HARNESSING THE AFTERLOAD

By

Ali Muhammad M.B., F.R.C.P. F.A.C.C.D.

Pump Failure is characterised by rise in venous pressures and decrease of cardiac output. The traditional treatment of pump failure, therefore, involves therapeutic modalities aimed at countering and conquering these haemodynamic abnormalities. Improvement in cardiac output is usually obtained by increase in heart rate and pressure and by improvement in myocardila contractile state. The failing myocardium is working against peripheral vascular resistance which is often raised due to hypercateho laminemia thus increasing myocardial work for which the already depleted myocardial reserve is not prepared. The resistance to ventricular ejection is called after load. Increase in afterload leads to decrease in left ventricular storke volume and rise in left ventricular end-diastolic and end-systolic volumes. The elevation of left ventricular end-diastolic pressure which is transmi ted through the left atrium to the pulmonary venous system effecting increase in pulmonary venous pressure which is a precursor of pulmonary congestion and oedema. Decrease in afterload will promote better left ventricular ejection, increase in stroke volume and cardiac output and decrease in left ventricular en-diastolic and end-systolic volumes. This paves the way for reduction in the left ventricular end-diastolic pressure and, in conseque ence, fall in pulmonary venous pressure, decrease of pulmonary congestion and oedema and increase in lung complicnce which further reduces oxygen requirements. Nitroglycerine or amyl nitrate may produce beneficial haemodynamic consequences by reducing the afterload, increase in cardiac output and reduction of left ventricular end-diastolic presure in congestive heart failure.

In acute myocardial infarction, early recognition and promopt treatment of arrhythmias in coronary care units has considerably reduced the immediate mortality. Morbidity and mortality from cardiac failure and cardiogenic shock are still alarmingly high and frequent and are unaffected by coronary care inroads. It has been demonstrated that heart failure and cardiogenic shock are mainly determined by the size of infarct; cardiogenic shock, for instance, is usual when more than 40 percent of myocardium is necrosed. Around the infarcted myocardium is the ischaemic zone which may be retrieved by reduction of myocardial oxygen consumption. This may be achieved by lowering the force and velocity of contraction, reducing tension in the ventricular wall and slowing the heart rate (Maroko et al., 1971). The size of infarct may be predicted from the rate of rise of serum creatinine phosphokinase (CPK) during the first seven hours after admission to the hospital. The effect of any therapeutic modality on the infarct size

Consultant Physician and Cardiologist, Hyderabad.

may, therefore, be assessed by comparing the predicted and actual infarct size as judged by serial serum creatinine phosphokinase (Sobel et al., 1972). It has been reported that by reducing blood pressure in hypertensive patients suffering from acute myocardial infarction, the size and intensity of myocardial infarction may be reduced directly proportional to the reduction of predicted serum creatinine phosphokinase values (Shell et al., 1973; Shell and Sobel, 1974). Vasodialator therapy in such a clinical profile reduces left ventricular end-diastolic pressure and mean left atrial pressure. The use of sublingual nitroglycerine in acute myocardial infarction has been reported to reduce the size of myocardial infarction by its vasodilator effect causing fall in the afterload (Gold et al. 1972). Braunwald and Maroko (1974) have listed several thereputic interventions including those which reduce the afterload which may reduce the infarct size.

In heart failure due to acute myocardial infarction, lowering of afterload effected by vasodialator therapy may improve cardiac output and decrease left ventricular end-diastolic and pulmonary venous pressure. The improvement in cardiac output is witnessed in patients with elevated left ventricular end-diastolic pressure (LEVDP greater than 15 mm.Hg.) irrespective of their arterial blood pressure. In patients with normal left ventricular end-diastolic pressure, there is usually no increase of cardiac outputs on decrease of afterload. In some patients with heart failures in acute myocardial infarction with raised left ventricular end-diastolic pressure, cardiac output may infact fall following the exhibition of vasodialator therapy. This is a subgroup in which the use of vasodialtors probably causes disproportionate and inappropriate dilatation of capacitance vessels resulting in decrease of venous return and cardiac output. The use of vasodilators may be harmful since by reducing the aortic pressure, they may encroach on coronary perfusion tipping supply and demand balance to the patient's disadvantage. Their use, therefore, should invite monitoring of cardiac output, left ventricula end-diastolic pressure, peripheral vascular resistance, heart rate, blood pressure, serum enzymes and electrocardiogram.

Reducing the afterload has recently been reported to lower the regurgitation fraction when used in mitral regurgitation. The outcome is determined by two factors: resistance to left ventricular ejection and severity of mitral regurgitation. (Bruanwald et al. 1957). Increased resistance to left ventricular ejection would increase regurgitant fraction. This can be demonstrated by exhibition of phenylephrine. Reduction of systemic vascular resistance as by amyl nitrite would reduce mitral regurgitation. This has been well illustrated by change in the intesity and duration of regugitant murmur by phenylephrine and amyl nitrite, the former accentuating and the latter reducing the pansystolic murmur of mitral regurgitation. Harshaw et al. (1975) have demonstrated significant enhancement of forward flow with concominant decrease in regurgitant fraction in severe mitral regurgitation during vasodilator therapy. Vasodilator therapy may be indicated in severe mitral reguargitatin, mitral regurgitation complicating acute myocardial infarction and in mitral reguargitation with heart failure or perhaps heart failure causing mitral regurgitation by dysfunction of subvalvular apparatus.

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