Our results show that the occurrence rate of *C pylori* in various gastric diseases in China and western countries are similar.[1-3] Our results confirm that there is a close correlation between *C pylori* and peptic ulcer as well as active chronic gastritis. But this relation was not found in gastric carcinoma and other gastric diseases.

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THROMBOLYTIC THERAPY AND PERCUTANEOUS CORONARY ANGIOPLASTY

SIR,—Immediate angiography and percutaneous transluminal coronary angioplasty (PTCA) in patients with acute myocardial infarction requires 24-hour availability of facilities for coronary angiography. The cost of this procedure, including personnel, equipment, and disposables (PTCA catheters), has been estimated at £3600,[1] which is three times as high as the cost of intravenous administration of recombinant tissue-type plasminogen activator (rTPA). Our trial (Jan 30, p 197), which Dr Timmis criticises (March 5, p 531), was designed to verify whether the effort and cost of immediate angiography and PTCA would be worthwhile. Contrary to expectations, immediate angiography and PTCA had no benefit over treatment with intravenous rTPA without angiography.

There was a slight imbalance in baseline characteristics, with more patients who were admitted with severe heart failure or shock being allocated to the invasive strategy, as noted by Timmis. However, this does not account for the difference in mortality between the two groups, as is evident from our table III. 4 patients with severe heart failure or shock allocated to the invasive strategy died within two weeks. If these patients are excluded from analysis, 14-day mortality is 5 patients (2·7%) in the non-invasive group compared with 8 patients (4·4%) in the invasive group. This difference, although not significant, does not suggest that immediate angiography and PTCA would be beneficial.

The potential beneficial effects of immediate PTCA were offset by a high rate of early reocclusion and/or early recurrent ischaemia. Infarct size, estimated from myocardial α-hydroxybutyrate dehydrogenase release, was not smaller in the invasive group (actually it was 6% larger). In patients who survived and underwent left ventricular angiography between days 10 and 22 there was no evidence of better left ventricular function than in those patients who underwent immediate PTCA.

Neither the clinical course, measurements of infarct size and ventricular function, nor survival suggested that immediate PTCA would be beneficial. This would not have been altered if the study had continued until 400 patients had been included. Actually 367 patients were included in the trial, which was only 33 less than originally designed. The data monitoring and ethical committee, and the steering committee of the trial considered it unethical to continue patient enrolment since it was evident that PTCA did not offer any benefits to the patients and might even be harmful. It would be unwise to spend further effort and funds on the additional procedure without benefit to the patient.

The mortality and complications in patients treated with rTPA without immediate angiography were low (3% mortality and 10% reinfarction at 3 months). Mortality was lower than in any other reported trial with thrombolytic therapy. For example, the preliminary data of the AIMS study of APSAC (March 12, p 545) show a 6·4% mortality at 30 days despite exclusion of patients with cardiogenic shock and inclusion of patients with less severe ST-segment elevation than in the rTPA trial. In view of the low mortality and reinfarction rates observed after rTPA, it is uncertain whether systematic coronary bypass surgery in patients with three-vessel disease, as suggested by Timmis, would further improve outcome. To reveal or rule out further improvement a new trial would be required with a different design from ours.

The present recommendations for optimum therapy of patients with acute myocardial infarction, based on our results, studies from the USA and Australia, and the AIMS study are: (1) immediate intravenous treatment with rTPA, or possibly APSAC, with acetylsalicylic acid and heparin to prevent reocclusion, and (2) coronary angiography followed by PTCA or bypass surgery in about a quarter of the patients with early post-infarction angina or who have myocardial ischaemia during the pre-discharge exercise test.

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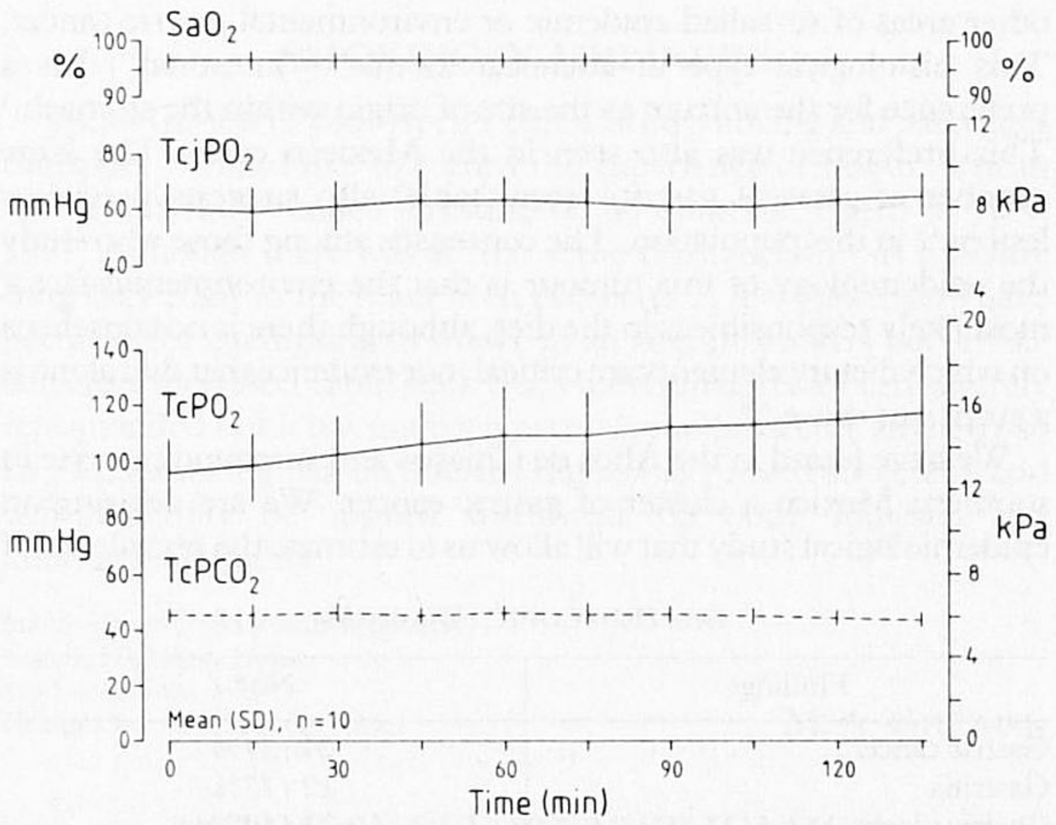
WHY DOES PLACEBO IMPROVE SEVERE LIMB ISCHAEMIA?

SIR,—Dr Vayssairat and colleagues (Feb 13, p 356) report that placebo may improve severe limb ischaemia, as demonstrated by a rise in transcutaneous oxygen tension ($TcPO_2$) between the beginning and end of a three hour observation period (from a mean of 32 (SD 12) to 36 (15) mm Hg, a 12·5% increase). No data on the calibration procedure, operating temperature, or the in-vitro and in-vivo drift of the electrode were provided. Our observations on in-vivo drift suggest that the reported increase in $TcPO_2$ is due to changes in the skin-electrode interface caused by the heating element of the transcutaneous polarographic electrode. In other words, placebo does not improve severe limb ischaemia.

We did a two-point dry gas calibration at 45°C on three commercially available, transcutaneous, combined oxygen and carbon dioxide electrodes, and applied the electrodes to the arms of normal adults breathing air.[1] After equilibration or 30 min, we compared transcutaneous values with end-tidal gas values measured by mass spectrometer. Although no significant changes occurred in either $TcPCO_2$ or end-tidal values, the $TcPO_2$ signal drifted upwards by 16–21% of baseline values after 140 min continuous recording. These observations could not be explained by in-vitro calibration shifts of the electrodes.

We also measured the in-vivo drift in adults of a pulse oximeter, a transcutaneous dual electrode, and a transconjunctival oxygen electrode,[2] which measures oxygen tension at the inner surface of



In-vivo drift of $TcPO_2$.

Note upward drift in $TcPO_2$ but not $TcPCO_2$ in normal adults breathing air, despite stability of $TcjPO_2$ and arterial oxygen saturation (SaO_2) measured by pulse oximeter. A 30 min equilibration preceded measurements.