of cytokines such as transforming growth factor β1 is likely to be an epiphenomenon of the inflammatory process.

Finally, we support the view of Cavallini et al that while calcification might contribute to the pathogenesis of chronic pancreatitis in its later stages, there is no well documented evidence fulfilling Koch's postulates that it is probably a significant aetiological factor in the disease. Hence, we also cannot support the hypothesis of the Marseilles school.7

We consider that the disease known as 'chronic pancreatitis' is not a single pathological entity but rather a group of different aetiologies and pathogenetic processes sharing a few common morphological endpoints.3 Within this overall group, we anticipate that a precise and meaningful definition of the genetic abnormality is the primary likely aetiopathological factor responsible for at least a proportion of cases of chronic pancreatitis. Whether this defect occurs within the pathway of alcohol metabolism or is responsible for promoting an inappropriate cell mediated cytotoxic response to some pancreatic cellular antigen is presently unknown. Nevertheless, systematic studies are vital if biologically appropriate treatment regimens for different aetiologically and pathogenetically distinct types of 'chronic pancreatitis' are to be developed and affected patients treated more rationally than at present.

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Diagnosis of invasive amoebiasis: renaissance of the morphology era

EDITOR,—In his leading article entitled Diagnosis of invasive amoebiasis: time to end the morphology era (Gut 1994; 35: 1018–21), Professor Ravdin rightly emphasises that microscopy cannot differentiate between pathogenic and non-pathogenic strains of Entamoeba histolytica. This applies equally to the cysts, and cultured trophozoites. Professor Ravdin goes on to suggest that microscopy should be abandoned in the diagnosis of invasive amoebiasis. He has underestimated the diagnostic value of one of the most important diagnostic points described by Losch in 1875.1 In clinical specimens from patients with amoebic dysentery trophozoites of E histolytica may be seen with ingested red cells. These erythroagphagocytic trophozoites are a specific feature of infection with invasive strains of E histolytica and may be seen on microscopy of fresh faecal specimens or in fixed smears stained with Field's stain. The correct recognition of this finding in amoebiasis are microscopical examination of fresh stools and stained fixed faecal smears, and amoebic cultures of stool specimens.3 A study4 of patients with dysentery, diarrhoea, and asymptomatic carriage of pathogenic and non-pathogenic E histolytica confirms that microscopy is a highly efficient diagnostic method for amoebic dysentery. The sensitivity and specificity of erythroagphagocytic trophozoites were 96% and 100%, respectively, when compared with amoebic culture and subsequent zymodeyme typing of the isolated strains to confirm pathogenicity. We agree with Professor Ravdin that there is a need for diagnostic methods that do not depend on the detection of intact parasites. Tests that detect faecal or circulating amoebic antigen, or both, are required. To validate such assays they must be compared with a gold standard. With the current state of knowledge the standard should include the finding of erythroagphagocytic trophozoites. We do not agree that currently available antigen detection tests have supplanted microscopy in the diagnosis of amoebiasis. Many things have changed since 1875 but the findings of erythroagphagocytic amoebic trophozoites in stool smears and cultures remains the simplest, cheapest, and most reliable test in the diagnosis of amoebic dysentery. Welcome to the morphology era!

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Reply

EDITOR,—González-Ruiz et al have shown that in expert hands the detection of haematrophagous trophozoites has a high predictive value in diagnosing infection by pathogenic Entamoeba histolytica strains.1 This study is very helpful and reaffirms the value of microscopy of faecal samples when it is readily available and highly skilled. Unfortunately, in many areas of the world the results are not readily available and are not accessible. In addition, clinical laboratories in community settings may vastly overdiagnose amoebiasis and report leucocytes in stool as E histolytica trophozoites. I suggest that in the future microscopy will not be needed as serological studies for anti-amoebic antibodies and studies of antigen and DNA detection will be sufficiently developed and field tested.3 The gold standard for comparison of these new methods is stool culture and zymoideyme determination, not microscopy for erythroagphagocytic trophozoites as suggested by González-Ruiz and Bendall. Simple agglutination tests, based upon current enzyme linked immunoabsorbent assay and hybridisation technology, will evaluate for infection by multiple enteric parasites. This will be the most cost effective, sensitive, and specific methodology available for diagnosis of infections by E dispar and E histolytica. Currently, we should use practical criteria as suggested by González-Ruiz and Bendall, but in the 21st century, let us advance beyond the technology of the 19th century.

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Relation of acupuncture and vagal gastric acid secretion

EDITOR,—We read with interest the paper of Lux et al (Gut 1994; 35: 1026–9), which examines the effects of various forms of acupuncture on sham feeding and stimulated acid output in healthy volunteers. These results are entirely in accord with our own findings, previously published both in abstract form (the first of which was in this journal six years ago) and in a peer reviewed journal that acupuncture produces a significant decrease in sham feeding stimulated acid output under randomised, placebo controlled conditions in humans.

In those studies we also showed that the effects of acupuncture decreased sham feeding stimulated acid output was through naloxone sensitive opioid mechanisms, involving vagal efferent pathways. Furthermore, acupuncture produced neither a decrease in gastrin release nor a diminished parietal cell sensitivity to gastrin. While we agree with Lux et al that the mechanism through which acupuncture exerts its effect is not fully elucidated it seems to be at least in part related to opioid pathways, which may be similar to the mechanisms participating in the analgesic properties of acupuncture.

In their report, Lux et al cite another of our publications as concluding that acupuncture accelerates peptic ulcer healing.1 This is incorrect; the cited study was conducted in healthy volunteers to examine the effects of acupuncture on sham feeding stimulated acid output. In our comprehensive review of all the