73 COMPARISON OF THE ACUTE EFFECTS OF AMRINONE AND NITROPRUSSIDE ON HAEMODYNAMICS AND MYOCARDIAL METABOLISM IN PATIENTS WITH LEFT VENTRICULAR FAILURE

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Amrinone and nitroprusside are vasodilators, but it has been suggested that amrinone is also a positive inotropic agent. We compared the effects of the drugs [IV] in patients with cardiac failure. Eight had dilated cardiomyopathy and six had coronary artery disease. Left ventricular pressure, cardiac output, coronary sinus flow, and oxygen and metabolite contents of arterial and coronary sinus blood were measured. Aortic flow was measured in eight patients. All patients received 55 μg/min nitroprusside and nine (five with cardiomyopathy) also received 110 μg/min. Subsequently all received 1.3 g/kg amrinone and nine had 3.5 g/kg. Responses to the drugs was not affected by the etiology of cardiac failure. Both drugs reduced left ventricular end-diastolic and systolic pressures and oxygen consumption, increased cardiac index and myocardial efficiency in dose related manners. The effects of the two drugs on myocardial substrates were indistinguishable. Pressure derived indices of left ventricular contractility were unaltered by either drug. In individual patients relatively large increases in max dp/dt were seen with amrinone. However in these arterial free fatty acid concentration ([FFAA] ART) rose considerably, whilst in the group (FFAA) ART was unaltered. Changes in cardiac index but not contractility were linearly related to log plasma amrinone concentration. Amrinone altered max dPowers/dt linearly with the change in (FFAA) ART (p < 0.05). These data suggest that in the groups of patients with cardiac failure the effects of amrinone are indistinguishable from those of nitroprusside, and are due to vasodilatation. In individual patients who show evidence of an inotropic effect with amrinone, the coincident changes in (FFAA) ART suggest that this effect may be secondary to catecholamine release.

74 EXERCISE DESATURATION IN C.O.A.D.-TREADMILL VERSUS BICYCLE

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Several patients with severe chronic obstructive airways disease (FEV₁, range 0.52-1.251) showed marked arterial oxygen desaturation during a self-paced treadmill walking test (Beaumont et al) but to a much lesser extent during 1' incremental bicycle exercise test. In four patients this was further investigated by means of a self-paced 6 minute treadmill walk followed by a 6 minute bicycle exercise with work loads adjusted to mimic the oxygen consumption achieved during the treadmill exercise. During both tests, heart rate, oxygen saturation, ventilation, oxygen consumption, carbon dioxide production and breathlessness were recorded.

All four patients showed greater desaturation on the treadmill (mean 4.65% to 7.4%) than on the bicycle (mean 6.8% to 7.7%) despite similar patterns of oxygen consumption during the two forms of exercise.

One man was re-studied with arterial blood samples, revealing similar levels of PaCO₂ during the two types of exercise but rising lactate, higher Ve', VO₂ and RQ during bicycle exercise. This suggests anaerobiosis during cycling, not present during walking.

PaCO₂ fell by 12 mmHg during treadmill exercise but only 6 mmHg during bicycle exercise. The subject experienced less breathlessness on the bicycle despite the fact that Ve' was higher, as was PaO₂.

Clearly, testing on a bicycle can seriously underestimate arterial desaturation due to exercise in patients with severe chronic obstructive airways disease.


75 INCREASED DIAPHRAGM ACTIVATION IN CHRONIC VENTILATORY FAILURE

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In patients with chronic airflow obstruction (CAFO) and chronic CO₂ retention, a reduced increment in diaphragm electromyogram (Edi) has been found during CO₂ rebreathing, suggesting a reduced central respiratory drive (Lowrence, RV and Miranda, JH [1968] NEJM 279, 53-59). However in this study Edi was not normalized, making interpretation of incremental changes difficult. We have previously shown that Edi, recorded by a stabilized bipolar oesophageal electrode, can be normalized by expressing peak moving average Edi for each tidal breath as a percentage of maximum diaphragm activation (Edi max) at full inflation (Gribbin, HR et al [1982] Clin Sci, 63, 41P). We have measured Edi max and tidal Edi in 6 normal subjects and in 5 patients with severe CAFO (mean FEV₁ = 0.54 ± 0.17 l) and CO₂ retention (mean mixed venous PCO₂ = 8.35 ± 0.23 kPa) during hyperoxic hypercapnic rebreathing. At the start of rebreathing Edi/Edi max was much greater in the patients than in the normal subjects (59.6% vs 16.3%). In the normal subjects as rebreathing continued Edi/Edi max tended to plateau at values ranging from 24-64% (mean 43.8%). In the patients Edi/Edi max rose progressively and approached maximum values at the end of rebreathing (range 76-96%). Therefore in these patients diaphragm activation both at the start and end of rebreathing was increased compared to normal subjects suggesting that central respiratory drive was well preserved.