Cox multivariate regression analysis for prediction of cardiac death from data at various time points

	B	SE	Wald χ^2 statistic	P value
Baseline				
QTc dispersion	0.021	0.0069	9.40	0.002**
Age	0.080	0.0312	6.61	0.010*
Systolic blood pressure	0.016	0.0080	4.16	0.041*
Sex	0.682	0.5130	1.77	0.183
QT dispersion	0.018	0.0068	7.07	0.008**
Age	0.075	0.0314	5.74	0.017*
Systolic blood pressure	0.016	0.0081	3.96	0.047*
Sex	0.534	0.5016	1.13	0.287
QTc max	0.0166	0.0042	15.45	0.0001**
Age	0.0699	0.0322	4.69	0.0303*
Sex	1.143	0.5269	4.71	0.0300*
Systolic blood pressure	0.0139	0.0077	3.27	0.0707
Year 3				
QTc dispersion	0.017	0.0074	5.15	0.023*
Systolic blood pressure	0.019	0.0118	2.58	0.108
Age	0.046	0.0371	1.51	0.219
Sex	0.569	0.6090	0.87	0.351
QT dispersion	0.018	0.0070	6.46	0.011*
Systolic blood pressure	0.018	0.0115	2.46	0.117
Age	0.045	0.0370	1.48	0.225
Sex	0.539	0.0604	0.80	0.372
QTc max	0.017	0.0054	9.79	0.002**
Sex	0.910	0.6440	2.00	0.157
Age	0.051	0.0380	1.85	0.174
Systolic blood pressure	0.015	0.0117	1.63	0.202
Year 6				
QTc dispersion	0.036	0.0113	10.29	0.001**
Sex	1.667	0.9790	2.90	0.089
Age	0.034	0.0610	0.31	0.575
Systolic blood pressure	0.000	0.0160	0.00	0.986
QT dispersion	0.024	0.0105	5.37	0.020*
Sex	1.219	0.8530	2.04	0.153
Age	0.050	0.0540	0.87	0.351
Systolic blood pressure	0.003	0.0160	0.04	0.838
QTc max	0.035	0.0110	10.36	0.001**
Sex	1.827	0.9210	3.94	0.047*
Age	0.038	0.5400	0.51	0.477
Systolic blood pressure	0.015	0.0190	0.599	0.439

*P<0.05, **P<0.005. Although diuretics were significant in univariate analysis, they were not significant in multivariate analysis.

ratio for microalbuminuria was only 1.8 in a recent overview.[5]

The question arises why analysis of QT interval should be able to predict cardiac death. QT dispersion may be a composite term reflecting electrical inhomogeneity as a result of ischaemia, left ventricular dilatation, left ventricular hypertrophy, cardiac fibrosis, and autonomic neuropathy. Each one of these individually confers increased cardiac risk, and this may be why QT dispersion, as a composite of them, is highly predictive of cardiac death. The clinical value of analysing the QT interval may therefore be that it could be used as a screening test to select diabetic patients for more extensive cardiac investigations. Importantly, the time between measuring a prolonged QT interval and the subsequent cardiac death is many years, which provides ample opportunity to intervene.

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Contributors: AAON analysed all the electrocardiograms and wrote the first draft of the paper. SO performed the statistical analysis. CT, NCD, and FC helped to extract data on each patient. RWN and RTJ ran the United Kingdom prospective diabetes study in Dundee. ADS supervised the project and wrote the final draft of the paper.

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How often does surgery for peptic ulceration eradicate *Helicobacter pylori*? Systematic review of 36 studies

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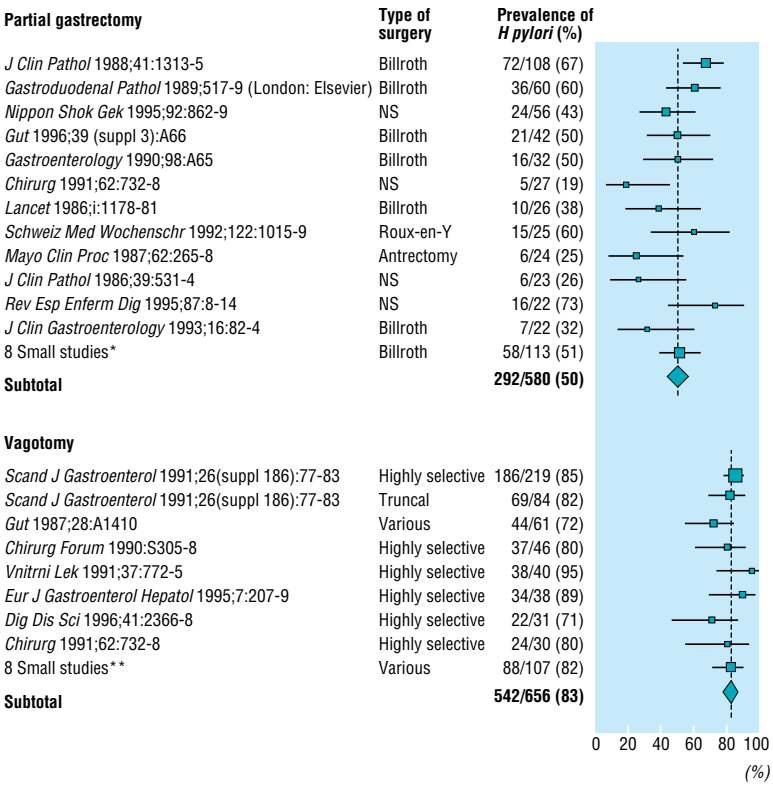
Most peptic ulceration is due to chronic infection with *Helicobacter pylori*, and antibiotic treatments can generally cure both the infection and the ulceration.[1] In previous decades, however, persistent peptic ulceration was often treated surgically either by vagotomy, which merely reduces symptoms, or by partial gastrectomy, which removes the ulcer and parts of the stomach likely to be infected with *H pylori*.[2] There have been several surveys on the prevalence of persistent *H pylori* infection in patients who have undergone surgery for peptic ulceration, often many years previously. We present a systematic review of these surveys and compare the type of surgery with the likelihood of persistent *H pylori* infection.

Methods and results

We checked in databases, reference lists, and gastroenterology journals for any studies published before January 1997 that assessed *H pylori* infection after surgery for peptic ulceration. Studies were included if they provided information on the indication for surgery and the type of surgery. We tabulated the type of surgery, the mean interval between surgery and testing

for *H pylori* (average 10 years), the method of testing for *H pylori* (mostly histology), the site and number of gastric biopsies, and the prevalence of infection. Among the 33 reports identified we excluded five: one included unrepresentative patients (*Hepato Gastroenterol* 1994;41:542-5), and four did not provide separate results for patients with peptic ulceration (*Helicobacter* 1996;1:270; *Z Gastroenterol* 1993;31:115-9) or patients who had had partial gastrectomy (*Surg Gynecol Obstet* 1993;176:594-8; *Mat Med Pol* 1994;88:13-6). From 28 publications, 36 studies were included. Prevalences from different studies were combined by direct summation of their numerators and denominators. The results from the small studies—that is, those with fewer than 20 patients—were combined in the figure and when displaying the results from separate studies and calculating standard χ^2 tests of heterogeneity.

Among patients who had undergone vagotomy alone the prevalence of persistent *H pylori* infection was about 83% (542/656), whereas for partial gastrectomy it was only about 50% (292/580; figure). There were insufficient data to compare the prevalence of *H pylori* infection after particular types of partial gastrectomy—for example, Billroth *v* Roux-en-Y—or vagotomy—for example, highly selective *v* truncal. The heterogeneity within the two subtotals ($\chi^2_{12} = 47$ and $\chi^2_8 = 14$) was much less extreme than the heterogeneity between the two subtotals ($\chi^2_1 = 147$, $P < 0.0001$). Thus the difference in prevalence between the subtotals remained informative.



Prevalence of *Helicobacter pylori* after surgery for peptic ulcer: 36 studies. Size of black area proportional to number of patients. NS = not specified (**Dig Dis Sci* 1991;36:1697; *J Clin Gastroenterol* 1993;16:82-4; *Gastroenterology* 1989;96:A247; *Mat Med Pol* 1994;88:9-12; *Gastroenterology* 1989;97:958-64; *Ann Chir* 1991;45:905-8; *Gut* 1989;30:1552-7; *Pol Arch Med Wewn* 1991;86:13-7. ***Lancet* 1986;1:1178-81; *Gastroenterology* 1989;97:958-64; *Gastroduodenal pathology and Campylobacter pylori* (London: Elsevier) 1989: 517-9, 525-7; *Ann Chir* 1991;45:905-8; *Zentralbl Chir* 1995;120:364-72; *Gastroenterology* 1990;98:A65; *Mat Med Pol* 1994;88:9-12)

Comment

Other studies have shown that most patients with active peptic ulcers are infected with *H pylori*—about 95% of those with duodenal ulcer and 85% of those with gastric ulcer.[3] The prevalence of *H pylori* in such patients remains high after vagotomy (83% (95% confidence interval 78% to 86%)) but falls to about 50% (45% to 56%) after partial gastrectomy. This difference cannot be explained by the methods used for testing for *H pylori* or for gastric tissue sampling as both were similar across studies, or by differences in reinfection rates postoperatively. Despite the inclusion of studies reported as abstracts or in languages other than English some publication bias may remain, although this should not alter the main conclusions. Remission of *H pylori* infection after partial gastrectomy may be due partly to the resection of distal gastric tissue, a usual site of infection, and partly to the bactericidal effects of prolonged bile acid reflux in surgical patients.[4] Whatever the reason, this decrease represents one way surgery could contribute to the cure of peptic ulcer disease.

The main clinical implication of the persistently high prevalence of *H pylori* infection postoperatively is that patients who have undergone gastrectomy or particularly vagotomy should be reviewed and considered for antibiotic treatment that will cure their chronic infection.

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Contributors: JD is guarantor; he also initiated the study, identified and abstracted information from publications, performed statistical analyses, interpreted the data, and drafted the report. PA plotted the findings, discussed statistical issues, and edited the report. RP provided the statistical methods, interpreted the data, and drafted the report.

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Corrections

Birth weight and cognitive function in young adult life: historical cohort study

An authors' error occurred in this paper by Henrik Toft Søreson et al (16 August, pp 401-3). The correct mean (SD) score for parity should have been 0-1: 43.6 (9.4); 2: 42.2 (9.7); ≥3, 41.0 (10.1). These values did not lead to any errors in the risk calculations, and there were no consequences for any of the results.

Childhood energy intake and adult mortality from cancer: the Boyd Orr cohort study

An editorial error occurred in this paper by Frankel et al (14 February, pp 499-504). In table 5 the third cause of death under each of the three main headings (Both sexes, Men, and Women) should have read: Cancers not related to smoking [not Cancers not related to cancer, as published].