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## Coronary hemodynamics in heart failure

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In chronic systolic heart failure, there is a significant change in both myocardial oxygen demand and oxygen supply. In systolic heart failure myocardial oxygen consumption (MVO2) is increased. The heart rate is usually increased but the systolic blood pressure is lower than normal. Thus the heart rate blood pressure product, a determinant of myocardial oxygen demand remains unchanged. Left ventricular contractile function is reduced. Thus increased MVO2 is not related to changes in contractile function.

In patients with systolic heart failure, left ventricular end-diastolic and end-systolic volumes are markedly increased. There is little or no change in its wall thickness. Thus there is a substantial increase in wall stress which is associated with increased myocardial oxygen demand and MVO2. Increased left ventricular mass is also associated with increased myocardial oxygen demand. Insulin resistance which is associated with increased MVO2 is increased in systolic heart failure.

In systolic heart failure myocardial perfusion is impaired. The activation of vasoconstrictor neurohormones such as norepinephrine, angiotensin and vasopressin increases coronary vascular resistance and decreases coronary blood flow. Both endothelium dependent and independent coronary vasodilatation is decreased. Decreased transmyocardial pressure gradient reduces subendocardial perfusion and precipitate subendocardial ischemia which may be associated with myocyte necrosis and apoptosis.

Increased myocardial oxygen extraction, increased levels of 2, 3diphosphoglycerate, increased p50 and a right ward shift of oxygen dissociation curve tends to maintain oxygen supply but these compensatory mechanisms are inadequate. Thus myocardial ischemia may occur from an imbalance of myocardial oxygen demand and supply in systolic heart failure.

## **Biography**

Kanu Chatterjee obtained MBBS degree from R.G.Kar Medical College, Kolkata, India. He is a fellow of Royal college of Physician of London and Edin. He is currently Kanu and Docey Edwards Chatterjee Chair in Cardiovascular Medicine, University of Iowa. He is also Emeritus Professor of Medicine, University of California San Francisco.

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