Progress report

Vagotomy for gastric ulcer

In all aspects of the surgical treatment of peptic ulcer the trend, in the past decade, has been to substitute vagotomy and a drainage procedure for gastric resection. When considering gastric ulceration, no uniformity of opinion can be found on its aetiology, and so various explanations are put forward for the success of vagotomy and a drainage procedure. The rationale for the vagal denervation of the stomach largely rests on the theory that gastric ulcer is due to antral overactivity25. If delay in gastric emptying is thought to be important, a pyloroplasty may be done with the aim of avoiding stasis and also of preventing further hold up if truncal vagotomy is performed. However, if some form of selective vagotomy is performed, a drainage procedure may not be required for the normal emptying of the stomach26,27,28, so the surgeon will then have to decide whether he should add a pyloroplasty to the procedure for its own sake. Certainly, the cases reported in which a drainage procedure has been the only treatment, have had a high recurrence rate, for example, 22%17 or 50%13 or 57%29. On the other hand, if regurgitation of bile is thought to be important in the aetiology of gastric ulcer30,31,32, pyloroplasty should be avoided, if at all possible, or the success of the procedure should be explained by suggesting that a regurgitant jet of bile has been converted to a flow through the widened pylorus.

The conversion to vagotomy for gastric ulcer has been less pronounced than in the case of duodenal ulceration. Several factors contribute to the reluctance surgeons feel in adopting vagotomy for gastric ulcer. It is widely held that the results of gastric resection for gastric ulcer are superior to those when duodenal ulcer is treated in this manner. The explanations advanced are that since hypersecretion of acid and pepsin is not a factor in the aetiology of the majority of gastric ulcers, there is little risk of stomal ulceration and less of the stomach needs to be resected so that the troubles of the ‘small stomach syndromes’ are less obvious, especially as the gastric remnant can usually be safely anastomosed to the duodenum (Billroth I gastrectomy)1,2. The result is that surgeons who have changed to vagotomy for duodenal ulcer are still waiting to be convinced that they should abandon Billroth I gastrectomy for gastric ulcer in favour of vagotomy and pyloroplasty.

Four main lines of evidence are required to establish a change in this aspect of surgical practice: (1) definition of the risks of the operation; (2) knowledge of the long-term incidence of recurrent ulceration; (3) assessment of the functional after effects of the operation; and (4) estimation of the likelihood of the gastric ulcer being malignant and of the risks of misdiagnosis when the ulcer is not being removed.

Risks of the Operation

A reduced mortality and morbidity are strongly advanced as reasons for
favouring vagotomy over Billroth I gastrectomy. In fact, the technical aspects of the operation were amongst the first suggestions for preferring vagotomy. It was put forward as a solution for the treatment of a gastric ulcer high on the lesser curvature of the stomach, where a gastrectomy would be technically difficult, and would have increased chance of fistula formation and of encroaching on the oesophagus with the suture line, with resulting stenosis of the cardia\(^3\). It is always difficult to be precise about the mortality involved in any operation because most of the published reports come from centres with a special interest in the procedure so that the mortality may be less than could be expected throughout the country. Despite this difficulty, most recent estimates of the mortality of Billroth I gastrectomy show no real difference from vagotomy and pyloroplasty (Tables I and II). However, a greater risk of postoperative complications can be expected when a gastroduodenal anastomosis is performed. In our controlled comparison of vagotomy and pyloroplasty and Billroth I gastrectomy, no deaths occurred, but the immediate morbidity was twice as great after gastrectomy, although no long-term effects resulted\(^4\).

<table>
<thead>
<tr>
<th>Series</th>
<th>No. of Patients</th>
<th>Operative Mortality (%)</th>
<th>Recurrence (%)</th>
<th>Length of Follow Up (yr)</th>
</tr>
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<tbody>
<tr>
<td>Walters et al(^1) (1957)</td>
<td>113</td>
<td>1-2</td>
<td>4-4</td>
<td>5-10</td>
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<tr>
<td>Hermon Taylor(^4) (1959)</td>
<td>42</td>
<td>0</td>
<td>0</td>
<td>10</td>
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<td>Harvey(^4) (1961)</td>
<td>448</td>
<td>2-9</td>
<td>1-5</td>
<td>3-25</td>
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<tr>
<td>Angel et al(^1) (1967)</td>
<td>127</td>
<td>1-6</td>
<td>1-6</td>
<td>—</td>
</tr>
<tr>
<td>Hampson et al(^4) (1968)</td>
<td>46</td>
<td>2-2</td>
<td>—</td>
<td>—</td>
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<tr>
<td>Stemmer et al(^4) (1968)</td>
<td>87</td>
<td>0</td>
<td>2-15</td>
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<tr>
<td>Welch and Burke(^4) (1969)</td>
<td>347</td>
<td>1-7</td>
<td>1</td>
<td>1-10</td>
</tr>
<tr>
<td>Duthie et al(^4) (1970)</td>
<td>42</td>
<td>0</td>
<td>2-4</td>
<td>up to 5</td>
</tr>
<tr>
<td>Total</td>
<td>1,252</td>
<td>1-8</td>
<td>1-5</td>
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</tr>
</tbody>
</table>

Table I Results of Billroth I gastrectomy for gastric ulcer

\(^1\)Including some other types of gastrectomy

<table>
<thead>
<tr>
<th>Series</th>
<th>No. of Patients</th>
<th>Operative Mortality (%)</th>
<th>Recurrence (%)</th>
<th>Length of Follow Up (yr)</th>
</tr>
</thead>
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<td>55</td>
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<tr>
<td>Hendry and Bahrami(^1) (1965)</td>
<td>42</td>
<td>0</td>
<td>2-4</td>
<td>Up to 6(^4)</td>
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<tr>
<td>Farris and Smith(^4) (1966)</td>
<td>29</td>
<td>7-0</td>
<td>0</td>
<td>Up to 6</td>
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<tr>
<td>Kraft et al(^1) (1966)</td>
<td>28</td>
<td>0</td>
<td>0</td>
<td>Up to 17</td>
</tr>
<tr>
<td>Read(^1) (1966)</td>
<td>40</td>
<td>0</td>
<td>0</td>
<td>Up to 2</td>
</tr>
<tr>
<td>Burke(^1) (1966)</td>
<td>72</td>
<td>0</td>
<td>0</td>
<td>Up to 3</td>
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<tr>
<td>Dortor(^4) (1966)</td>
<td>30</td>
<td>6-7</td>
<td>6-7</td>
<td>5</td>
</tr>
<tr>
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<td>14</td>
<td>0</td>
<td>35-7</td>
<td>6-7</td>
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<tr>
<td>McNell et al(^4) (1969)</td>
<td>30</td>
<td>3-3</td>
<td>3-3</td>
<td>2(^4)</td>
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<tr>
<td>Duthie et al(^4) (1970)</td>
<td>35</td>
<td>0</td>
<td>14-3</td>
<td>Up to 5</td>
</tr>
<tr>
<td>Total</td>
<td>375</td>
<td>1-4</td>
<td>5-2</td>
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Table II Results of vagotomy and pyloroplasty for gastric ulcer

Recurrent Ulceration

One obvious assessment of any operation for gastric ulcer is its efficiency in curing the ulcer. It has been established in several series by radiological studies that ulcers, left in the stomach after vagotomy and pyloroplasty do heal\(^5,6,7\), so that the initial step in the cure is well documented. However, bed rest alone can heal gastric ulceration, so that it is more important to assess the long-term results, which are not quite so uniform. Several series have been reported with recurrent ulcer rates ranging from 0 to 36\% (Table II). In this collected series, the average incidence of recurrence is 5\%. It is noticeable that the recurrences may become evident after several years, so
that the incidence in some of these series might be expected to increase with the passage of time. In this respect, the data available for Billroth I gastrectomy are more reliable (Table I) and give a mean recurrence rate of 1.5%.

The evaluation of recurrence is made more difficult by the inclusion of combined gastric and duodenal ulcers in some of the reported studies. The aetiology of pyloric ulcers, and of gastric ulcer associated with duodenal ulcer, may well be different from that of gastric ulcer alone, although some workers8 maintain that every gastric ulcer is associated with some disease in the pyloric canal or duodenal bulb. In a large collected series, Johnson21 found an incidence of 22% of duodenal ulcer in 5,023 patients operated on for gastric ulceration. He believed that three main groups of ulcers could be defined in terms of secretory data and of blood group preponderance. Other reports22,23,24 have emphasized the lack of uniformity in the clinical features and secretory characteristics of gastric ulceration. Most workers agree that gastric ulcers associated with duodenal ulceration and those found in the pyloric or prepyloric region have secretory characteristics more like duodenal ulcers and should be treated in a similar manner. This means that about one fifth of patients with gastric ulcer would be treated by vagotomy and pyloroplasty, with results similar to that of duodenal ulcer alone. Unfortunately, it is not possible to separate the combined ulcers from the other gastric ulcers in most of the reported work so that evidence is not clear whether the combined group fare particularly well or not.

If the level of secretion of acid and pepsin by the stomach is important in the pathogenesis of gastric ulcer, then recurrence may be due to ineffective vagal denervation. Information about the completeness of vagotomy is not available in all reports, and, even when it is presented, the method of testing for completeness of vagotomy varies from being done at operation28 to stimulating the stomach by insulin hypoglycaemia at different intervals after operation24,25,26. No consensus of surgical opinion can be found on the relative merits of this testing. However, it has been shown that recurrent gastric ulceration, after vagotomy, can be cured by re-operation to complete the vagotomy5,17 or by improving inadequate drainage of the stomach37. In our Sheffield series, the five patients with recurrent ulcer have all had complete vagotomy, as judged by the insulin test of gastric secretion, and four had no radiological evidence of gastric stasis4: these circumstances were also found by Stemmer18 in one case. Thus, we have to look for some other explanation, such as increased bile reflux, to account for these recurrences. In an endeavour to avoid some of the difficulties of having a mixed population of types of ulcers, we have excluded all combined ulcers from our trial. Over a follow-up period of six months to five years (average 36 months) we have had one recurrence in 42 patients treated by Billroth I gastrectomy and five in 35 patients treated by vagotomy and pyloroplasty. The difference is not statistically significant.

Functional Results

Apart from recurrent ulceration, a good result of a gastric operation may be marred by side effects occurring after a meal, such as epigastric fullness and distension, bilious vomiting, oesophagitis or vasomotor symptoms, which may lead to a diminished dietary intake. This, together with some malabsorption, can be followed by extreme weight loss and anaemia and the 'gastric cripple' results. The increased attention paid to such consequences after partial gastrectomy for duodenal ulceration has been the main impetus to the increasing use of vagotomy and a drainage procedure. The lesser resection involved in treating gastric ulcer, together with the reconstitution
of the alimentary canal, so that the duodenum is in continuity, makes these sequelae less common and less troublesome. Thus, the Billroth I gastrectomy is more difficult to displace from surgical practice than the Polya gastrectomy. As more long-term follow-up results after vagotomy and a drainage procedure become available, it has been found that postprandial symptoms, including vasomotor dumping, are quite common, but the incidence and severity of weight loss is less than has been found after gastrectomy. Reports of the results of vagotomy for gastric ulcer usually give general statements that good results had been obtained in most patients. Statements abound such as 'side effects following pyloroplasty (and vagotomy) have been infrequent and mild', followed by a list of side effects in up to 10% of cases. Workers in Britain have tended to emphasize the symptoms after operation and many use a modification of Visick's classification of such symptoms. In this, four grades are established: (1) with no gastric symptoms; (2) with mild symptoms easily corrected by avoiding some types of food or by changing the size of meals; (3) symptoms not controlled by simple measures but not interfering with social or working life; (4) symptoms interfering with social or working life, and as bad or worse than before operation.

Grades 1 and 2 accounted for 94% of 39 cases followed for up to six and a half years, and in a shorter follow-up using this classification, the most recent series of 33 cases reported that 88% were in grade 1. These claims of a high proportion of excellent results must be set against previous reports on Billroth I partial gastrectomy for gastric ulcer. Walters reported 93% of 113 patients with excellent or good results with over seven years of follow up, and Hermon Taylor observed only abdominal distension after a full meal in 18 of 42 patients after 10 years.

In our Sheffield series, direct comparison of the two operations shows a slightly higher proportion of patients after Billroth I gastrectomy with a grade I result (40%) than after vagotomy and pyloroplasty (34%). The reason for this is the greater number of postvagotomy patients with feelings of distension and mild intestinal colic or dumping symptoms, which brings a large proportion into grade 2. Too few cases have been reported to be able to assess whether selective or truncal vagotomy is the better type of vagal section to use for gastric ulceration.

Risk of Cancer

It is a matter of history that the belief that gastric ulcer was a strong aetiological factor in the development of gastric cancer has waned steadily to the extent that a review on the epidemiology of gastric cancer in 1967 stated that 'the general consensus of opinion holds that, in the western world, no significant relationship occurs'. The problem which faces the surgeon who is considering leaving a gastric ulcer in situ is not this epidemiological concept, but the question whether the ulcer, at that moment, is benign or malignant. By the time the patient has come to operation, many malignant ulcers will have been excluded, usually by radiology, often by gastroscopy and, occasionally, by gastric cytology, and these ulcers will be treated by gastric resection. In spite of the high overall diagnostic accuracy of these methods, about 95%, there still remains a group in whom the ulcer is wrongly diagnosed, before operation, as benign. If the surgical procedure is to resect the stomach, this diagnostic error will not be significant, unless the ulcer is above the incisura, when an inadequate local clearance will result. On the other hand, leaving the ulcer and performing a vagotomy and pyloroplasty would fail to remove the cancer in all cases.

The extent of the risk involved can be assessed from the reports of cancer.
of the stomach in series of patients treated conservatively for gastric ulcer. It depends also on the further diagnostic procedures undertaken at surgery. Naked-eye examination of the ulcer was accurate in all the series of 42 cases of Hendry and Bahrani. Two patients in a group of 33 died of gastric cancer (one definite, one suspected) after vagotomy and pyloroplasty for gastric ulcer\textsuperscript{20}, and another patient similarly died of gastric cancer out of a series of 60\textsuperscript{22}. Even when ‘four quadrant biopsy’ was used, one patient with cancer was not diagnosed in 84 patients\textsuperscript{16}. By using excision biopsy of all the edge of the ulcer, we have found six cases of cancer in 92 gastric ulcers which looked benign on naked-eye examination. No matter how small the risk of making a false diagnosis of a benign ulcer, it is incumbent on the surgeon to avoid it, as far as possible. This is both to protect his patient from an incorrect operation for gastric cancer and also to ensure that a radical gastrectomy is not done for a benign lesion. Ideally, each gastric ulcer should have a full biopsy of all the edge where any early malignant change would be found. All the cases found in our own series looked benign to the naked eye and no local manifestations were observed to direct suspicion to one particular quadrant or another. If histological examination cannot be made at the time of the operation, the delay of two or three days will not materially effect the outcome if it is found necessary to re-operate for gastric cancer. It does expose the patient to a further operation but the number of cases in which this will occur is small and is preferable to ‘playing safe’ at the first operation by doing a radical resection when any doubt arises: preferable, both in terms of immediate mortality and long-term morbidity.

**Conclusion**

No general agreement is to be found on the best policy for surgical treatment of gastric ulceration. Vagotomy and pyloroplasty has not yet been established as a routine treatment for all gastric ulcers. The attraction of a potentially lower mortality rate, especially when high lesser curve ulcers have to be treated by surgeons not widely experienced in gastric resection, cannot be gainsaid. However, an ulcer in this situation gives rise to technical difficulties if it has to be exposed through a separate gastrotomy incision, to permit a full inspection and an adequate biopsy. The lower morbidity of vagotomy and pyloroplasty has to be balanced against the two factors: first, recurrent ulceration is not less than after Billroth I gastrectomy and may be more: secondly, the risk of leaving a gastric cancer in situ. This small, but definite, risk must be avoided. The surgeon must ensure that this operative diagnosis is correct. Short of excision of the ulcer, this requires a biopsy of all the edge of the lesion and not just of four quadrants. The results reported to date do not justify the abandonment of partial gastrectomy in the treatment of benign gastric ulcer.

H. L. DUTHIE  
*University Department of Surgery,*  
*The Royal Infirmary, Sheffield*

**References**

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