considerably reduced the myocardial concentration of oxygen radicals,² improved recovery of energy metabolism^{3,4} and of contractile function in isolated hearts^{3,4} and in anaesthetised dogs,⁵ and reduced infarct size in a long term canine model of coronary artery occlusion and reperfusion.⁶

Thus although the issue is not yet settled,⁷⁻⁹ there seems to be substantial evidence to indicate that, under certain circumstances, oxygen radicals can induce specific myocardial damage, which is separate from ischaemic damage, in various experimental models of post-ischaemic reperfusion. The studies that I draw attention to go some way to proving that scavengers of oxygen radicals given at the moment of reperfusion are effective in countering this specific myocardial damage.

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

Sir,

The results of many studies confirm the occurrence of a reperfusion event associated with the formation of oxygen free radicals in animals.¹ There is similarly convincing evidence that in the animal heart this event is associated with myocardial injury and that the extent of this injury may be modified by agents altering free radical mechanisms.² The optimal timing for such therapeutic intervention remains open to debate, but certainly some benefit may be achieved from treatment given after the ischaemic period.

However, small animals may be particularly susceptible to oxidative stress,^{3,4} and therefore more likely to benefit from antioxidant treatment, and the applicability of animal studies to patients with acute myocardial infarction is in some doubt.

From a practical point of view, long term prophylactic treatment for patients at risk of infarction with agents such as allopurinol seems unlikely to become widely established even if a small benefit were shown, and the potential beneficial effects of free radicals⁵ may be jeopardised by such treatment. An "anti-free radical" cocktail administered at the same time as a thrombolytic agent (that is after the ischaemic period, immediately before reperfusion) is the most promising starting point, and we agree with Ambrosio that any evidence that this may be beneficial in animals^{6,7} is encouraging. However, this does not alter our conclusion that therapeutic trials are not justified without further elucidation of free radical interactions in humans (1989;61:4-8).

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Notices

British Cardiac Society

The Annual General Meeting will take place at the English Riviera Centre, Torquay on 22 to 25 May 1990. The closing date for receipt of abstracts is 19 January 1990.

Advances in diagnostic and therapeutic cardiac catheterization

A meeting entitled Advances in Diagnostic and Therapeutic Cardiac Catheterization, organised by Dr Carl J Pepine, will take place at the Peabody Orlando, Orlando, Florida on 16 to 18 February 1990. Further information from Margaret Sgro, Extramural Programs Department, American College of Cardiology, 9111 Old Georgetown Road, Bethesda, MD 20814, USA.

Hyperlipidemia in childhood and the development of atherosclerosis

A meeting on Hyperlipidemia in Childhood and the Development of Atherosclerosis sponsored by The New York Academy of Sciences will take place at the Hyatt Regency, Bethesda, Maryland on 2 to 4 May 1989. Further information from the Conference Department, The New York Academy of Sciences, 2 East 63rd Street, New York, NY 10021, USA.