frequency of abnormalities on histological examination would mean that few subjects had a normal mucosa.

The clinical value of pathological findings depends on their ability to classify subjects with common features—for example, having symptoms—or to give information on prognosis. The endoscopic findings in our study, with the possible exceptions of peptic ulcer disease and endoscopic duodenitis, showed no association with dyspeptic symptoms that was of clinical value. This corresponds well with the results of therapeutic trials that show a beneficial effect both on symptoms and peptic ulcer disease and endoscopic duodenitis[29 34] whereas convincing evidence of a therapeutic effect on other mucosal inflammations is lacking. In the population the degree of mucosal inflammation apparently represents various degrees in an inflammatory process rather than separate diagnostic entities. Traditionally, clinicians have used as a cut off point between no disease and disease the presence of an ulcer in the mucosa. The ulceration process must, however, have an initial stage at which the ulcer is invisible through the endoscope.[25] The location, distribution, and severity of the inflammation might contribute when considering alternative cut off points. In the paired analysis we found no association between symptoms and isolated inflammation in the corpus, antrum, proximal, or distal duodenum. Location and distribution of the inflammation therefore had poor discriminatory power in this study. Although Toukan *et al* have shown that a high neutrophilic cell count could discriminate between symptom causing gastritis and inflammation not causing dyspepsia,[35] we think that the only rational basis for therapeutic considerations is the symptoms, and not endoscopically or histologically diagnosed inflammation of a mild to moderate degree.

The surprisingly high frequency of positive diagnoses in our study cannot be explained by use of odd provincial diagnostic criteria: both endoscopy and histological examination were performed according to internationally accepted and diagnostic standards of widespread use. When, despite this, the proportion of normal findings on standard endoscopy amounts to a modest 10% it reflects the use of a normative concept of normality, where the norm is the non-prevalent, non-inflamed gastrointestinal mucosa. Our findings challenge both endoscopists and pathologists to search for new distinctions between disease and non-disease.

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Correction

Is risk of Kaposi's sarcoma in AIDS patients in Britain increased if sexual partners came from United States or Africa?

Because of an editorial oversight the authors of this paper (Dr Valerie Beral and others; 16 March, p 624) did not see the abstract before it was published. There were several errors in the printed abstract, and the authors' corrected version is published below.

***Objective*—To determine whether the risk of Kaposi's sarcoma in patients with AIDS is increased by sexual contact with groups from abroad with a high incidence of Kaposi's sarcoma.**

***Design*—Analysis of risk of Kaposi's sarcoma in patients with AIDS, according to country of origin of their sexual partners.**

***Setting*—United Kingdom.**

***Patients*—2830 patients with AIDS reported to the Communicable Disease Surveillance Centre and the Communicable Disease (Scotland) Unit up to March 1990, of whom 566 had Kaposi's sarcoma.**

***Main outcome measures*—Percentage of patients with AIDS who had Kaposi's sarcoma.**

***Results*—537 of 2291 homosexual or bisexual men (23%) with AIDS had Kaposi's sarcoma; 10% (14/135) of the men and women who acquired HIV by heterosexual contact had Kaposi's sarcoma. None of the 316 subjects who acquired HIV through non-sexual routes had Kaposi's sarcoma. Kaposi's sarcoma was more common among homosexual men whose likely source of infection included the United States (171/551, 31%) or Africa (9/34, 26%) than among those infected in the United Kingdom (119/625, 19%) ($p<0.05$).**

***Conclusion*—The data suggest that Kaposi's sarcoma is caused by a sexually transmissible agent which was introduced into the British homosexual population mainly from the United States.**