patients had severely restricted elbow movements and, therefore, had repeat radiographs taken. One patient had an undisplaced fracture of the radial head and the other an undisplaced fracture of the radial neck. Treatment was unaltered by these findings, but full recovery was delayed to 28 and 35 days, respectively.

We therefore agree that the recognition and review of elbow effusions is important but believe that the decision to obtain repeat radiographs should be made on clinical grounds.

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Compartment syndromes in unconscious patients

SIR,—Mr A C Macey (6 June, p 1472) highlights a not uncommon problem seen in many surgical specialties, in particular orthopaedic, vascular, and plastic surgery. We would support the view that not only in uncooperative and unreliable patients should the compartment pressures be measured but also routinely in any suspect limb. We believe, however, that Mr Macey has ignored or misinterpreted several important points.

It is now widely accepted that the lower leg consists of four discrete compartments: anterior, lateral (peroneal), deep posterior, and superficial posterior. In our experience,[1] and according to Seddon,[2] it is most often the deep posterior and anterior compartments that are affected in acute compartment syndromes after tibial fractures. To describe the compartments as "peroneal" and "posterior" is hardly sufficient. One of the main reasons for monitoring compartment pressures is to locate accurately the compartment, or compartments, affected,[3] thereby minimising the extent and trauma of the surgery. Hence it is vital that all suspected compartments are monitored.

The Whitesides technique is suitable only for one off measurements and cannot, under any circumstances, be used for continuous monitoring[4]; the needle will become obstructed by a blood clot within a few minutes, and hence any subsequent reading will reflect the pressure required to remove the clot from the end of the needle rather than the compartment pressure. This might account for what are in our opinion excessive pressures in the case report described. The substitution of a wick or slit catheter for the needle will not make the measurement more accurate, as the accuracy depends entirely on the skill of the user; it will merely improve the comfort of the patient. It reduces, however, the convenience of the measurement because neither wick nor slit catheters are widely available in any hospital. An alternative would be to use a cut down epidural catheter, but care would still have to be taken to watch for blockages.

Continuous monitoring is essential to identify trends (such as whether the pressure is increasing or decreasing), to allow the initial trauma of the needle insertion into the skin to disperse, and to enable the clinician to relate signs and symptoms to pressure. For continuous monitoring it is necessary to use either a slow infusion (0·1 ml/h) or heparinised saline—preferably both. Without this, long term measurement may be uncertain.

From our experience, the pressures that Mr Macey has recorded are excessively high. A guide to their accuracy would be to examine the peripheral pulses. These are usually present in a compartment syndrome, but with the pressures recorded in this particular case the peripheral pulses would be expected to be absent, and verifying this clinically would indicate whether this was a true reading or caused by a blocked needle.

The rather drastic surgery used would have been unnecessary if all four compartments (in both legs) had been monitored. This allows only those compartments with raised pressures to be decompressed. Even if all compartments had shown raised pressures the double incision fasciotomy of Mubarak is now considered to be the surgery of choice for adequate decompression, as this carries less risk of morbidity than the partial fibulectomy used here.[5]

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Exertional gastro-oesophageal reflux

SIR,—In their study of oesophageal pH in patients with exertional chest pain but normal coronary arteries, Dr P M Schofield and colleagues (6 June, p 1459) report that 28 of 52 patients had no oesophageal reflux. They suggest abnormalities of the left ventricular wall motion as an alternative cause of chest pain in this group.

There are, however, other important non-cardiac mechanisms that produce pain, which should not be ignored. The association of chest pain with symptoms suggestive of hyperventilation is well documented.[1-4] Hypocapnia may be induced or exacerbated by exercise,[5] and we have recently shown that 77% of 24 patients with typical exertional chest pain and no electrocardiographic evidence of ischaemia developed hypocapnia during a treadmill exercise test (unpublished findings). A particular pattern of response, with the carbon dioxide pressure failing to rise within the first minute of exercise, was strongly correlated with psychiatric morbidity. Despite this, however, overbreathing at rest is reported to reproduce pain in only about half of patients suspected of inducing symptoms by hyperventilation.[6] There is evidence for a differential response to various provocations (including exercise and voluntary overbreathing at rest), and in some cases symptoms are reproduced only when hyperventilation is triggered by appropriate emotional stress.[7,8]

The aetiology of chest pain similar to angina is probably complex for several reasons. Firstly, high rates of psychiatric morbidity have been detected in patients with motility disorders of the oesophagus.[9] Secondly, hyperventilation may induce oesophageal contraction abnormalities and myocardial oxygen demand and may cause coronary artery spasm.[10-12] Finally, hyperventilation may be associated with organic lung disease, most commonly airways obstruction.[13]

There is much evidence to suggest that the syndrome of "chest pain with normal coronary arteries" is heterogeneous: causal agents include both organic and psychological factors, and important interactions probably occur between causal factors. Simple unitary theories of aetiology probably have limited explanatory power. We believe that a collaborative, interdisciplinary approach is required to unravel the complex interactions between hyperventilation, oesophageal abnormalities, respiratory disorders, and psychiatric morbidity in the aetiology of this disorder.

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Coventry: a no drinking-driving city by the year 2000?

SIR,—We applaud the initiative being taken by the Coventry Safe Driving Team (11 July, p 71) in planning to make Coventry a no drinking-driving city by the year 2000. We should, however, like to sound a note of warning that an isolated local effort may have serious adverse effects on neighbouring localities.

During the same year, 1985, in which 25 people died on the roads in Coventry (population 314 000) 75 died on the roads of Warwickshire (population 478 000). It is difficult to obtain precise local data, but if national trends are followed we can expect one third of the dead drivers to have had a blood alcohol concentration over the legal limit.[1] An observation in the records of the Warwick hospital accident and emergency department is that around one third of all the serious accidents involved people from outside the county. A significant proportion of these are from the neighbouring cities of Coventry and Birmingham.

Local knowledge indicates that many residents of the city of Coventry already drink and drive outside the city. This proportion will almost certainly increase if the city tightens up its approach to drinking and driving. We therefore suggest that any initiative directed towards drinking and driving should be coordinated on a subregional basis, taking into acount the popular recreational pursuits and commuting patterns of the city dwellers. We propose to pursue this approach actively with the Coventry Safe Driving Team and