wonders whether it is related to closing time in public bars.

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This letter was shown to the authors, who reply as follows:

SIR,—We thank Professor Tunstall-Pedoe for his interesting comments on our paper. The confirmation of the finding of a secondary midnight peak in the onset of chest pain in acute myocardial infarction in a five year retrospective analysis of a five year prospective study makes it unlikely that the peak is a chance finding. Our discussion reaches no conclusion about its possible cause but our data do not exclude the possibility that the midnight peak is a local phenomenon whose explanation might lie in local circumstances. We find the explanation in terms of public house closing times intriguing but unlikely to be correct. Our data, which have also been analysed for the frequency of acute myocardial infarction on days of the week (unpublished), do not provide significant support for the Saturday hypothesis. Moreover, a change of licensing hours applied to Leicester which liberalised weekday public house opening, occurred during the prospective data collection and did not result in a measurably stronger trend in support of the midnight peak during the later stages of the study.

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Syndrome X and hyperventilation
SIR,—Syndrome X and hyperventilation have long been associated, and Lewis and colleagues (British Heart Journal 1991; 65:94-6) are to be congratulated for their discriminating contribution to the field and for raising several important questions. A central issue is whether hyperventilation can be dismissed on the basis of a rather limited schedule of testing. For example, it is no longer thought that the forced hyperventilation provocation test (FHPVT) is the "gold standard" of hyperventilation testing,1 but that provocation by personally relevant stressors2 is at least as important. Indeed, in the context of cardiological patients, the latter stressors, which can in the instance activate the energy vasoconstrictive pathways3 are probably the triggers most relevant in everyday life. Furthermore, besides the interplay between hyperventilation and sympathetic nervous system activation (with consequences such as magnesium depletion) it seems likely that it is the neuroendocrine setting that can determine whether or not a given episode of hyperventilation has vasoconstrictive consequences.

It is a commonplace finding that many patients with recurrent hyperventilation illnesses do not hyperventilate during an exercise test, but readily overbreath in response to an emotional challenge, particularly when the challenge involves the recall of feelings of being trapped or of anger.4 As far as the data presented by Lewis et al are concerned, besides noting the absence of figures for end tidal pressure of carbon dioxide (Pco2) for their controls, we also note that the mean Pco2 values of 38 mm Hg at 50% of maximum exercise and 37 mm Hg at maximum exercise are well below expected normal values.5 The demonstration of increased minute ventilation for given minute carbon dioxide (VE/Vco2) is quite consistent with chronic hyperventilation. It is due either to reduced respiratory centre buffering6 or a flywheel effect which regularly over-activated pathways promote breathing,7,8 or both. The observation of a raised VE/Vco2 may well be the key indicator of a longer term tendency to hyperventilation, because as stated above, the FHPVT is a single act of hyperventilation and is dependent upon the patient’s starting point in terms of other influences upon vasoconstrictive activity, arterial arousal and depletion of the body’s buffering systems.

A basic question about syndrome X is whether their responsiveness to recognised vasoconstrictive influences is greater than average, just as morning and in general are more responsive than the Japanese.9 We suggest that Lewis et al have sharpened the definition of characters in the drama of syndrome X and hyperventilation but have not yet brought down the final curtain.

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This letter was shown to the authors, who reply as follows:

SIR,—We appreciate the interest shown by Dr Nixon and colleagues in our paper. Our study refers of course to carefully characterised patients—an important consideration given the widely differing patient groups represented in published reports on patients with unexplained chest pain. Clearly the filing category of syndrome X is coming to the end of its useful life, particularly in view of the confusion now arising over a different “syndrome X.”

We comment on the points raised by Dr Nixon and colleagues—our patients with syndrome X all describedtypical angina and, in every case, exercise induced the same symptom. We would argue that an exercise test in such a group represents a “personally relevant stressor”. The finding of normal arterial Pco2 values throughout exercise in our syndrome X patients formed the basis for our conclusion that we were indeed hyperventilating inappropriately (which in common usage is the implication inherent in the term). Because we showed that end tidal Pco2 correlated only poorly with arterial Pco2 in these patients, their end tidal measurements, we agree, provided no evidence either for or against a diagnosis of hyperventilation. Chronic hyperventilation might indeed increase the VE/Vco2 slope but only in the presence of low arterial Pco2 (the modified alveolar gas equation states: VE = 863 Vco2/Paco2(1-Vdw/VT). The normal arterial Pco2 in our patients might well have increased in deadspace ventilation.1 We did not measure arterial Pco2 in our control patients for ethical reasons.

We share the view that the final curtain is not yet drawn on the various players on this ill-list stage, while our spotlight continues to wander. . . .

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Alcohol ablation of atrioventricular conduction
SIR,—Dr Cunningham and Dr Rowland (British Heart Journal 1991;65:115) favour the use of “conventional” methods of ablation of atrioventricular conduction rather than the new method of ethanol ablation. “Conventional” methods include, according to them, radiofrequency ablation (with its known low success rate), higher energy shocks (with a known high complication rate), and low energy shocks (only three published reports of this—two abstracts and a small series). Long-term results with ethanol ablation remain excellent.1 No deaths have been reported after alcohol ablation. Its success rate, even when analysed on the basis of intention to treat, is comparable to “conventional” methods. The results of “conventional” methods were never presented on an intention to treat basis.

It looks to me as if there are no “conventional” or “established” methods for the ablation of atrioventricular conduction. Only long-term results will show which method offers the best results at the lowest risk. Meanwhile, investigators and physicians