German homosexual community and may increase exponentially. The incubation period of AIDS infection suggests that the increase will parallel that observed in 1981–82 in the USA[4] but with a time lag of 1½–2 years (figure, B). This assumption is also supported by the observation of clusters in those cities (Munich, Frankfurt, Berlin) where the first AIDS patients with direct contact with the US AIDS epidemic lived.

Cases with unexplained lymphadenopathy, fever, weight loss, malaise, and laboratory-proven dysfunction of cellular immunity, mainly in homosexual males,[5] have also been brought to our attention. The number of such cases in West Germany is estimated to be 5–10 times higher than the number of full-blown AIDS cases. Patients with these symptoms, that have preceded AIDS in some cases,[6] are again most frequently seen in these cities.

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HAEMOPHILIA AND AIDS IN THE UK

SIR,—In their otherwise clear account of a fatal case of acquired immunodeficiency syndrome (AIDS) in a haemophiliac in the UK `r Daly and Dr Scott (Nov 19, p 1190), referring to our account of .IDS surveillance,[7] state that: "No definite case of AIDS in a haemophiliac has yet been reported in Britain although one patient may have early features of the syndrome". The Communicable Disease Surveillance Centre (CDSC) does indeed collect data on patients who may have early features of AIDS, but our paper[1] included only those cases which met the definition of AIDS compiled by the Centers for Disease Control, Atlanta, on March 15, 1983. The information kindly provided to us about the haemophiliac we mentioned led us to include him as a definite case of AIDS.

May we thank all clinicians and microbiologists who have reported suspected cases to CDSC and to appeal to all doctors to report such cases. We have received 35 reports, 26 of which fit the Centers for Disease Control's definition of the syndrome.

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LOCAL RECURRENCE AND RECTAL CANCER

SIR,—We agree with Mr Umpleby and colleagues (Oct 29, p 1020) that, besides residual lymphatic and venous tumour, implantation of exfoliated cancer cells may be one factor causing rectal anastomotic recurrence. It is curious, however, that some recurrences happen in early (Dukes grade A) tumours or in tumours with an apparently adequate margin of resection.[8,9] Our experience with chemically induced colonic carcinomas in the rat has shown that the anastomosis itself is highly susceptible to tumour formation and more susceptible than the rest of the bowel.[10]

Moreover, we have observed tumours at or close to unabsorbed suture material. Transitional mucosa, adjoining or away from colonic tumours, is demonstrable by histochemical techniques.[11] Given the right stimulus this mocosa may undergo neoplastic change. We are now testing the idea that suture material may be one of the promoting factors in this mucosal transformation. If so, the choice of suture material and staples for rectal anastomosis may be a factor in local anastomotic recurrence.

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H_2-BLOCKERS: NIGHT-TIME ONLY

SIR,—Dr Dammann and colleagues (Nov 5, p 1078), report their experience of 24 h intragastric acidity with single night-time doses of three H_2-receptor antagonists, but do not refer to our abstract published in May.[1] Dammann and colleagues' conclusion that a single, large, night-time dose of cimetidine or ranitidine is at least as effective as a twice daily regimen cannot be drawn from the data presented, since twice daily and nocturnal dose regimens were not directly compared. Nor did they study overnight acid output, so they cannot claim effects on nocturnal acid secretion. Their comparison with our previous work[2] is invalid because different populations were studied.

Our studies[1,3] of 24 h intragastric acidity and nocturnal acid output in twelve duodenal ulcer patients receiving randomised treatments of placebo, cimetidine 400 mg twice daily, cimetidine 800 mg at night, ranitidine 150 mg twice daily, or ranitidine 300 mg at night showed that "a single night time dose of an H_2-receptor antagonist is as effective as a twice daily regimen of these drugs in decreasing hydrogen ion activity and acid output". And we suggested that a single night-time dose be evaluated by clinical trial.

The formula for famotidine, the third H_2-receptor blocker studied by Dammann et al, is 3-([(2-[-(diaminomethylene) amino]-4-thiazolyl)-methyl] thio)-N-sulfamoylpropionamidine.

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PREDNISONE AND METHYLPREDNISOLONE DISPOSITION IN THE LUNG

SIR,—Dr Braude and Dr Rebuck (Oct 29, p 995) have introduced a novel approach to the management of lung disease with systemic corticosteroids. However, prednisone is converted to its biologically active metabolite prednisolone in the liver. If both prednisolone and prednisone had been assayed the results may well have been different. A comparison of the results for two drugs administered by different routes and in non-equivalent doses must be interpreted with caution, especially when the plasma availability of a drug may not be reflected by just one plasma level.[4] The use of 20 ml volumes of saline may preferentially sample airways rather than peripheral lung,[5] so the lung surface may not always have been consistently washed out. Furthermore, Braude and Rebuck do not give technical details, such as lavage recovery rates; nor do they explain why creatinine rather than albumin was used to standardise drug recovery.

Whilst welcoming the idea of studying drug availability in the lung, we believe that Braude and Rebuck's results should be interpreted with extreme caution.

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