



Long-term Prognosis after Partial Gastrectomy for Gastroduodenal Ulcer

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Abstract. The decline in duodenal ulcer disease and the established relation of peptic ulcer to *Helicobacter pylori* have virtually abolished the need for elective ulcer surgery. However, a substantial proportion of the population around retirement age has previously been subjected to partial gastric resection due to peptic ulcer, and the long-term outcome of these patients is of continuing relevance. Patients subjected to elective surgery could represent a selected group of healthy subjects with a lower overall morbidity, but reports indicate that patients operated on for peptic ulcer have more advanced disease associated with excess smoking and a different pattern of social behavior. The surgical procedure induces enterogastric reflux, leading to profound changes in the remnant mucosa and the formation of carcinogens in the gastric juice. In addition, metabolic abnormalities are common, especially fat malabsorption. Evaluation of the impact of these factors on morbidity and mortality is difficult. Increased mortality in gastrointestinal tumors (especially gastric stump carcinoma), respiratory diseases and other smoking-related malignancies, and suicide are found in the long-term follow-up after partial gastric resection due to peptic ulcer. However, these hazards to life are offset by a decreased mortality in cardiovascular disease. Preventive measures against suicide and especially tobacco smoking are recommended to improve the outcome for this cohort.

The decline in duodenal ulcer disease, together with the possibility of permanently healing a peptic ulcer through eradication of *Helicobacter pylori* (HP), has dramatically diminished the need for ulcer surgery. In 1981 it was estimated that about 1% of the adult Swedish population had been subjected to surgery for benign gastroduodenal disease [1]. Although the frequency in the population will eventually decline, there will still be a substantial proportion of the elderly population previously operated on due to peptic ulcer in our country during the next decades [2, 3], and for an even longer period in the rest of Europe and the United States [4–6]. In Sweden, there was a decline from an annual rate of ulcer surgery of 72.1/100,000 inhabitants in 1956 to about 45.0/100,000 ten years later [3], whereas peptic ulcer surgery was performed on 71.1/100,000 British men in 1970 [6] and about 120,000 Americans the same year (approximately 60/100,000) [5]. Most of these patients have been subjected to partial gastric resection; and as the median age at ulcer surgery was about 44 years [7], many of those operated on before the 1970s have not yet reached retirement age. Thus the long-term outcome of these patients is of continuing interest.

In 1922 Balfour published the first report on gastric stump carcinoma as a long-term sequela after partial gastrectomy [8]; and some years later Beatson reported an increased risk of gastric

carcinoma after remote surgery with gastroenterostomy [9]. Gastric stump carcinoma was later defined as a carcinoma developing in the gastric remnant more than 5 years after surgery for benign gastroduodenal disease [10]. There has been a lively debate as to whether it is the gastric resection that increases the risk of subsequent gastric carcinoma or if other factors are of importance. The impact of stump carcinoma on the long-term outcome of these patients, compared to the other hazards for which they are at risk, has also been questioned.

Which Ulcer Patients Develop Gastric Stump Carcinoma?

Several reports have indicated an increased risk of developing stump carcinoma after surgery for gastric ulcer [10–17], whereas duodenal ulcer patients seemed to be unaffected [7, 18]. However, when large cohorts with long follow-up were investigated, an increased risk was found in duodenal ulcer patients as well [13–16] but with a delay of about 20 years [12, 19]. In contrast to that in duodenal ulcer patients, the mucosa in gastric ulcer patients manifests a degree of gastritis and atrophy [20], which might account for the earlier development of carcinoma in these patients. Janunger and coworkers found the differences between remnant mucosa after duodenal and gastric ulcer surgery to have vanished after a period of about 20 years, except for intestinal metaplasia, which was found only in patients who had had surgery for gastric ulcers [21]. Duodenal ulcer patients are known to have chronic antral gastritis due to HP infection [22] but an otherwise intact mucosa [23]. After resection a synergistic effect between HP infection and bile reflux undermine the mucosa in the gastric remnant with subsequent malignant transformation [24]. If however, antral mucosa with chronic gastritis is subjected to bile reflux, one might expect earlier cancer development.

Watt and colleagues investigated duodenal ulcer patients after truncal vagotomy and pyloroplasty and found the median interval between ulcer surgery and death due to gastric cancer to be only 13 years [25]. This finding was later confirmed in a Canadian study where the mean interval between partial gastrectomy and gastric cancer development was 26.2 years, whereas the interval between vagotomy with pyloroplasty and subsequent gastric cancer was only 12.5 years (esophagocardiac cancer excluded in both groups) [26]. As the distal part of the stomach is most prone to cancer development, these observations probably explain the decreased risk of stump carcinoma during the first 15 to 20 years postoper-

Table 1. Mortality due to gastric cancer in patients with previous partial gastric resection or gastroenteroanastomosis and vagotomy due to benign gastroduodenal disease: literature survey.

Study	Year	Country	No. in cohort	Traced (%)	Dead (%)	SMR	Significance	Length of follow-up (years)	Gastric ulcer (%)
Helsingen [10]	1956	Norway	229	76	19	2.12	*	10–35	43
Krause [42]	1958	Sweden	385	94	58	2.21	*	23–50	?
Liavåg [40]	1962	Norway	679	91	9	0.93	NS	15–27	41
McLean Ross [18]	1982	Scotland	856	91	46	0.77	NS	15–33	?
Eriksson [12]	1983	Sweden	1575	89	54	1.16	NS	19–49	31
Fischer [7]	1984	Denmark	1025	95	54	1.27	NS	21–29	0
Tokudome [45]	1984	Japan	3827	97	28	0.21	*	11–33	57
Caygill [13]	1986	England	5018	89	55	1.58	*	25–45	31
Asano [46]	1987	Japan	6662	93	12	0.31	*	7–22	50
Offerhaus [32]	1988	Holland	2633	90	37	1.38	NS	15–44	32
Lundegårdh [14]	1988	Sweden	6459	90	59	1.03	NS	27–35	27
Macintyre [47]	1994	Scotland	2241	90	62	1.07	NS	20–40	0
Staël von Holstein [36]	1995	Sweden	1575	92	78	1.67	*	29–59	31

From Staël von Holstein et al. [16], with permission.

*Risk significantly different from expected ($p < 0.05$); 95% confidence interval, Poisson distribution.

SMR: standardized mortality ratio; ?: proportion of underlying gastric versus duodenal ulcer disease not stated.

actively, with a subsequent increasing risk though with a longer delay after surgery for duodenal ulcer patients [12, 13, 16, 27]. It also explains the delay of cancer formation found in duodenal ulcer patients compared to those with gastric ulcer [12, 19].

A high proportion of duodenal ulcer patients in a study population thus demand a long follow-up to detect an increased cancer risk. As surgery for duodenal ulcers is performed on patients at a younger age than on those with gastric ulcers [12], the observed decreasing delay of cancer onset with increasing age at surgery, evident in several reports [12, 19, 27], could be explained by a larger proportion of older patients being operated on due to gastric ulcer. This inverse relation with decreased lag period from gastric surgery to stump carcinoma with increasing age at ulcer surgery is probably the result of reduced resistance of the gastric mucosa to noxious agents (e.g., bile reflux) with increasing age. Siurala et al. have shown gastric mucosal atrophy to be more prevalent in the elderly [28], and there is a more pronounced atrophy of the gastric mucosa in patients with gastric ulcers than in those with duodenal ulcers [29]. With a median time interval of 30 years between ulcer surgery and stump carcinoma, mortality due to other causes makes only those cancers developing after a short interval apparent in the elderly [27].

Differences in cancer risk due to underlying ulcer disease have been further elucidated in two recent reports concerning nonoperated ulcer patients [17, 30]. An increased risk for gastric cancer in gastric ulcer patients has been established, whereas duodenal ulcer disease actually protects against cancer development. The standardized incidence ratio for gastric cancer among patients with duodenal ulcers was only one-third of that among patients with gastric ulcers [30].

Impact of Time since Surgery, Gender, and Surgical Procedure

The risk of stump carcinoma increases with time since surgery [13–15, 19, 27, 31–36] and patients operated on at young age constitute a definite risk group [12, 14, 27, 36, 37] even after controlling for duration of follow-up after operation [14] although not agreed on by all [27]. Difference in stump cancer risk due to

gender has also been found by several authors, some reporting a slightly increased risk in females [13, 14, 33], but in one study an increased risk was found in men only [38]. The incidence of stump cancer has by some been found to be unaffected by type of operation [10, 19, 39–41] but larger studies have confirmed the increased risk after the Billroth II procedure proposed by Krause [42–44].

Mortality Associated with Gastric Stump Carcinoma

An increased risk of gastric stump carcinoma after partial gastric resection, regardless of underlying ulcer disease, is apparent. Differences in gastric cancer mortality reported in the literature are probably the result of the case mix in the investigated cohorts, where differences in length of follow-up and the proportions of duodenal and gastric ulcer patients have the largest impact, although gender, regional differences and type of surgical procedure might be important as well. If the cohort contains a large proportion of duodenal ulcer patients, a longer follow-up is required before the malignant development is apparent. On the other hand, a relatively short follow-up after partial gastric resection in a region at high risk for gastric cancer [45, 46] elucidates a preventive effect of surgery rather than an increased risk of stump cancer (Table 1).

Pathogenesis of Stump Carcinoma: Impact of Bile Reflux

Increased bile reflux after a Billroth II gastrectomy is the most frequently proposed explanation for gastric stump carcinoma. This theory is supported by several animal experiments in which operations that promote entero-gastric reflux increased the susceptibility of the stomach to neoplastic change without the administration of initiating carcinogens [48–53]. It has also been shown that nitrosated derivatives of taurocholic and glycocholic bile acids synthesised in vitro produce gastric adenocarcinoma in the rat [54].

Thus there seems to be strong support for the bile reflux theory, substantiated by trials in humans where removal of the pyloric barrier results in entero-gastric alkaline reflux, which degrades the

mucosal protection [55] and gradually creates chronic atrophic gastritis [56]. Bile acids, lysolecithin, and trypsin digest gastric mucus, thereby increasing the backflow of hydrogen ions. Exposure of gastric mucosal cells to hydrogen ions results in atrophic gastritis, in turn leading to loss of gastric acid secretion [57, 58]. This is followed by colonization of bacteria [59, 60] capable of degrading bile acids into carcinogenic forms [61, 62], transforming ingested nitrate to nitrite [63], and catalyzing the formation of *N*-nitroso compounds known to be highly carcinogenic [63–66].

If entero-gastric reflux is the sole reason for the development of gastritis and premalignant conditions and carcinoma in the gastric stump, one would expect deviation of the bile from the gastric remnant to induce healing of the mucosa or at least lead to some improvement. Surprisingly, however, in a recent study a progression of both gastritis and precancerous changes (i.e., atrophy, intestinal metaplasia, and dysplasia) has been found after diversion of the enteric reflux from the gastric remnant with a Roux loop. The median follow-up was 12 years (Åhsberg, Hammar, Staël von Holstein, in press). This is in contrast to the results in previously published studies where improvement was seen for some precancerous changes [67–69], though most histologic changes remained unchanged [68–70]. The follow-up time in those studies varied from 6 months to around 4 years. Factors other than bile reflux must also be considered in the pathogenesis of stump carcinoma.

Lack of Gastrin Response

There are several possible cofactors to bile reflux acting on the operated stomach and undermining the integrity of the mucosa. Gastrin exerts a trophic effect on the gastric mucosa [71, 72], and basal and postprandial gastrin release is lower after a Billroth II procedure than after antrectomy and gastroduodenostomy or in unoperated controls [73]. Although extragastric and extraduodenal gastrin release keeps basal gastrin levels within the normal range, the response to a meal is weak and appears late [73, 74]. Animal studies have shown that increased serum gastrin levels reduce the incidence of *N*-methyl-*N*-nitro-*N*-nitrosoguanidine-induced adenocarcinomas in intact stomachs [75] and after a Billroth II procedure [76], although the exact mechanism of the effect is unknown. Decreased gastrin and its effects could perhaps undermine the integrity of the gastric mucosa and make it more susceptible to exogenous carcinogens.

Helicobacter pylori

Helicobacter pylori (HP) has been shown to play a major role in many pathologic conditions of the upper gastrointestinal (GI) tract. The initial response to a HP infection is acute gastritis, later often progressing to active chronic gastritis [77]. HP infection is a risk factor not only for atrophic gastritis and intestinal metaplasia [78] but also for gastric cancer in the intact stomach [30, 78, 79]. Correa presented a human model of gastric carcinogenesis in the nonoperated stomach where infection with HP is linked to the initial stages of the histologic changes in the gastric mucosa: gastritis and atrophy, which later proceeded to intestinal metaplasia and dysplasia [79]. This model is probably also valid for the resected stomach. A synergistic effect of duodenogastric bile re-

flux and HP infection has been suggested, as the highest prevalence of intestinal metaplasia [80] and the highest levels of cell proliferation [81] are found in patients with both conditions. Most patients operated on for peptic ulcer disease are HP-positive; but after partial resection the occurrence of HP is diminished to around 22% to 47% [82–84]. HP is shown not to thrive in an alkaline environment [85, 86], and colonization of HP in the gastric epithelium has been shown to be inversely related to the concentration of bile salts in vivo [87] and in vitro [88]. This indicates a decreasing role of HP and an increasing role of other factors with time in the malignant development in the mucosa. This theory is supported by the fact that the incidence of stump carcinoma is low during the first decades after surgery but increases with time thereafter.

Role of Nutritional Deficiencies

A substantial proportion of patients subjected to partial gastric resection develop fat malabsorption [89, 90] with an ensuing risk of deficiency of fat-soluble vitamins. We also know that unoperated duodenal ulcer patients have high levels of ascorbic acid in the gastric juice probably correlated with antral gastritis due to HP [91]. This point is especially interesting, as unoperated patients with duodenal ulcer have a 50% reduction in the risk of developing gastric carcinoma [17, 30]. The fat-soluble vitamin α -tocopherol and the water-soluble ascorbic acid block intragastric nitrosation [92], and diminished levels of these vitamins might contribute to cancer formation. Whether ascorbic acid levels in gastric juice are affected by resection of the antrum remains to be established. Another factor that might contribute indirectly to malignant development through atrophy of the gastric mucosa is inadequate repair of epithelial loss owing to impaired general nutritional status [92].

Is Smoking the Most Important Factor?

Albeit the source of a variety of carcinogens (among others, nitrosamines [62]), smoking has not been proven to promote stump or gastric carcinoma. However, recent studies suggest that smoking might be important for cancer development throughout the GI tract [94] and in the stomach [95]. Theoretically, it could act as a cofactor; the irritant effect of dietary factors such as alcohol could also play a role [93, 96, 97]. Much controversy still exists regarding the pathogenesis of the malignant disease in patients after partial gastrectomy. An increased risk of developing carcinoma in the buccal cavity [98], lungs [12, 18, 34, 36, 46, 99–102], esophagus [101, 103], liver [100], biliary tract [101, 103], pancreas [18, 36, 103, 104], colon and rectum [18, 36, 100, 103, 105], bladder [34, 102], breast [103], and brain and nervous system [34] has been reported; and overall increased mortality due to malignant disease is apparent in most studies. Mortality is also higher in various nonneoplastic diseases, mainly respiratory and GI diseases [12, 18, 36, 42, 46, 99–102]; and most of these diseases are associated with smoking. Increased mortality due to cancer and diseases in the respiratory organs is apparent regardless of time since surgery, whereas deaths due to tumors in the GI tract are increased with a latency of 20 years or more since surgery [16, 103]. This lends support to the theory proposed by Caygill et al.

that gastrectomy produces a circulating carcinogen that acts at distant sites, probably enhanced by smoking [103].

Can Ulcer Surgery Reduce the Risk of Cardiovascular Disease?

A lower mortality than expected for various diseases has been found in several studies, especially in those with a somewhat short follow-up. It might reflect an effect of selection during the preoperative phase rather than an effect of surgery. An intriguing fact is the reduced mortality associated with diseases of the heart and vascular system found in many studies [16, 36, 99, 101, 102], although not in all [18, 47, 106]; the picture is not entirely uniform. Mortality due to cardiovascular disease (CVD) has been found to be decreased among both medically treated and surgically treated gastric ulcer patients with a follow up of 8 to 18 years [107]. Asano et al. found short-term (7–22 years) mortality due to CVD to be increased only among the postgastrectomy patients who were smokers [46], whereas a Dutch study found significantly more CVD deaths among patients operated on due to gastric ulcer and a lower CVD death rate in those operated on for duodenal ulcer [102].

Mortality rates differing from the expected ones may have several explanations unrelated to the surgical procedure. A patient with a high risk of complications is not subjected to surgery, making the expected risk of CVD lower during the first years after the operation. This lower risk may persist, as a cohort of patients subjected to surgery could represent a selection of healthy subjects with lower overall morbidity than the population at large. This is not an entirely likely explanation, however, as we know that ulcer patients smoke more frequently than the general population [2, 18, 46, 47, 99, 108], especially those coming to surgery [99], and smoking is a major risk factor for CVD [109]. In our study, patients operated on before the age of 45 years manifested a 75% increase in mortality rate due to CVD during the first 19 years after operation, decreasing to an 18% lower mortality from 20 years postoperatively and onward, whereas in those operated on when over age 45 the CVD-induced mortality seemed lower regardless of time since surgery [36]. Doll and Peto found the correlation between smoking and ischemic heart disease to be strong in young people and to weaken with increasing age [109]. However, coronary sclerosis, which has been found to be especially severe in young and middle-aged men with peptic ulcer [110], probably manifests less often during the preoperative selection phase among young patients.

The finding of long-term lower mortality due to CVD suggests the possibility of some protective effect of the operation. Fat malabsorption is a common finding after partial resection [89, 90, 99, 111], resulting in lower concentrations of serum triglycerides and cholesterol [99, 111]. Dietary and medical intervention trials have shown a reduction in serum lipid levels not only to arrest coronary lesion growth [112] but also to reduce mortality due to ischemic heart disease significantly [113, 114].

In addition there is a favorable pattern of other supposed risk factors for CVD mortality [115, 116] that develops in patients after partial gastric resection: low concentrations of stored iron, low hematocrit concentrations, low blood pressure, and low body weight [2, 42, 99, 111, 117]. Blood pressures have been found to be

lower among operated ulcer patients than in nonoperated ulcer patients [118].

Suicide: Threat to the Ulcer Patient

Excess mortality due to suicide [7, 16, 18, 36, 42, 100–102, 106] and violent death has been a common finding after ulcer surgery, but it is probably related to the personality of the ulcer patient and not to the surgery per se. Viskum studied 2619 ulcer patients in Copenhagen after attempted or committed suicide and found no difference in frequency between operated and nonoperated patients among those committing suicide [119]. A study from Massachusetts reported excess mortality due to suicide among medically treated gastric ulcer patients [107]. A close correlation to alcohol abuse has been found among those committing suicide [120, 121], and in many the abuse started after the operation, which implies a correlation with the surgery [121], though confounding factors are probably more likely.

Long-term Metabolic and Nutritional Effects

Surgical treatment of peptic ulcer may lead to metabolic abnormalities, although the mean energy intake in long-term survivors after partial gastrectomy usually is close to that reported for healthy subjects [2, 118, 122, 123]. Fat malabsorption is a constant finding [89, 90, 99, 111], especially after procedures that bypass the duodenum [124, 125]; but other possible mechanisms of fat malabsorption such, as rapid upper intestinal transit [126] and bacterial overgrowth leading to bile salt deconjugation, may also play a role [127, 128]. Loss of duodenal passage for chyme, leading to fat malabsorption and inadequate uptake of fat-soluble vitamin D, is the most popular theory regarding defective calcium homeostasis leading to a significant reduction in bone mineral density [129–132]. The mechanism of the commonly found iron deficiency is more obscure [42, 99, 117].

There is generally a poor correlation between nutritional status and the observed absorption defects. Weight loss is usually attributed to inadequate intake as a result of postprandial symptoms [89, 122], which are more pronounced after a Roux-en-Y reconstruction [124, 133]. Long-term supplementation with vitamins can be recommended to all patients after partial gastric resection as well as iron supplementation in selected cases.

Total Long-term Mortality in Operated Peptic Ulcer Patients

In summary there are pros and cons for the operated peptic ulcer patient that affect the long-term outcome. Some cohort studies on patients after partial gastrectomy for peptic ulcer have found increased total mortality [7, 36, 42, 102, 134]. The study by Krause comprised patients operated on during the first decades of the twentieth century, and mortality in the population has changed considerably since then. If excess mortality due to tuberculosis is disregarded, total mortality is no longer significantly different from that of the general population [42]. More recent studies indicate an overall unchanged [46, 100] or significantly decreased [101] total mortality. When the cohort in our study was investigated 10 years ago, no increased mortality was found (postoperative deaths excluded) [12], but with longer follow-up the dismal

Table 2. Total mortality in cohorts of patients previously operated on with partial gastric resection or gastroenteroanastomosis and vagotomy due to benign gastroduodenal disease: literature survey.

Study	Year	Country	No. in cohort	Traced (%)	Dead (%)	SMR	Significance	Follow-up (years)
Krause [42]	1958	Sweden	385	94	58	1.29	*	23–50
Cheli et al. [134]	1977	Italy	517	?	32	1.43	*	9–28
Eriksson [12]	1983	Sweden	1575	89	54	1.03	NS	19–49
Fischer [7]	1984	Denmark	1025	95	54	1.18	*	21–29
Inokuchi [100]	1984	Japan	3827	97	28	0.99	NS	11–33
Asano [46]	1987	Japan	6662	93	12	0.91	NS	7–22
Tersmette [102]	1991	Holland	2633	99	67	1.19	*	27–56
Lundegårdh [101]	1994	Sweden	6459	90	59	0.94	*	27–35
Staël von Holstein [36]	1995	Sweden	1575	92	78	1.10	*	29–59

SMR: standardized mortality ratio.

*Risk significantly different from expected ($p < 0.05$); 95% confidence interval, Poisson distribution.

outcome became apparent, especially for those operated on at a young age [36]. Many patients are subjected to surgery at a young age and the supposed adverse effects of partial gastrectomy seem to develop with a latency of at least 20 years. This makes a long follow-up and a high proportion of deceased patients essential in mortality studies. Unfortunately, many published reports on mortality after partial gastrectomy describe cohorts in which most patients have a relatively short follow-up and at least half of the patients are still alive (Table 2).

Ulcer patients coming to surgery are subjected to a dual selection procedure that might affect mortality statistics in various ways. On one hand, it can be assumed that these patients have more advanced ulcer disease associated with excess smoking [99] and a different pattern of social behavior [108]; on the other hand, it is likely that the preoperative workup results in rejection of patients at increased operative risk, especially among those in the older age group.

An increased risk of GI tumors, respiratory diseases, and smoking-related malignancies, as well as excess deaths due to suicide, are common findings at long-term follow-up after peptic ulcer surgery. These hazards to life are, to some extent, offset by the decreased mortality due to CVD. Whether factors related to life style, preoperative selection of patients, or the surgical procedure have the largest impact on the long-term outcome of this group of patients cannot be established with certainty. An increased incidence of and mortality due to stump carcinoma, evident in several reports and with a clear correlation to surgery, is of substantial importance to long-term mortality. Screening procedures leading to early endoscopic diagnosis has been suggested. This strategy, however, has so far not been able to show any benefit in terms of mortality [135, 136]. Attempts to establish preventive measures against suicide and especially tobacco smoking might be more worthwhile for improving the outcome of this cohort.

Résumé

La disparition de la maladie ulcéreuse duodénale et le rapport actuellement bien établi entre l'ulcère et l'*Helicobacter pylori* ont virtuellement aboli le besoin de chirurgie électorale pour ulcère. Cependant, comme une proportion non négligeable de la population aux alentours de l'âge de la retraite a déjà eu une résection gastrique partielle pour ulcère, l'évolution à long terme de ces patients est aujourd'hui un souci bien connu. On aurait pu penser que la population devant avoir une intervention électorale

pour ulcère était une population sans tares, à mortalité moindre, mais les publications actuelles font état de patients opérés pour maladie ulcéreuse avancée, en rapport avec un abus de tabac, et ayant un comportement social particulier. La chirurgie induit un reflux entérogastrique, provoquant de profonds changements dans la muqueuse du moignon gastrique de même que la formation de carcinogènes dans le suc gastrique. En plus, des anomalies métaboliques sont fréquentes, surtout en ce qui concerne la malabsorption des graisses. L'évaluation de l'impact de ces facteurs sur la morbidité et sur la mortalité est cependant difficile. Une mortalité augmentée par tumeurs gastro-intestinales, surtout en ce qui concerne le cancer du moignon, les maladies respiratoires et d'autres maladies en rapport avec le tabac, de même qu'une mortalité excessive par suicide, sont retrouvées lors du suivi au long terme après résection gastrique partielle pour maladie ulcéreuse. Ce risque, cependant, est compensé par une mortalité cardiovasculaire moindre. Des mesures préventives contre le suicide, d'une part, et surtout, le tabac, sont recommandées, pour améliorer l'évolution de la maladie dans cette cohorte.

Resumen

El declinar de la enfermedad ulceroosa del duodeno junto a su relación etipatológica con el "*Helicobacter pylori*" ha abolido virtualmente la cirugía electiva en la úlcera gastroduodenal. Sin embargo, un importante número de la población que ronda la edad de la jubilación, ha sufrido una resección gástrica parcial como tratamiento de su úlcera péptica y los resultados a largo plazo de estos pacientes siguen conservando su actualidad e importancia. Los pacientes gastrectomizados pueden constituir un grupo de sujetos sanos, con una morbilidad global baja, pero diversos estudios han demostrado que también los pacientes tratados quirúrgicamente por úlcera péptica presentan enfermedades más avanzadas relacionadas con el tabaquismo y con una conducta social diferente. En efecto, la gastrectomía aumenta el reflujo enterogástrico lo que no sólo produce profundos cambios en la mucosa del remanente gástrico, sino también, propicia la formación de carcinógenos en el jugo gástrico. Además, las anomalías metabólicas son frecuentes, especialmente la mala absorción grasa. Evaluar el impacto de todos estos factores sobre la morbilidad y mortalidad no es fácil. Los resultados a largo plazo de pacientes gastrectomizados por úlcera péptica, demuestran un incremento en: la mortalidad por

tumores gastrointestinales, sobre todo por el carcinoma del muñón gástrico, enfermedades respiratorias, así como otras patologías malignas debidas al tabaquismo. Se ha encontrado también una excesiva mortalidad por suicidio. Sin embargo, estos riesgos para la vida se compensan por un descenso en la mortalidad por enfermedades cardiovasculares. Se recomiendan medidas contra el suicidio y especialmente contra los fumadores, con objeto de mejorar la calidad de vida a este conjunto de pacientes.

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