**LETTERS TO THE EDITOR**

- The British Heart Journal welcomes letters commenting on papers that it has published within the past six months.
- All letters must be typed with double spacing and signed by all authors.
- No letter should be more than 600 words.
- In general, no letter should contain more than six references (also typed with double spacing).

Nocturnal hypoxaemia after myocardial infarction

Sir,—Galatius-Jensen and colleagues1 chose to monitor the arterial saturation and the electrocardiogram on days two to six after acute myocardial infarction (AMI). It is not clear why they did not study changes in the first 24 hours. We have communicated with all centres caring for patients in the UK and found that few prescribe oxygen in the first 24 hours and none routinely measures oxygen saturation. In a pilot study we also have found a high incidence of hypoxaemia in these patients (83% of those who did not receive oxygen) but we studied this in the initial 24 hours after admission with AMI.2 We have subsequently repeated our study with synchronous measurement of the Spo2 segment and arrhythmia analysis using 24 hour Holter monitoring during the first 24 hours after admission.

After studying 50 patients (25 received supplemental oxygen and 25 did not) we found hypoxaemia (Spo2 < 90%) in 43% of all patients—in 18% of those who received oxygen supplementation and in 68% of those who did not. There was severe hypoxaemia (Spo2 < 80%) in 16% of patients and 86% of these were in the group that did not receive supplemental oxygen. Because in an earlier study there was a good correlation between the incidence of hypoxaemia and opiates and because hypoxaemia was reversed with oxygen1 we attribute the high incidence of hypoxaemia to the use of opiates for analgesia immediately after AMI. Unlike Galatius-Jensen and colleagues2 we found no association between arrhythmia and the timing, incidence, or degree of hypoxaemia. Like them we did find a patient whose ST segment changes exactly corresponded in reciprocal changes in oxygen saturation and therefore we conclude that hypoxaemia may influence the degree of ischaemia. As in our original pilot study we again conclude that all patients who have had an AMI should be monitored with a pulse oximeter and that hypoxaemia should be treated appropriately.

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

Sir,—We chose nocturnal monitoring because we expected more episodes of hypoxaemia at night. We chose to monitor the patients 2–6 days after acute myocardial infarction (AMI) and found that most occurred during the first 24 hours partly for practical reasons but mainly because the finding of hypoxaemia and coincident electrocardiographic abnormalities in the subacute phase after infarction in a group of patients who were not predisposed to hypoxaemia would be a much more important finding. This is because oxygen saturation is not usually monitored 2–6 days after an acute myocardial infarction. There have been many other studies in patients with AMI,1 with severe left ventricular heart failure,2 and postoperatively3 Wilson and colleagues surprisingly found no arrhythmias coincident with hypoxaemia in 50 patients with AMI.

The high incidence of hypoxaemia after AMI may partly be explained by the use of opiates, as pointed out by Wilson et al.: but we also found, in the patentdole in the subacute phase of AMI where the use of opiates is limited. Also Hung et al.4 often found hypoxaemia in 101 men 6–61 days after AMI, which suggests that there may be other important explanations for this phenomenon.

We would be cautious about recommending monitoring of pulse oximetry and treatment of moderate hypoxaemia after AMI as standard procedure until their value has been evaluated. Trier Mellor found a high incidence of hypoxaemia perioperatively, and though treatment reduced the extent of hypoxaemia it did not reduce the postoperative complications including those affecting cognitive function.

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Increase in plasma β endorphins preceeds vasodepressor syncpe

Sir,—Wallbridge et al. further implicate β endorphins in vasodepressor syncpe. There are striking similarities between the symptoms and signs of vasodepressor syncpe and the first dose hypotension reported with converting enzyme inhibitors.2 These include vasodilatation, hypotension with bradycardia, sweating and the absence of a compensatory increase in plasma noradrenaline. These similarities raise the possibility of common pathogenetic mechanisms. Indeed, converting enzyme inhibitors can also increase plasma β endorphins,1 and the opiate antagonist naloxone blocks the acute hypotensive response to captopril in humans,2b materially implicating an endogenous opioid mechanism. This may provide support for the speculation that naloxone may also mitigate vasodepressor syncpe. On the basis of this proposition, I surmise then that captopril administration (50 mg) may improve the positive predictive value of the tilt test and obviate the need for repeated blood sampling for endorphins. This hypothesis requires testing. Unfortunately, Wallbridge et al. did not measure the plasma noradrenaline response to tilt. Examination of the relations between plasma β endorphin concentration, noradrenaline response to orthostasis, and cardiac vagal tone assessed by heart rate variability in power spectral analysis would have further clarified the relevance to the mechanism of vasodepressor syncpe in humans of sympathetic inhibition and/or vagal vagal activation induced by endogenous opioids.2b

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

Sir,—Dr Ajayi discusses the potential interaction of the endogenous enko-angiotensin and opioid systems and raises the hypothesis that plasma β endorphins may be involved in both the pathogenesis of vasovagal syncope and first-dose hypotension with converting enzyme inhibitors. It is interesting to note the experience that plasma β endorphin concentrations increase only slightly after 1 week of treatment with captopril.1 The administration of captopril before beta-endorphin's release is an interesting idea, but its sensitivity and specificity would need to be formally assessed.

Samples for plasma catecholamines were obtained during tilt testing. Changes in plasma noradrenaline concentrations in the positive (baseline 2·6 v syncpe onset 4·5 ± mmol/l) and negative (baseline 2·2 v end of test 4·6 ± mmol/l) tilt test groups were similar.

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NOTICE

The 1995 Annual Meeting of the British Cardiac Society will take place at the Conference Centre, Harrogate, North Yorkshire from 23 to 25 May.