of cytokines such as transforming growth factor β_1 is likely to be an epiphenonemon 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.[2]

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 end points.[3] Within this overall group, we anticipate that a predisposition based upon an identifiable genetic abnormality is the likely primary aetiological 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, answers to such questions 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.

C S FOSTER
Department of Pathology, University of Liverpool, Liverpool L69 3BX

S D SLATER
Department of Histopathology, Royal Postgraduate Medical School, Du Cane Road, London W12 0NN

R C N WILLIAMSON
Department of Surgery, Royal Postgraduate Medical School, Du Cane Road, London W12 0NN

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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 important point, first 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 erythrophagocytic 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 current recommendations for the diagnosis of amoebiasis are microscopical examination of fresh stools and stained fixed faecal smears, and amoebic cultures of stool specimens.[2 3] A study[4] 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 erythrophagocytic trophozoites, were 96% and 100% respectively, when compared with amoebic culture and subsequent zymodeme 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, would be suitable for this purpose. 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 erythrophagocytic 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 erythrophagocytic amoebic trophozoites in stool specimens remains the simplest, cheapest, and most reliable test in the diagnosis of amoebic dysentery. Welcome to the morphology era!

A GONZÁLEZ-RUIZ
Department of Medical Microbiology, London School of Hygiene and Tropical Medicine, Keppel Street, London WC1E 7HT

R P BENDALL
Department of Clinical Parasitology, Hospital for Tropical Diseases, 4 St Pancras Way London NW1 0PE

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Reply

EDITOR,—González-Ruiz *et al* have shown that in expert hands the detection of haematophagous 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 complete laboratory facilities are not readily accessible. In addition, clinical laboratories in community settings may vastly overdiagnose amoebiasis and report leucocytes in stool as *E histolytica*.[2] Lastly, stool culture for *E histolytica* has repeatedly shown that microscopy has a low sensitivity.[3 4]

Clearly, there is a consensus that better, quantitative methodology is needed that ideally can be brought directly to the field. I suggested 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.[5] The gold standard for comparison of these new methods is stool culture and zymodeme determination, not stool microscopy for erythrophagocytic trophozoites as suggested by González-Ruiz and Bendall. Simple agglutination tests, based upon current enzyme linked immunosorbent 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.

J L RAVDIN
Department of Medicine, Case Western Reserve University School of Medicine, and the Cleveland Veterans Affairs Medical Center, Cleveland, Ohio, USA

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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 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!)[1 2] and in a peer reviewed journal,[3] 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 through 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