Anticoagulation after intracoronary stent insertion

Sin—Anticoagulation after intracoronary stent insertion is a controversial issue and, because the anticoagulation protocols used in different hospitals are short-lived, nurses and junior staff have to adjust to ever-changing protocols at a rapid pace. The article by Brack et al gives the impression that the proposed anticoagulation strategy is an accepted mode of treating patients after stent implantation. This is misleading and requires comment.

The paper was accepted for publication on the 9 February 1994. In the meantime most institutions have relaxed their anticoagulation scheme considerably. As Brack et al state in their last sentence, low molecular weight heparin has replaced intravenous heparin in many centres and no anticoagulation variables are monitored. The relatively elaborate approach proposed in this article adds considerably to the work of the nursing staff and to the cost. Also, heavy anticoagulation can cause several local and systemic complications. The fact that Brack et al saw no stent thrombosis in their last 50 procedures after this relatively strict anticoagulation protocol does not mean that the antithrombotic regimen was responsible for the good outcome. It may well be that the learning curve of the stent implantation overlaid use of this protocol. Most investigators involved in this topic now agree that it is the primary result after stenting that determines the incidence of thrombus occlusion rather than the anticoagulation regimen. If the primary result is impeccable without any residual stenosis (as judged by digital angiography and intravascular ultrasound) and with excellent apposition of all stent struts in all segments and good flow, anticoagulation with heparin and warfarin can probably be dispensed with. Aspirin may then be the only recommended treatment.

I recommend that heavy anticoagulation be reserved for cases where the results of stenting are less than perfect.

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An image in the cardiology section of the October 1994 issue, Dr Davies presented an unusual case of poor cardiac output. The discussion of this case raises a perennial issue—that of the cause of dyspnoea in heart diseases, including congestive heart failure.

Dr Davies stated that in his patient the dyspnoea on exertion was caused by poor cardiac output that was the result of inadequate venous return. This is improved when the dose of diuretic was reduced. This seems a classic case of, to use the old-fashioned term, forward cardiac failure rather than the backward cardiac failure, which would present with pulmonary congestion. Though Dr Davies’ observations are undoubtedly correct, they raise yet again the question why this man was dyspnoeic when there is no aspiration of any pulmonary congestion, and when, in fact, the implication was that the patient was somewhat hypovolaemic. These patients complain of dyspnoea well before the onset of lactic acidosis, so that this alone is not a sufficient explanation.

For several years we have been studying the mechanisms underlying the excessive ventilatory response to exercise in patients with chronic heart failure in whom the dose of diuretic is adequate. We have evidence that one contributory factor may be an abnormally active muscle signal transmitted via unmyelinated afferents from so-called “ergoreceptors” within skeletal muscle. This explanation has the advantage of combining the mechanisms of fatigue and dyspnoea in one mechanism—that of abnormal peripheral signal, which thus sends an abnormally active signal to the cortex, mediating both fatigue and dyspnoea. Both end stage liver disease and some of the non-metastatic manifestations of neoplastic disease can present with unexplained fatigue and dyspnoea. Both conditions are also frequently associated with a peripheral myopathy.

Throughout these studies I have been surprised, even amazed, by the strength of the firmly held belief that we already know the cause of dyspnoea in heart failure. Even to question whether pulmonary congestion is the primary cause is met with incredulity, and if expert referees’ reports on grant applications are the yardstick, then clearly even expert opinion does not accept there is a problem to investigate. Yet despite this we see case after case in which the accepted explanations for the generation of dyspnoea are patentely unable to explain this disabling symptom.

Could this be another case of the facts getting in the way of firmly held belief?

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NOTICES

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

A course on practical adult cardiovascular pathology organised by Professor M J Davies and Dr M N Sheppard will take place at the Royal Brompton National Heart and Lung Institute on Monday 16 October 1995. For further information, please contact Dr Mary N Sheppard, National Heart and Lung Institute, Dovehouse Street, London SW3 6LY (tel: 0171 351 8172; fax: 0171 376 3442).

The ninth annual meeting of the Mediterranean Association of Cardiology and Cardiac Surgery will take place on 20-25 October 1996 in Tel Aviv, Israel. For further information, please contact The Secretariat, 9th Annual Meeting of Cardiology and Cardiac Surgery, PO Box 50006, Tel Aviv 61500, Israel (tel: +972 3 514 0014; fax: +972 3 517 5674/514 0077).