

dorsal or ventral roots.³ Complete recovery follows herpes zoster paresis in 50–70% of reported cases.⁴

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Right heart failure following acute myocardial infarction

Sir,

Clinical signs of right heart failure are not uncommon after acute myocardial infarction. However, we believe that inadequate emphasis is given to the greatly differing implications of such signs following inferior compared to anterior infarction.

Inferior left ventricular infarction usually results from an occlusion of the right coronary artery. If such an occlusion is proximal enough to involve the right ventricular branches then either transient right ventricular dysfunction (60–80% of cases) or true right ventricular infarction (18–40%) will accompany the left ventricular damage.¹ Thus when clinical signs of right heart involvement occur after inferior myocardial infarction, they are usually prominent and out of proportion to any co-existing left ventricular impairment—so much so as to constitute a distinct form of cardiogenic shock.² By contrast, anterior infarction is usually associated with occlusion of part of the left coronary tree, the resultant damage being confined to the left ventricle and totally sparing the right ventricle. In this situation signs of right heart dysfunction are rare. When they do occur they result from secondary backward failure due to a proportionate major disturbance of left ventricular function rather than to any right ventricular involvement.

This distinction is well recognized to be of importance from a therapeutic standpoint, particularly with regard to the use of diuretics. However, it also has important diagnostic implications since it is clear from the above that the presence following acute anterior myocardial infarction of right heart signs which are prominent, out of proportion to any left ventricular impairment and which

persist after treatment with diuretics should be regarded with suspicion and lead to a search for independent but co-existing causes of right ventricular strain.

We have recently had two such cases. In both cases ventilation-perfusion scanning allowed the recognition of multiple pulmonary emboli, occurring despite prophylactic low dose subcutaneous heparin, as the causative pathology of the right heart strain. The co-existence of myocardial infarction and pulmonary emboli is not at all surprising since ischaemic heart disease and particularly congestive cardiac failure are major risk factors for the development of pulmonary embolic disease^{3–5} and of course the possibility of right ventricular mural thrombus adds further to this risk.^{6–7}

Consequently, consideration needs to be given to the diagnostic implications of right heart signs following acute anterior myocardial infarction lest co-existing pathology and particularly pulmonary emboli be overlooked.

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Ketamine tolerance

Sir,

Anaesthetic/analgesic properties of ketamine are now well established. We have observed tolerance to ketamine in burn cases. A young male of 25 years was admitted to the Combined Military Hospital Rawalpindi with 60% burns. Repeated dressings were done under ketamine anaesthesia. He weighed 55 kg. Ketamine, 100 mg i.v., was given on the first dressing and the patient started responding to surgi-