Hospital, London	78	0	60	0	1	0
5	D Hancock	Sunderland General Hospital	55	0	60	0	0	0
6	D Cowley	University Hospital of South Manchester	38	0	50	0	0	1
7	D Hopton	York County Hospital	24	0	50	1 (?)	0	0
8	R Hall	Royal Victoria Hospital, Belfast	66	2	40	1	3	2
9	T Kennedy	Royal Southern Hospital, Liverpool	160	1	(90) 50	0	1	0
10	J McFarland	General Hospital, Birmingham	20	0	?	0	0	0
11	J Alexander Williams	Airedale General Hospital	76	1	50	0	0	0
12	W A F McAdam	Central Middlesex Hospital, London	38	0	75	0	—	—
13	J F Newcombe	Lewisham and St John's Hospital, London	34	1	70	1	0	0
14	H I Le Brun	University College Hospital, London	24	1	—	0	5	3
15	J H Wylie and C G Clark	Pontypriid and Rhondda Hospitals, Wales	99	1	90	1	2	2
16	R J Williams	General Infirmary, Leeds	15	0	100	0	0	—
17	J C Goligher and D Johnston	General Infirmary, Leeds	300	0	97	0	4	2
<i>Scandinavia</i>								
18	O Kronborg	Bispebjerg Hospital, Copenhagen	37	0	60	0	1	0
19	P Madsen	Bispebjerg Hospital, Copenhagen	200	0	50	0	?	2
20	E Amdrup	Kommunehospital, Arhus	139	1	33	0	0	1
21	H-E Jensen and E Amdrup	Kommunehospital, Copenhagen	158	0	100	0	0	1
22	I Liavag	Kommune Aker Sykehus, Oslo	301	0	100	0	0	6
23	S Kedenstedt	Nacka Sjukhus, Sweden	350	0	100	0	1	0
24	A Faxen and N Kock	Surgical Department III Goteborg	90	0	65	1	1	1
25	L Olbe and U Grotzinger	Surgical Department III Goteborg	111	0	64	0	2	1
26	G Liedberg	Department of Surgery, University of Lund	180	0	85	0	1	3
27	P Heimann	Department of Surgery, University of Bergen	150	1	?	1	?	?
28	L-A Civalero	Sodersjukhuset, Stockholm	134	0	90	1	1	0
<i>Continent of Europe</i>								
29	J De Miguel	Valladolid, Spain	160	1	95	0	0	0
30	L Imperati	Foggia, Italy	420	1	100	1	0	0
31	G Grassi	Rome, Italy	488	0	85	0	0	0
32	G Heberer	University Surgical Clinic, Munich	243	3	100	1	5	3
33	H Muller	Academisch Ziekenhuis, Rotterdam	40	0	50	0	1	0
34	M Allgower and A H Amery	Kantonsspital, Basel	260	1	100	0	?	?
35	T Charlo*	Seville, Spain	152	0	? 90+	0	—	—
36	C Pera	Barcelona, Spain	30	0	?	0	—	—
37	R Picaud	Marseilles, France	50	0	50-75	0	2	0
38	H de Kok	Gorinchem, Netherlands	30	0	—	—	—	—
<i>United States of America</i>								
39	P H Jordan	Baylor College of Medicine, Houston	75	0	50	0	0	1
40	G A Hallenbeck	University of Alabama, Birmingham	56	0	80-85	0	2	0
41	K Eng	University Hospital, New York	21	0	50	0	1	0
42	E Harada and J Zubiran*	Hosp ISS, STE, Mexico	45	0	?	0	0	0
<i>Australia</i>								
43	G A E Coupland	Royal North Shore Hospital, Sydney	65	1	85-90	0	"10%"	1
Total			5539	17 (0.31%)		10 (0.18%)	36 (0.65%)	35 (0.63%)

*Data derived from published work.

Lesser-curve necrosis—The causes of death are shown in table II. Five deaths (0.1%) were attributable to necrosis of the lesser curvature or fundus of the stomach. The total incidence of necrosis of the lesser curvature or fundus was 10 cases (0.2%). Four patients died of myocardial infarction, three of pulmonary embolism, and one from mesenteric vascular occlusion (table II).

TABLE II—Causes of death in 5539 highly selective vagotomies

	No of cases	% of all operations
Myocardial infarction	4	0.07
Pulmonary embolism	3	0.05
Pneumonia/respiratory failure	2	0.04
Haemorrhage { Intraperitoneal	1	0.04
Haemorrhage { Gastric erosions	1	
Mesenteric vascular occlusion	1	0.02
Necrosis of lesser curvature*	4	0.09
Necrosis of fundus	1	
Total	17	0.31

*Signs of peritonitis appeared between 2 and 10 days after operation.

Gastric stasis—Delay in gastric emptying lasting three days or more occurred in 36 (0.7%) of the patients in the early postoperative period, but only seven (0.1%) needed reoperation, the remaining cases resolving spontaneously. Gastric stasis necessitating drainage some months later occurred in 35 patients (0.6%). Thus the total incidence of persisting gastric retention was 0.8% (42 cases).

Discussion

These results show clearly that HSV is an extremely safe

procedure, the operative mortality in 5539 elective operations for duodenal ulcer being 0.3%. For comparison, the operative mortality of truncal vagotomy with drainage (TV+D) was 0.8% in 6490 patients, and that of truncal vagotomy with antrectomy (TV+A) was 1.6% in 1725 patients.¹ The operative mortality of elective TV+A in 1300 mainly private patients in Nashville was about 1%.² The operative mortality for elective Polya partial gastrectomy for duodenal ulcer is difficult to ascertain because of the relative paucity of recent published reports,¹⁰⁻¹⁵ but in skilled hands it is probably between 1 and 2%.

All these mortality rates are doubtless biased, because they are based on reports of large series of patients who had usually been operated on by surgeons of unusual experience and skill. Moreover, surgeons are less inclined to report poor results than good results. Such bias may have been present to an unusually large degree in the present study because many of the surgeons concerned are noted exponents of the techniques of selective and highly selective vagotomy. The reverse, however, might equally be true. Quite possibly this study was biased against HSV, because it reflects early experience with the operation; the results might be even better when surgeons acquire greater familiarity with the technique. The results that were obtained certainly support the initial hypothesis that HSV is safer than any of the standard operations for duodenal ulcer. If it is permissible to apply tests of significance to results from collected series, HSV appears to carry *significantly* less risk to life (HSV *v* TV+D: $\chi^2 = 10.77$; $P < 0.01$) than does any of the other procedures.

The second main conclusion is that even when allowance is made for the possibility of technical error in some cases, lesser-curve necrosis is a specific complication of HSV. It occurred in

about 1 in 500 cases in this series and caused death in 1 in 1000. The exact pathogenesis is not clear, but the necrosis is presumably ischaemic in origin and caused by the laying bare of most of the lesser curvature. It may be attributable to the relatively poor submucosal blood supply in that region as compared with the rich anastomotic network in the submucosa of the anterior and posterior walls of the stomach.¹⁶⁻¹⁸ Whatever the cause may be, knowledge of this potential complication should help in its prevention. Devascularisation of the stomach should be kept to a minimum by careful preservation of all arteries to the stomach except the branches of the left gastric artery, which must be sacrificed. The uppermost short gastric arteries should be preserved when possible, and if the spleen has to be removed on account of iatrogenic trauma the increased risk of subsequent lesser-curve necrosis must be borne in mind. At the end of the operation it might be wise to cover the raw area on the lesser curvature with peritoneum by suturing the cut edges of serosa.

Necrosis of the lesser curvature seems to be particularly likely to occur when HSV is used prophylactically for duodenal ulceration in patients about to undergo renal transplantation. Death due to necrosis of the lesser curvature occurred in two such patients in Zurich¹⁹ and in one in Munich.²⁰ The total number of transplant patients treated by HSV in these two centres was not ascertained precisely, but the mortality of HSV in such patients certainly seems to be high. Hence the use of HSV in these patients is contraindicated, at least until more information is available. Finally, awareness of the possibility of lesser-curve necrosis should prompt surgeons to re-explore the abdomen of any patient who develops signs of an intra-abdominal disorder during the first 10 days after the operation.

Twelve of the 17 deaths after HSV in this series were not due to gastric necrosis, no fewer than eight being due to thrombosis, embolism, or myocardial infarction (table II). Thus it seems reasonable to suggest that the use of prophylactic low-dose heparin might lower the operative mortality of HSV still further.^{21 22}

The third main conclusion is that when vagotomy is confined

to the proximal two-thirds or so of the stomach, as in HSV, troublesome gastric stasis is rare, only 0.8% of the patients in this study subsequently needing drainage. Thus provided that in the long term HSV proves as effective as vagotomy with drainage in curing the ulcer, the use of drainage procedures could be abandoned and their attendant complications such as dumping, diarrhoea, and bilious vomiting could be largely prevented.^{5 23 24}

I wish to thank all the surgeons listed in table I for kindly furnishing details of their patients treated by HSV.

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Local infusion of urokinase and heparin into renal arteries in impending renal cortical necrosis

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Summary

Two patients with presumed impending cortical necrosis, after haemolytic uraemic syndrome in one and after

concealed accidental haemorrhage in the other, were treated by local infusion of urokinase and heparin into the renal artery. Both recovered and one regained normal renal function. Local infusion of anticoagulants or thrombolytic drugs into one renal artery offers the possibility of a controlled examination of the efficacy of this treatment in preventing cortical necrosis.

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Introduction

The mortality rate among patients with the haemolytic uraemic syndrome has fallen with the availability of supportive measures, particularly peritoneal dialysis,¹ but there remain patients in whom bilateral cortical necrosis leads to irreversible renal failure. Heparin has been advocated for arresting intravascular coagulation, and more recently thrombolytic treatment in the form of streptokinase has been used in an attempt to lyse intrarenal fibrin.^{2 3} Both these treatments may lead to generalised haemorrhage, and the risks of treatment may outweigh the