studies suggest a substantial benefit from thrombolysis in patients with AMI. Why should a group who happen to infarct with non-diagnostic ECGs be different? Dr Timmis aims to treat the ECG—we aim to treat the infarct.

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SIR,—Dr Downie and colleagues' data are especially relevant for old people, in whom early accurate diagnosis presents special difficulties. Atypical presentations rise with increasing age and most infarcts occur in elderly people. Missed therapeutic opportunities are therefore a major difficulty. In your accompanying commentary, Dr Gwilt says that 80% of all AMIs can be diagnosed at the time of admission from "careful assessment of the history and the ECG". This statement is unlikely to be true for older people. In the Framingham cohort about 40% of all infarctions in people over 75 years were estimated to have been unrecognised as such.[1] Similarly, in series of necropsy-proven myocardial infarction there was poorer clinical and pathological agreement in older age groups.[2] In addition, diagnostic delays in clinically recognised infarcts are much longer in elderly people.[3] Changing symptomatology and the presence of coexisting diseases in part underlie delays or failure to diagnose.[4] The frequency of chest pain as the presenting symptom of AMI ranges between 20% and 80%.[5-7] Acute onset dyspnoea may be more common than chest pain in the very elderly, and unexplained syncope, falls, and acute confusion also increase in frequency;[6] diagnostic ECG changes are also less common[3] whereas rises in cardiac enzymes may be more reliable.[3,7] Therefore, the development of sensitive and specific tests to improve early diagnostic accuracy would be especially useful in older patients in whom the greatest impact could be made. The method Downie et al describe is a step in the right direction. It would be useful to know the ages of their patients. Specificity of total creatine phosphokinase might not be as high in older people when rises may be attributable to somatic injury after a fall during the course of an acute illness. Finally, although a level of diagnostic certainty is required before thrombolytic therapy is administered, an argument could be made always to give aspirin even in the face of diagnostic uncertainty (providing there are no specific contraindications), since the benefits are equal (and additive) to those of thrombolytic therapy.[8]

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SIR,—Dr Downie and colleagues achieve correct diagnosis and treatment with thrombolytic therapy in 17 of 29 cases of confirmed AMI. In our hospitals we have used a strategy involving emergency measurement of serum creatine kinase MB concentration by immunoassay in the biochemistry laboratory to increase the number of patients with AMI treated with a thrombolytic.[1] The assay itself takes 10 min to do and we judged that it was not suitable for bedside use by nursing staff in a busy district general hospital on a routine basis. However, audit of our system showed that the median time taken from the doctor requesting the analysis to receiving the report was 34 min. This does not seem to result in a greater delay than the 2·4 h from admission to treatment cited by Downie et al. Furthermore, in our hands and those of others, the diagnostic accuracy of creatine kinase MB concentration is better than that of total creatine kinase activity.[2-4]

Our protocol used measurement of serum creatine kinase MB on admission in patients in whom there was reasonable suspicion of AMI, but the clinical and ECG evidence was insufficient to justify thrombolytic treatment. This test was followed by a repeat test 2 h later if the first result was low and the diagnosis remained in doubt. In 228 patients investigated with this strategy, 79 had a discharge diagnosis of AMI, of which 74 were identified by a high creatine kinase MB and 69 treated with a thrombolytic. The diagnostic sensitivity and specificity of our strategy were both 94%, and in 22 cases (32%) the diagnostic blood sample was taken within 6 h of onset of symptoms, unlike the 1 of 17 (6%) in Downie's study.

Although measurement of serum creatine kinase MB concentration in the laboratory is more expensive than measurement of total CK activity at the bedside, the difference is marginal, and small compared with the cost of thrombolytic drugs.[1]

We now know that it is possible to identify with emergency enzyme analyses most patients with equivocal ECG on admission as having AMI, but as Downie et al rightly point out, we do not know whether thrombolytic treatment is beneficial in this group. International studies such as ISIS-3[5] should contain data on the mortality encountered in thrombolysis versus placebo in the subgroup of patients who have non-diagnostic ECG on admission, but a diagnosis of confirmed AMI on discharge. As far as we are aware analysis of this particular subgroup has not been published.

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Handedness and autoimmune disease

SIR,—Your Jan 2 commentary by Dr Skrabanek, who claims that Crohn's disease is more common in left-handers than right-handers, is criticised by Professor Janowitz (Feb 27, p 565) on the basis of his study[1] that showed that the incidence of left-handedness was much the same in cases and controls. Janowitz concludes by hoping "that there will be no further resurrection of this false linkage" of left-handedness and Crohn's disease and ulcerative colitis.

That hope is probably premature. We have completed a meta-analysis (Bryden MP, McManus IC, Bulman-Fleming MB, unpublished) of studies of a range of putatively autoimmune disorders that the Geschwind-Behan-Galaburda model[2] has suggested are linked with left-handedness. 21 837 cases and 34 457 controls were examined, aggregated across fourteen separate disease categories. Overall there was a small tendency for left-handers to show a higher incidence of autoimmune disorders (odds ratio 1·062, 95% CI 1·003–1·126) with a highly significant heterogeneity ($p < 0·0025$) between conditions. In the three published studies that have considered ulcerative colitis/Crohn's disease[1,3,4] (and excluding the original report of Geschwind and Behan[5]), left-handers were 2·007 (95% CI 1·350–2·893) times more likely to have inflammatory bowel disease, with no significant heterogeneity between studies. In addition, left-handers showed a higher frequency of allergies (odds ratio 1·128, 95% CI 1·046–1·218) and

asthma (1·184, 1·004–1·351), and a lower incidence of arthritis (0·745, 0·594–0·932) and myasthenia gravis (0·267, 0·099–0·722). Such results are not compatible with Geschwind, Behan, and Galaburda's theory.[2] However, in the event of further replication they undoubtedly need explanation. One possibility is that left-handers and right-handers differ in their HLA haplotypes,[6] thereby resulting in different patterns of disease.

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Adverse effects from traditional Chinese medicine

SIR,—Dr Tomlinson and colleagues (Feb 6, p 370) point out that there were more cases of aconite poisoning in Hong Kong than we reported (Nov 21, p 1254). Our study population comprised 7 cases of herb-induced aconite poisoning that were prospectively documented over 4 months, and 10 others retrospectively identified. Recruitment required establishment beyond reasonable doubt that aconites from aconitum rootstocks were the only plausible cause for intoxication. We are aware that the problem of herb-induced aconite poisoning is likely to be great in Chinese communities worldwide. In China alone there were over 600 reported cases in the past 30 years,[1] which prompted the introduction in 1984 of legal regulations on aconites and other potent herbs. The true incidence of aconite poisoning in Hong Kong might be higher; indeed, since our report there have been another 5 well-documented cases. However, identification of such cases requires clinicians to be aware of the dangers of aconites. In none of our retrospectively identified cases was the diagnosis of aconite intoxication suspected while the patient was in hospital, despite the availability in every case of herbal prescription forms that clearly included aconitum rootstocks. However, we caution against overdiagnosis in the absence of sufficient evidence, and agree with Dr But (March 6, p 637) that proper identification of herbs is essential.

The current lack of legal control over herbal practice in Hong Kong dates back to the Nanking Treaty of 1842, in which Chinese medicine was protected from control as part of "Chinese customs and usages". In 1989, a government-appointed working party on Chinese medicine was established to review its status and to identify areas for improvement. Since the report of a mini-epidemic of life-threatening aconite poisoning by our group in October, 1991, local education measures have been instituted to alert the medical community and the public.[2] Initiatives were also taken by the Chinese Medicinal Material Research Centre to inform and work with the herbal industry, and associations of practitioners of traditional Chinese medicine, to find measures to minimise further incidents of aconite poisoning. An interim report of the working party[3] in January, 1992, identified the following issues as high priority: education in the proper use of traditional Chinese medicine, the drawing up of a "potent herbs" list to facilitate control, and the introduction of registration and regulation of practitioners of traditional Chinese medicine. These suggestions might be applicable in many other parts of the world.

The report by Professor Vanherweghem and colleagues (Feb 13, p 387) of nephrotoxicity associated with a slimming regimen that included Western medicines and herbal ingredients from Chinese and European plants, confirms the need for appropriate use of medicines. The prescription of herbal medicines requires as much vigilance and caution as that of Western medicines. In the cases reported by Vanherweghem and colleagues, the herbs were not used according to established principles and cannot be accepted as Chinese medicine. As pointed out by Professor Atherton and colleagues (March 6, p 637), the cases reported in Belgium raise the additional issue of the risk of adverse interactions between herbs and modern medicine. In China, there is much research into the possibility of integration of traditional Chinese and modern medicine involving careful experimental and clinical design, and systematic observation and monitoring of efficacies, interactions, and side-effects. The Belgian cocktail is not an integration of traditional and modern medicine, but a confusion of the two.

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SIR,—Professor Vanherweghem and colleagues report rapidly progressive interstitial renal fibrosis in 9 young women who had followed a slimming regimen including Chinese herbs. We studied 6 women with renal failure secondary to this regimen (2 of whom are included in the report by Vanherweghem and colleagues). We describe the unusual presentation of 1 of these women with bilateral ureterohydronephrosis secondary to extensive periureteral fibrosis.

This 28-year-old woman had two uneventful pregnancies in 1988 and 1990. She was referred to us in October, 1991, during the 12th week of her third pregnancy because she had sterile microscopic haematuria and leucocyturia of recent onset. Ultrasonography disclosed major bilateral ureterohydronephrosis extending to the pelvis. Cystoscopy and magnetic resonance imaging were unhelpful. Her serum creatinine rose from 101 to 176 μmol/L at delivery, while her haemoglobin fell from 9·6 to 6·9 g/dL, necessitating transfusion. After delivery of a normal boy in April, 1992, her ureterohydronephrosis did not regress and her renal function further deteriorated. Bilateral retrograde ureteropyelography showed a severe narrowing of both distal ureters, the cause of which remained elusive despite intravenous pyelography, magnetic resonance imaging, and laparoscopy. Despite the successful insertion of ureteral catheters, her renal function continued to deteriorate. We searched, therefore, for a cause of primary renal disease and learned in August, 1992, that the patient had followed the slimming regimen of clinic X described by Vanherweghem and colleagues. After her first pregnancy, from December, 1988, to June, 1989, she took the apparently harmless formula 1 preparation, and after her second pregnancy, from July 1990, to August, 1991, she took the apparently toxic formula 2. Haemodialysis was started in January, 1993, and the ureteral catheters were removed. As part of her pretransplantation programme, a left nephroureterectomy was performed. Macroscopically, major thickening of the pelvis and ureter and a homogeneously shrunken end-stage kidney were observed. Microscopically, intense sclerosis of the renal cortex without primary glomerular or vascular lesions was observed. In addition, the pelvis and the ureter were embedded in a 0·5 to 1 cm thick sheath of dense fibrotic tissue.

This observation suggests that the Chinese herbs incriminated in the reports of Vanherweghem and colleagues induce a generalised fibrotic process. Not only do these herbs cause renal sclerosis, but they may also stimulate extensive fibrosis around both ureters. As in