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 colleagues[1] 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 coronary care units 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 ST 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 (Spo_2 < 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 (Spo_2 ⩽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 oxygen[3] we attribute the high incidence of hypoxaemia to the use of opiates for analgesia immediately after AMI. Unlike Galatius-Jensen and colleagues 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 with 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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1 Galatius-Jensen S, Hansen J, Rasmussen V, Bildsøe J, Therboe M, Rosenberg J. Nocturnal hypoxaemia after myocardial infarction: association with nocturnal myocardial ischaemia and arrhythmias. *Br Heart J* 1994;72:23–30.
2 Wilson AT, Reilly CS, Woodmansey P, Channer KS. Oxygen therapy in myocardial infarction. *Clin Sci* 1993;84:21p.
3 Marjot R, Valentine SJ. Arterial oxygen saturation following premedication for cardiac surgery. *Br J Anaesth* 1990;64:737–40.

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 not 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 AMI. Unlike our study and other studies in patients with AMI,[1] with severe left ventricular heart failure,[2] and postoperatively[3] 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 a high incidence late in the subacute phase of AMI when 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 with pulse oximetry and treatment of moderate hypoxaemia after AMI as standard procedure until their value has been evaluated. Trier Møller 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.[5]

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1 Toshiyuki S, Tatsusuke Y, Yujiro S, Keiji T, Tetsuo I, Ryo O. Sleep apnea in patients with acute myocardial infarction. *Crit Care Med* 1991;19:938–41.
2 Davies SW, John LM, Wedzicha JA, Lipkin DP. Overnight studies in severe chronic left heart failure: arrhythmias and oxygen saturation. *Br Heart J* 1991;65:77–83.
3 Rosenberg J, Rasmussen V, Von Jessen F, Ullstad T, Kehlet H. Late postoperative episodic and constant hypoxaemia and associated ECG abnormalities. *Br J Anaesth* 1990;65:684–91.
4 Hung J, Whitford E, Parsons R, Hillmann D. Association of sleep apnoea with myocardial infarction in men. *Lancet* 1990;336:261–4.
5 Trier Møller J. Anesthesia related hypoxemia: the effect of pulse oximetry monitoring on perioperative events and postoperative complications. *Acta Med Scand* (in press).

Increase in plasma β endorphins precedes vasodepressor syncope

SIR,—Wallbridge *et al*[1] further implicate β endorphins in the pathophysiology of vasodepressor syncope. There are striking similarities between the symptoms and signs of vasodepressor syncope 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,[3] and the opiate antagonist naloxone attenuates the acute hypotensive response to captopril in humans,[4,5] materially implicating an endogenous opioid mechanism. This may provide support for the speculation that naloxone may also mitigate vasodepressor syncope. 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, noradrenergic response to orthostasis, and cardiac vagal tone assessed by heart rate variation or power spectral analysis would have further clarified the relevance to the mechanism of vasodepressor syncope in humans of sympathetic inhibition and/or vagal augmentation induced by endogenous opioids.[6]

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1 Wallbridge DR, MacIntyre HE, Gray CE, Denvit MA, Oldroyd KG, Ras AP, Cobbe SM. Increase in plasma β endorphins precedes vasodepressor syncope. *Br Heart J* 1994;71:446–8.
2 Cleland JGF, Dargie HJ, McAlpine H, *et al*. Severe first dose hypotension after first dose of enalapril in heart failure. *BMJ* 1985; 291:1309–12.
3 Tan SA, Berk LS. Additive effects of clonidine and lisinopril on beta-endorphin levels in hypertensive diabetics. *Clin Res* 1994;42: 251A.
4 Ajayi AA, Campbell BC, Rubin PC, Reid JL. Effect of naloxone on the actions of captopril. *Clin Pharmacol Ther* 1985;38:560–5.
5 Rubin PC, Millar JA, Sturani S, Lawrie C, Reid JL. The influence of naloxone on the circulatory actions of captopril. *Br J Clin Pharmac* 1984;17:713–7.
6 Laubie M, Schmitt HH. Indication for central endorphinergic control of heart rate in dogs. *Eur J Pharmac* 1981;71:401–9.

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

SIR,—Dr Ajayi discusses the potential interaction of the endogenous renin-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 our experience that plasma β endorphin concentrations increase only slightly after 1 week of treatment with captopril.[1] The administration of captopril before tilt testing 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* syncope onset 4·5 μmol/l) and negative (baseline 2·2 *v* end of test 4·6 μmol/l) tilt test groups were similar.

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1 Oldroyd KG, Gray CE, Carter R, Harvey K, Borland W, Beastall G, Cobbe SM. Activation and inhibition of the endogenous opioid system in human heart failure. *Br Heart J* 1995;73:41–8.

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.