Challenges in managing dyspepsia in general practice

Lars Agréus, Nicholas Talley

Department of Family Medicine, Uppsala University, Uppsala, Sweden
Lars Agréus, *research associate*

Department of Medicine, University of Sydney, Nepean Hospital, Penrith, NSW 2751, Australia
Nicholas Talley, *professor of medicine*

Correspondence to: Dr L Agréus, Vårdcentralen, Råghusgatan 6, 740 71 Öregrund, Sweden
lars.agreus@allmmed.uu.se

BMJ 1997;315:1284–8

Although dyspepsia has many possible causes, the primary causes are peptic ulcer disease, gastro-oesophageal reflux disease, and functional (non-ulcer dyspepsia). General practitioners managing dyspepsia face several dilemmas.

- Is a diagnosis of organic dyspepsia reliable without an endoscopy?
- Do patients with dyspepsia need to be referred for endoscopy when nothing of pathological importance will be found in most cases?
- Are the new rapid blood tests for *Helicobacter pylori* helpful?

Dyspepsia subgroups: an irrelevant concept

Broad (and vague) definitions of dyspepsia have been proposed,[1] but a current internationally accepted definition of dyspepsia is chronic or recurrent pain or discomfort centred on the upper abdomen.[2] People with heartburn or acid regurgitation alone and those in whom reflux symptoms are dominant and epigastric pain or discomfort is minor should probably not be classed as having dyspepsia but as having symptomatic gastro-oesophageal reflux disease. The same applies to patients who have a disturbed bowel habit and dyspeptic symptoms, which should be classed as the irritable bowel syndrome.[2]

Dyspepsia subgroups

During the past decade dyspepsia has been divided into clinical subgroups based on medical history: ulcer-like (typical ulcer symptoms), dysmotility-like (symptoms suggestive of gastric stasis), reflux-like (retrosternal and concomitant upper abdominal symptoms), and unspecified (symptoms cannot be classified) (box 1). Findings in the history or examination that are suggestive of serious underlying disease (alarming features) should result in prompt referral for an upper endoscopy or other investigations (box 2). Various management plans for the rational care of patients who have new dyspepsia but without alarming features have been suggested by clinicians and pharmaceutical companies.[1–4] These plans have been based on the dyspepsia subgroups or on individual (predominant) dyspeptic symptoms.

Management controversies

Dyspepsia is treated empirically or by early referral for upper endoscopy, with or without a non-invasive test for *H pylori* infection to guide the management decision. General practitioners are the main target for current recommendations because most patients with dyspepsia are managed in primary care, where the need for a simple, cost effective approach is obvious.

The aim of grouping the symptoms of dyspepsia is to try to match symptoms to pathophysiological disturbances to enable rational prescribing. This has led to recommendations such as acid reduction in reflux-like and ulcer-like dyspepsia and the use of motility stimulants (prokinetic drugs) in dysmotility-like dyspepsia. Such a solution if appropriate would meet everybody's needs: general practitioners could provide prompt treatment, there would be fewer unnecessary endoscopies in the healthcare system, and the pharmaceutical industry could promote the right drug for the right patient.

However, a symptom based strategy may be applied only if most patients with peptic ulcer present with ulcer-like dyspepsia. This has became even more important now that ulcer disease can be cured by eradication of *H pylori* infection, which is present in almost all patients with a duodenal ulcer and in three quarters of those with a gastric ulcer. Antisecretory treatment alone for these patients is no longer routinely justified. Moreover, acid reducing treatment must be of value in those

Summary points

- Subgrouping of symptoms should not guide empirical treatment of dyspepsia
- Symptoms of underlying serious disease and use of non-steroidal anti-inflammatory drugs are indications for prompt endoscopy
- A breath test is superior to a blood test in documenting *H pylori* infection and guiding the management of dyspepsia in general practice
- Empirical treatment with acid reducing or prokinetic drugs for all patients with dyspepsia is inappropriate as peptic ulcer disease will be inadequately treated
- Empirical eradication treatment of *H pylori* in patients with dyspepsia who are positive for the organism is preferable to other empirical treatment if prompt endoscopy is not an option

Box 1: Dyspepsia subgroups[1 2]

- Reflux-like dyspepsia
  - Heartburn plus dyspepsia
  - Acid regurgitation plus dyspepsia
- Ulcer-like dyspepsia
  - Localised epigastric pain
  - Pain when hungry
  - Pain relieved by food
  - Pain relieved by antacids or acid reducing drugs
  - Pain that wakens the patient from sleep
  - Pain with remission and relapses
- Dysmotility-like dyspepsia
  - Upper abdominal discomfort (pain is not a dominant symptom)
  - Early satiety
  - Postprandial fullness
  - Nausea
  - Retching or vomiting
  - Bloating in the upper abdomen (without visible distension)
  - Upper abdominal discomfort often aggravated by food
- Unspecified dyspepsia

who have ulcer-like dyspepsia but do not have a peptic ulcer, which is the majority.[5] Indeed, the risk of having a peptic ulcer must be negligible in the other dyspepsia subgroups if a recommendation to tailor empirical drug treatment to specific dyspepsia subgroups is to be ethically justified.

Ulcer-like and dysmotility-like dyspepsia

Unfortunately for all parties, management strategies based on the dyspepsia subgroups as listed in box 1 fails to stand up to scientific examination. In endoscopy based studies about the same number of peptic ulcers are found regardless of whether the patients present with ulcer-like or dysmotility-like dyspepsia.[5-7] Moreover, only four out of 165 dyspeptic patients who did not have a peptic ulcer at initial endoscopy developed an ulcer over 10 years of follow up, although 62 (58%) of the 106 who still had dyspepsia 10 years later had ulcer-like symptoms.[8]

Population based epidemiological data also fail to support the concept. Most patients with dyspepsia report symptoms that fit more than one of the proposed subgroups, and the change in symptoms from one year to the next is considerable.[9] Prokinetic drugs are also at least as efficacious as H_2 receptor blockers in patients with ulcer-like dyspepsia,[10] especially in those in whom endoscopy confirmed the absence of peptic ulcer disease.[11]

Box 2: Alarming features in dyspepsia

Onset at age >45 years
Weight loss
Anaemia
Gastrointestinal bleeding
Dysphagia
Odynophagia
Persistent vomiting
Epigastric mass
Jaundice
Previous peptic ulcer
Use of non-steroidal anti-inflammatory drug

Division of dyspepsia into subgroups on the basis of the predominant complaint has also been proposed as a predictor of the pathophysiology.[4] This is also not clinically useful: the symptoms identified as typical ulcer-like and typical dysmotility-like in box 1 are common regardless of whether the endoscopic diagnosis is peptic ulcer disease or functional dyspepsia.[12-14] For example, Johannessen et al compared patients with peptic ulcer disease and functional dyspepsia: early satiety was reported by 33% and 29%, nausea by 35% and 46%, a distinct location of pain by 29% and 22%, respectively, and upper abdominal discomfort often aggravated by food by 36% in each group.[13] Kang et al reported that the sensitivity and positive predictive value of localised abdominal pain for duodenal ulcer were only 13% and 14%, respectively, in outpatients.[15] Stanghellini et al found that postprandial fullness was a clinically significant predictor of delayed gastric emptying (a purported mechanism for the induction of dysmotility-like dyspepsia) among younger women, but this has yet to be confirmed in general practice.[16] Bytzer et al have expressed the dilemma about empirical treatment for dyspepsia in another way: at least 50% of patients with a peptic ulcer will be given the wrong empirical treatment because their doctor is not aware of their ulcer when prescribing treatment.[17]

Gastro-oesophageal reflux symptoms

Reflux-like symptoms (box 1), whether considered as a part of the dyspepsia concept or not, are presumed to have a higher sensitivity for reflux oesophagitis,[2] but the symptoms are probably not sensitive enough now that ulcer treatment (brief treatment aimed at cure of *H pylori* infection) has became so different from the treatment of oesophagitis (acid suppression rather than curative treatment).[5 14] In one study Talley et al reported that the prevalence of peptic ulcer was similar among patients with reflux-like (11%), ulcer-like (9%), and dysmotility-like dyspepsia (7%).[5] If endoscopic signs of chronic peptic ulcer disease among those with dysmotility-like dyspepsia were included every 10th patient would have been treated inadequately if endoscopy had been omitted. Hungin similarly reported that 7% of patients presenting with reflux symptoms also had a peptic ulcer,[18] while Carlsson et al found that 20% of patients with a gastric ulcer presented with predominant reflux symptoms.[19] Although proton pump inhibitors may be used as a diagnostic tool to identify and treat gastro-oesophageal reflux disease,[20] such an empirical strategy will fail to treat concomitant peptic ulcer disease in a substantial proportion of patients. There is also a risk of prescribing unneeded and expensive acid reducing drugs long term. The placebo response with acid reducing drugs may be as high as 60%.[21] What many patients in general practice want rather than drug treatment is an explanation for their symptoms.[22] Reassurance and dispelling concerns about serious disease are the most important considerations when planning management.[2 23]

Endoscopy versus empiric treatment

Two well designed studies have shown that prompt endoscopy with treatment based on the results was preferable to blind empirical treatment, although it was initially more expensive.[24 25] They also found a noticeable decrease in drug consumption, visits to doc-

tors, and sick leave in the year after the endoscopy compared with the year before. Patients were more satisfied with their management, although the symptomatic outcome was similar with the two strategies.[24] Concerns about the inadequate correlation between dyspeptic symptoms and pathophysiology is shared by the British Society of Gastroenterology, which has avoided identifying dyspepsia subgroups in its new guidelines on management of dyspepsia.[26]

Non-invasive tests for *H pylori*: problems and opportunities

Accuracy of blood tests

The British Society of Gastroenterology's guidelines recommend that blood tests for *H pylori* should be used to help decide when to perform an endoscopy in younger patients who do not have sinister symptoms and are not taking non-steroidal anti-inflammatory drugs (box 2).[26] Such an approach has also been proposed by others.[27] Blood tests reduce the need for endoscopy by about a quarter because almost all peptic ulcer disease in patients who are not taking non-steroidal anti-inflammatory drugs is due to the infection. Stomach cancer in younger patients is rare but is often associated with the infection.[28] An important question is whether patients will be as reassured by normal results in these indirect peptic ulcer and cancer tests as they are by normal results on endoscopy.[24] A study by Patel et al suggests that patients are reassured by blood rest results,[27] while preliminary results from Lassen et al contradict this conclusion.[29]

The strategy of testing for *H pylori* infection is also dependent on the diagnostic accuracy of the serology kit used. Both rapid whole blood tests that can be done on the spot and enzyme linked immunosorbent assay (ELISA) kits that need laboratory facilities for the analysis are available. The accuracy of the commercial ELISA kits has recently been systematically reviewed.[30] The test cut off points gave an average sensitivity and specificity of 85% and 79%, respectively, with the low and high extremes being 49% and 85% and 99% and 63%, respectively (table). The rapid whole blood tests in preliminary reports seem to have similar sensitivity and specificity. For example, Enroth et al report some of the best results to date, with a sensitivity of 96% and a specificity of 85% (table).[31]

The clinical picture is, however, complex when blood test results are relied on to determine the next step in management. Some authors have recommended empirical eradication of *H pylori* in all cases with positive results on serology,[32] but most recommend that further management should be based on the results of upper endoscopy in those with a positive blood test result.[3 26] Empirical treatment of *H pylori* infection is probably preferable when access to prompt upper endoscopy is limited or when endoscopy is costly. If studies confirm that substantial relief of symptoms occurs after eradication of *H pylori* infection in infected patients with functional dyspepsia,[33] then an empirical eradication strategy will be of even more interest. In addition, the demands on the non-invasive screening test will be greater.

In general practice, patients with a negative result on testing for *H pylori* have to be reassured as they will usually not be retested. The negative test result must consequently be as certain as possible—that is, the ability of the test to identify those not infected (the negative predictive value) is most important. Unlike the sensitivity and specificity, the predictive value is dependent on the prevalence. In the relevant age group (<45 years of age) the prevalence of *H pylori* is estimated to be about 25% in the Western world.[34] A high, almost maximal, negative predictive value is possible with this prevalence and a serology kit that has a sensitivity and specificity of 99% and 63%, respectively (A in table, a negative predictive value of 99%). However, over half (53%) of the patients with a positive test result will be falsely positive and consequently will undergo endoscopy or be treated unnecessarily. Use of a kit with an average or lower sensitivity (B and C in table) does not help: four out of 10 patients with a positive result will still be given a wrong diagnosis, and almost two out of 10 (17%) will have false negative results. Even a better ratio of sensitivity to specificity, 96%:85% respectively (D in table), will result in three patients with false positive results out of 10. A recent study confirms the high rate of false positive results when using blood tests.[35] Moreover, good blood tests may perform poorly in different places in the same country because of variation in strains. Thus, application of a locally validated test is vitally important.[36]

Urea breath test

An alternative non-invasive test is the urea breath test. This test is easy to perform in primary health care. One disadvantage is that the answer is delayed because the breath sample has to be sent away for analysis. A breath test is more expensive (about £30 *v* £15 for a serological test) and to reduce false negative results should not be performed while a patient is taking potent acid reducing drugs.[37] However, the average sensitivity and specificity is 95%,[38] and even higher values have been reported.[39] In patients under 45 this means an almost negligible risk of having infection if the breath test gives negative results (a negative predictive value of 99% in table), giving one out of 10 unnecessary referrals for endoscopy because of a false positive test result (a positive predictive value of 89%). Consequently, because of the higher costs

Diagnostic value of blood and breath tests for *Helicobacter pylori* infection in population with prevalence of infection of 25

Type of test	Sensitivity (%)	Specificity (%)	Predictive value (%)		False positive result (%)	False negative result (%)
			Positive	Negative		
(A) ELISA kit with highest sensitivity[30]	99	63	47	99	53	1
(B) ELISA kit with average sensitivity[30]	85	79	57	94	43	6
(C) ELISA kit with lowest sensitivity[30]	49	85	52	83	48	17
(D) Whole blood kit with good sensitivity to specificity ratio[31]	96	85	69	98	31	2
(E) Breath test[38]	95	96	89	99	11	1

ELISA=enzyme linked immunosorbent assay.

associated with an increased number of endoscopies with blood testing, breath testing may be more cost effective despite its higher initial costs.

Because of the clinical and economic consequences of applying suboptimal blood tests for *H pylori* we question the rationale of using them in general practice, as recommended by the British Society of Gastroenterology.[26] The prompt use of the breath test seems a better option. Indeed, McColl et al found that half of those with dyspepsia and a positive breath test had a peptic ulcer on endoscopy.[40]

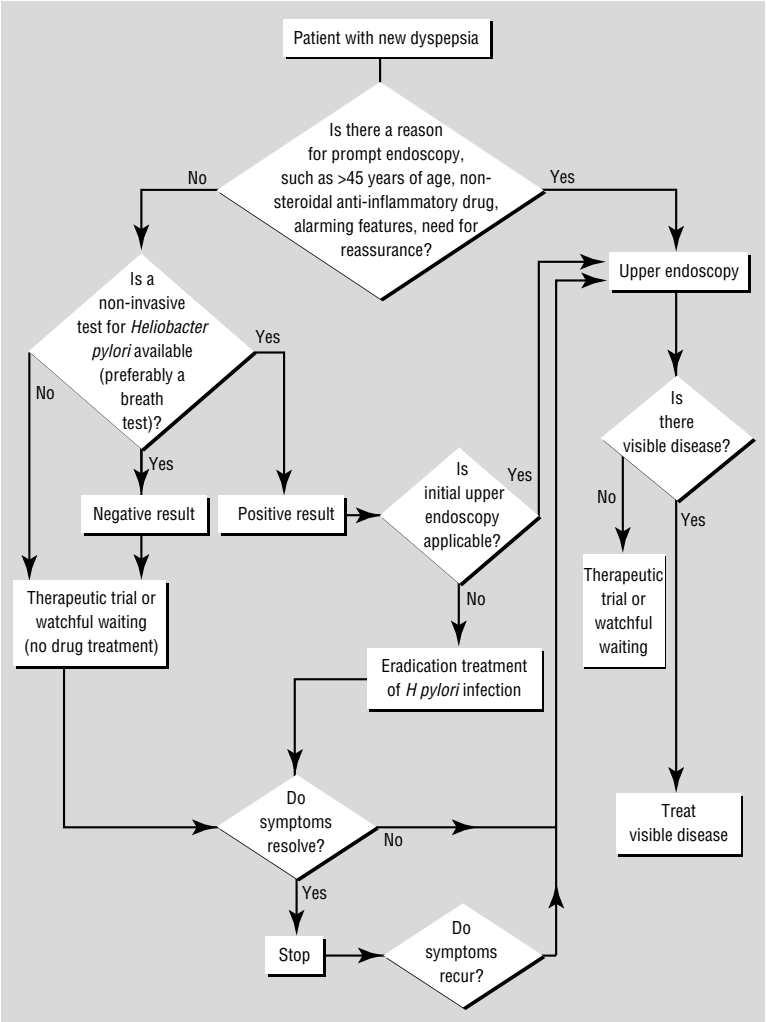
An alternative, but to our knowledge not studied, strategy would be to start with a blood test that has a high negative predictive value. A negative result would then be considered reliable but a positive result would be complemented by a breath test to confirm the presence of infection.

Management strategy for patients with new dyspepsia

We recommend that the management strategy for patients under 45 years of age with dyspepsia of new onset should comprise prompt endoscopy for patients with alarming features, for worried patients or patients demanding long term drug treatment, and for patients taking non-steroidal anti-inflammatory drugs or with a positive result on breath testing. This recommendation will be practical only if the patient is reassured by a negative breath test result and if the waiting lists at endoscopy units are short. Otherwise, a doctor will be obliged to prescribe empirically either acid reducing drugs or eradication treatment for *H pylori*, with the risk of overprescribing or providing inappropriate treatment.

The choice between empirical treatment with acid reducing drugs alone or prokinetic drugs in all patients with undiagnosed dyspepsia remains arbitrary, and all peptic ulcers will be inappropriately managed with such a strategy. Therefore this approach should be abandoned. If it is not applicable to arrange a prompt endoscopy for a patient we recommend empirical eradication treatment of *H pylori* instead, provided that the screening test for *H pylori* is reliable. Most patients with peptic ulcer disease will then be cured, and the total endoscopic load should be reduced since only patients in whom treatment fails or those with a symptom relapse will need to be investigated.

In patients who are negative for *H pylori* and are not satisfied with reassurance alone a trial of an acid reducing or prokinetic drug is reasonable. Our recommendations are summarised in the figure. The algorithm needs to be formally tested in well designed community trials that compare different management strategies.



Algorithm for management of patients with new dyspepsia in general practice

1 Colin-Jones D, Bloom B, Bodemar G, Crean G, Freston J, Gugler R, et al. Management of dyspepsia: report of a working party. *Lancet* 1988;i:576-9.
2 Talley NJ, Colin-Jones D, Koch KJ, Koch M, Nyrén O, Stanghellini V. Functional dyspepsia: a classification with guidelines for diagnoses and management. *Gastroenterology Int* 1991;4:145-60.
3 National Institutes of Health (NIH) Consensus Conference. *Helicobacter pylori* in peptic ulcer disease. *JAMA* 1994;272:65-9.
4 Whittaker MJ, Brun J, Carelli F. Controversy and consensus in the management of upper gastrointestinal disease in primary care. *Int J Clin Pract* 1997;51:239-43.
5 Talley NJ, Weaver AL, Tesmer DL, Zinsmeister AR. Lack of discriminant value of dyspepsia subgroups in patients referred for upper endoscopy. *Gastroenterology* 1993;105:1378-86.
6 Johannessen T, Petersen H, Kristensen P, Kleveland PM, Dybdahl J, Sandvik AK, et al. The intensity and variability of symptoms in dyspepsia. *Scand J Prim Health Care* 1993;11:50-5.
7 Bytzer P, Møller Hansen J, Schaffalitzky de Muckatell O. Symptom grouping in dyspepsia. Any predictive value? *Scand J Gastroenterol* 1993; 28(suppl 197):28.
8 Lindell GH, Celebioglu F, Graffner HO. Non-ulcer dyspepsia in the long-term perspective. *Eur J Gastroenterol Hepatol* 1995;7:829-33.
9 Agréus L, Svärdsudd K, Nyrén O, Tibblin G. Irritable bowel syndrome and dyspepsia in the general population: overlap and lack of stability over time. *Gastroenterology* 1995;109:671-80.
10 Heading RC. Upper gastrointestinal symptoms in general practice: a multicenter UK study. *J Drug Dev Clin Pract* 1995;7:109-17.
11 Hansen JM, Bytzer P, Schaffalitzky de Muckadell OG. Placebo-controlled trial of cisapride and nizatidine in unselected patients with non-ulcer dyspepsia. *Gut* 1994;34 (suppl 4):A9(34).
12 Talley NJ, McNeil D, Piper DW. Discriminant value of dyspeptic symptoms: a study of the clinical presentation of 221 patients with dyspepsia of unknown cause, peptic ulceration, and cholelithiasis. *Gut* 1987;28:40-6.
13 Johannessen T, Petersen H, Kleveland PM, Dybdahl JH, Sandvik AK, Brenna E, et al. The predictive value of history in dyspepsia. *Scand J Gastroenterol* 1990;25:689-97.
14 Adang RP, Ambergen AW, Talmon JL, Hasman A, Vismans JF, Stockbrügger RW. The discriminative value of patient characteristics and dyspeptic symptoms for upper gastrointestinal endoscopic findings: a study on the clinical presentation of 1,147 patients. *Digestion* 1996;57:118-34.
15 Kang JY, Ho KY, Yeoh KG, Guan R. Chronic upper abdominal pain due to duodenal ulcer and other and functional causes: its localization and nocturnal occurrence. *J Gastroenterol Hepatol* 1996;11:515-9.
16 Stanghellini V, Tosetti C, Paternicò A, Barbara G, Morselli-Labate AM, Monetti N, et al. Risk indicators of delayed gastric emptying of solids in patients with functional dyspepsia. *Gastroenterology* 1996;110:1036-42.
17 Bytzer P, Møller Hansen J, Havelund T, Malchow-Møller A, Schaffalitzky de Muckadell O. Predicting endoscopic diagnosis in the dyspeptic patient: the value of clinical judgement. *Eur J Gastroenterol Hepatol* 1996;8:359-63.
18 Hungin P. Open access gastroscopy [thesis]. Newcastle upon Tyne: University of Newcastle upon Tyne, 1996:pp112.
19 Carlsson R, Dent J, Glise H, Riley S, Torfgård K, Junghard O. Evaluation of a questionnaire for the diagnosis of symptomatic gastroesophageal reflux disease (GERD). *Gastroenterology* 1996;110:A76.

20 Fass R, Fennerty MB, Yalam JM, Camargo L, Garewall H, Grade A, et al. Evaluation of the "omeprazole test" in patients with typical symptoms of gastroesophageal reflux disease. *Gastroenterology* 1997;112:A114.
21 Veldhuyzen van Zanten SJ, Cleary C, Talley NJ, Peterson TC, Nyrén O, Bradley LA, et al. Drug treatment of functional dyspepsia: a systematic analysis of trial methodology with recommendations for design of future trials. *Am J Gastroenterol* 1996;91:660-73.
22 Williams S, Weinman J, Dale J, Newman S. Patient expectations: what do primary care patients want from the GP and how far does meeting expectations affect patient satisfaction? *Fam Pract* 1995;12:193-201.
23 Lydeard S, Jones R. Factors affecting the decision to consult with dyspepsia: comparison of consulters and non-consulters. *J R Coll Gen Pract* 1989;39:495-8.
24 Bytzer P, Hansen JM, Schaffalitzky de Muckadell OB. Empirical H_2-blocker therapy or prompt endoscopy in management of dyspepsia. *Lancet* 1994;343:811-6.
25 Hungin AP, Thomas PR, Bramble MG, Corbett WA, Idle N, Contractor BR, et al. What happens to patients following open access gastroscopy? An outcome study from general practice. *Br J Gen Pract* 1994;44:519-21.
26 British Society of Gastroenterology. *Dyspepsia management guidelines*. London: BSG, 1996.
27 Patel P, Khulusi S, Mendall MA, Lloyd R, Jazrawi R, Maxwell JD, et al. Prospective screening of dyspeptic patients by Helicobacter pylori serology. *Lancet* 1995;346:1315-8.
28 Kokkola A, Valle J, Haapiainen R, Sipponen P, Kivilaakso E, Poulakkainen P. Helicobacter pylori infection in young patients with gastric carcinoma. *Scand J Gastroenterol* 1996;31:643-7.
29 Lassen AT, Bytzer P, Schaffalitzky de Muckadel OB. H pylori testing or prompt endoscopy for dyspeptic patients in primary care. A randomized control trial of two management strategies. *Scand J Gastroenterol* 1997;32 (suppl 224):F93.
30 Loy CT, Irwig LM, Katelaris PH, Talley NJ. Do commercial serological kits for Helicobacter pylori infection differ in accuracy? A meta-analysis. *Am J Gastroenterol* 1996;91:1138-44.
31 Enroth H, Rigo R, Hultén K, Engstrand L. BM-test Helicobacter pylori for whole-blood samples: an easy and rapid detection method for H pylori infection. *Gut* 1996;2 (suppl):A117.
32 Malfertheiner P, Megraud F, O'Morian C, Bell D, Bianchi Porro G, Deltenre M, et al. Current European concepts in the management of Helicobacter pylori infection—the Maastricht consensus report. *Eur J Gastroenterol Hepatol* 1997;9:1-2.
33 Gilvarry J, Buckley MJM, Beattie S, Hamilton H, O'Morain CA. Eradication of Helicobacter pylori affects symptoms in non-ulcer dyspepsia. *Scand J Gastroenterol* 1997;32:535-40.
34 Agréus L, Engstrand L, Svärdsudd K, Nyrén O, Tibblin G. Helicobacter pylori seropositivity among Swedish adults with and without abdominal symptoms. A population-based epidemiological study. *Scand J Gastroenterol* 1995;30:752-7.
35 Sinha A, Treharne C, Murray L, Williams DW, Dew MJ. Value of the Helisal test in screening dyspeptic patients for H pylori infection. *Gut* 1997;40 (suppl 1):W7.
36 Mitchell HM, Lee A, Berkowicz J, Borody T. The use of serology to diagnose active Campylobacter pylori infection. *Med J Aust* 1988;149:604-9.
37 Sheu BS, Lin CY, Lin XZ, Shiesh SC, Yang HB, Chen CY. Long-term outcome of triple therapy in Helicobacter pylori-related non-ulcer dyspepsia: a prospective controlled assessment. *Am J Gastroenterol* 1996;91:441-7.
38 Atherton JC, Spiller RC. The urea breath test for Helicobacter pylori. *Gut* 1994;35:723-5.
39 Hamlet AK, Erlandsson KI, Olbe L, Svennerholm AM, Backman VE, Pettersson AB. A simple, rapid, and highly reliable capsule-based C^{14} urea for diagnosis of Helicobacter pylori infection. *Scand J Gastroenterol* 1995;30:1058-63.
40 McColl KE, el-Nujumi A, Murray L, el-Omar E, Gillen D, Dickson A, *et al.* The Helicobacter pylori breath test: a surrogate marker for peptic ulcer disease in dyspeptic patients. *Gut* 1997;40:302-6.

(Accepted 11 August 1997)

A memorable patient

A stitch in time

We met James in our former hospital. He was completely British in manner, speech, and affect. Unfortunately, his British genes included a propensity towards severe angina and occluded coronaries would hasten his demise. He was thin but not all his weight loss was coronary cachexia. He was also HIV positive. He claimed that he had acquired HIV through a transfusion after a car accident, an accident in which his wife was killed.

James accepted his HIV state, but not as readily the coronary disease. He knew his disease processes well, and was often better informed than we were. When angioplasties were performed to the locally allowed limit, he found a doctor 200 miles away who did laser angioplasty. He liked me because I had clerked as a student in cardiology at Hammersmith Hospital. That established my credentials.

His moods were labile. His demands for unproved medications alienated staff. He entertained us with stories of past travel, teaching travel software. But his constant anxiety required high doses of alprazolam and weekly psychotherapy to maintain the equanimity to live amid the ravages of recurrent angina and a CD4 count falling in parallel with his weight.

As debility overtook him, he arrived for a routine appointment and could barely sit on the table. We both sensed the end was in sight. I arranged an admission and asked about contacts. He claimed a daughter in London, whom he did not want informed until the end. "She will just take me back to Britain and I certainly don't want that." He told us to tell the British consul when he died; he had all the details. Should we offer advanced aggressive care? "It depends on the duration and quality of the response."

He deteriorated in the hospital with seizures, pneumonia, and increasing confusion. When evaluated by the psychiatry liaison service, a junior medical student asked him the usual proverbs. Even monkeys fall from trees: "Round pegs don't fit in square holes." A stitch in time saves nine: "Doesn't make sense at all." She learnt what we did not. A former wife did not die in a car wreck but survived; he had no children. A social worker later confirmed that he was gay.

I felt overwhelmed about the false image that he kept up with me for years and called the senior psychiatrist who knew him professionally as well as I did. She confirmed the story that he told me, to almost perfect detail. But with the medical student's information, I went in to see James. He was tremulous and scared and said more weakly than usual, "Good day, Dr S, how are you?" I stood in silence and held his hand, attempting to affirm him in this final moment of truth.

He died 12 hours later, from pneumonia and cardiac compromise. The consulate had only a passport registration. His personal belongings revealed one letter from England. Hospital records listing a next of kin stated "Betty, UK." Inquiries revealed her to be a young niece.

Medicine never stops humbling us. James entered many of our lives for five years. He must have felt compelled to fabricate a story, perhaps to win our approval, a type of "cry for help." In the end, a junior medical student was the confessor he needed to admit the truth. We were sure that he had travelled, that he was British, that he had AIDS, that he was unusually personable, and that in his mind there was something significant about Betty in London. Beyond that, the facts lie with eternity.

Wayne X Shandera, *assistant professor of internal medicine, Houston, Texas*

We welcome articles up to 600 words on topics such as *A memorable patient, A paper that changed my practice, My most unfortunate mistake,* or any other piece conveying instruction, pathos, or humour. If possible the article should be supplied on a disk. Permission is needed from the patient or a relative if an identifiable patient is referred to. We also welcome contributions for "Endpieces," consisting of quotations of up to 80 words (but most are considerably shorter) from any source, ancient or modern, which have appealed to the reader.