

# Pathophysiology of Cardiogenic Shock

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## Definition

Cardiogenic shock is defined as severe circulatory collapse due to primary defect in the pumping mechanism of the heart.

## Hemodynamics

The failing heart is unable to generate the pressure so there is hypotension. The heart is unable to pump out the blood received with resultant low cardiac output and usually increased left ventricular filling pressure.

## Causes

- |  |                                    |
|--|------------------------------------|
|  | Left Ventricular                   |
| 1. Acute myocardial infarction <   |                                    |
|  | Right Ventricular                  |
| 2. Cardiomyopathy  |                                    |
| 3. Myocarditis   |                                    |
| 4. Mechanical causes   |                                    |
|  | (i) Cardiac tamponade.             |
|  | (ii) Atrial myxoma or thrombus     |
|  | (iii) Massive pulmonary embolism   |
| 5. Acute mitral regurgitation due to papillary muscle dysfunction following myocardial infarction. |                                    |
| 6. Arrhythmias.  |                                    |
|  | (i) Tachyarrhythmia                |
|  | (ii) Bradyarrhythmia               |
|  | (iii) Loss of normal A.V. sequence |
| 7. Post-cardiopulmonary bypass surgery.  |                                    |
| 8. Myocardial suppression  |                                    |
|  | a) Hypoxia                         |
|  | b) Acidosis                        |
|  | c) Drugs                           |
|  | d) Hypoglycemia                    |

Cardiogenic shock following acute myocardial infarction is the prototype.

## Incidence

10-20%

## Determinants

- 1 Three vessel disease.
- 2 Amount of myocardial damage. This is the most important criteria. Usual amount of myocardial damage is about 40% or more.
- 3 Location of infarction.
- 4 Focal areas of necrosis apart from the infarct area.
- 5 High frequency of coronary arterial thrombi.
- 6 Valvular dysfunction.

## Hemodynamics

The following hemodynamic changes occur in the evolution of cardiogenic shock.

Stroke volume (amount of blood pumped per beat): As the pumping power of the heart is lost, the stroke volume is reduced.

Cardiac output (the amount of blood ejected per minute): As the stroke volume drops, there is drop in cardiac output. This drop in cardiac output is compensated to some extent by the increase in heart rate.

The systolic blood pressure: drops below 90 mm Hg as a result of drop in cardiac output.

Left ventricular end-diastolic volume increases: As a result of loss of ventricular pumping, following the loss of myocardium, the left ventricle is unable to pump the blood it receives with resultant increase in end-diastolic volume. This has beneficial effect because this increase in end-diastolic volume stretches the length of the muscle fibres and according to the Frank-Starling Law (Fig-1), the force of contraction is proportional to the initial length of muscle fibre. Therefore, as the initial length of the muscle fibres increase, these become more efficient. However, this occurs up to a certain limit and ultimately the curve will fall and the result is cardiogenic shock.

Left ventricular end-diastolic pressure rises as a result of the rise in end-diastolic volume. This rise in pressure exerts two major effects. One is that it transmits itself back from the left ventricle to the left atrium and the lungs. Second, the rise in end-diastolic pressure in combination with the low diastolic pressure, leads to reduction of coronary artery perfusion which results in sub-endocardial ischemia as the coronary perfusion depends on the gradient between the diastolic

Fig-1

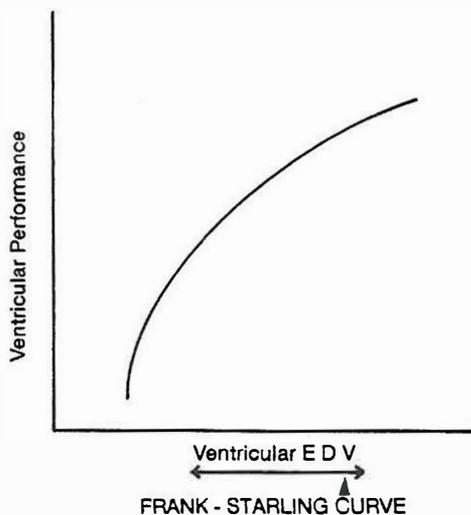


Fig. 1: Frank - Starling curve.

blood pressure and the end-diastolic left ventricular pressure, and this gradient is reduced.

As the left ventricular end-diastolic pressure rises, the left atrial pressure also rises and this transmits back with resultant rise in pulmonary capillary wedge pressure. This increase in hydrostatic pressure in pulmonary capillaries overcomes the oncotic pressure with resulting transudation of fluid into the alveoli leading to pulmonary oedema. With the passage of time, there will be rise in the pulmonary artery pressure, rise in right ventricular end-diastolic pressure and the central venous pressure as well.

As the blood pressure falls, there are sympathetic responses through baroreceptors and chemoreceptors and these acting via the vasomotor center constrict the peripheral vessels. There is constriction of arterioles and venules. When the arterioles constrict, the peripheral resistance or afterload increases and the left ventricle has to pump against high resistance and this will further deteriorate the left ventricular function. As the venules are capacitance vessels, when these constrict, the blood is pushed into the right atrium and right ventricle and on to the left ventricle. In the initial stages, this has beneficial effect as the increase in end-diastolic volume increases the force of contraction but with the passage of time, this will further increase the already elevated end-diastolic ventricular volume beyond the limits of the Frank-Starling curve.

## Effects On Various Systems

### CNS

In the central nervous system, the effects are because of hypotension and hypoxia.

Hypotension excites the baroreceptors which are pressure receptors. These normally send inhibitory impulses to the vasomotor center. These inhibitory stimuli are lost because of hypotension with resultant increase in sympathetic discharge. Hypoxia on the other hand excites the chemoreceptors which in turn stimulate the vasomotor center with resultant increase in sympathetic discharge. This increase in sympathetic discharge will cause:-

1. Tachycardia.
2. Increase in the force of contractility
3. Vasoconstriction.

Clinically there will be sweating, pale, ashen facies, extremities are cold and the sensorium is dull due to cerebral hypoxia (Fig. 2).

### Changes In The Lungs

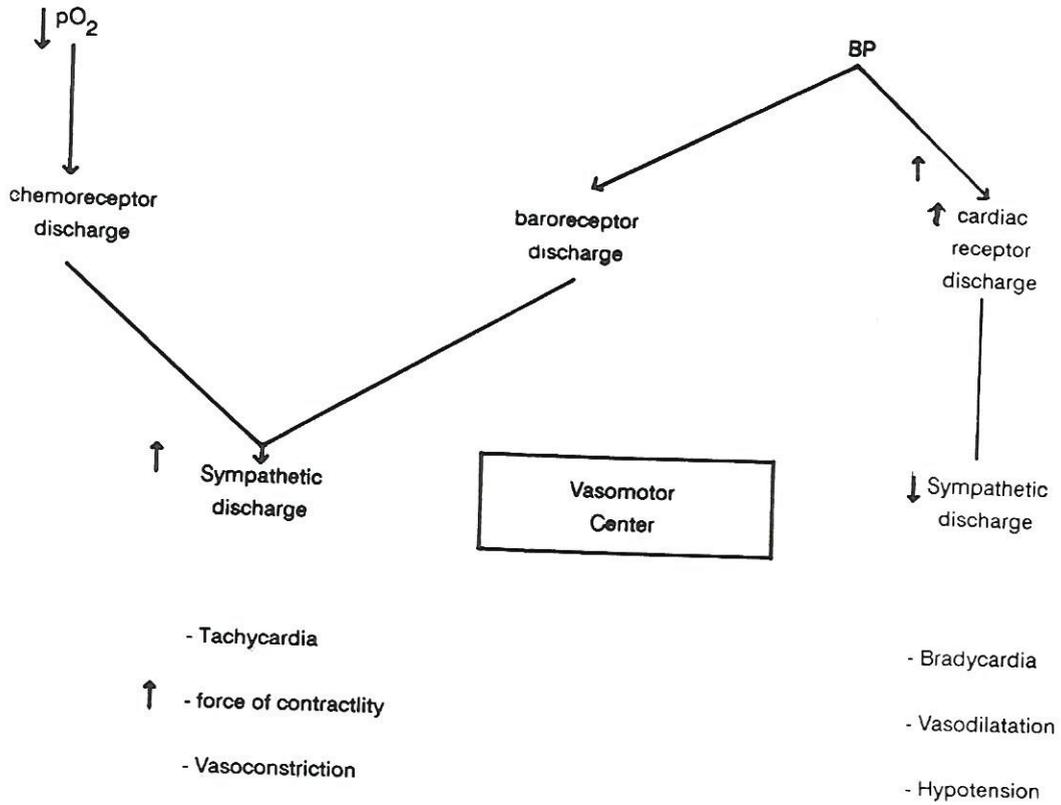
Due to left ventricular dysfunction, there is pulmonary venous hypertension which leads to pulmonary edema with resultant decrease in  $pO_2$ . There is increase in respiratory rate and work for which hypoxia and stretch of receptors within the lungs provide the stimulus. Due to the increase in respiratory rate and work, the fraction of already reduced cardiac output going to the respiratory muscles is increased. This increased fraction is, however, still lower than the normal absolute amount of blood supply to the respiratory muscles, so there will be anaerobic glycolysis with lactic acidosis. This lactic acidosis leads to further impairment of LV function.

### Clinical Correlation

- Tachypnoea.
- Orthopnoea.
- Excessive accessory muscle use.
- Basal crepitations.
- Rhonchi.

### Renal Changes

The decrease in cardiac output and blood pressure will decrease renal perfusion. This will lead to stimulation of renin-angiotensin system. Angiotensin II is a potent vasoconstrictor which leads to increase in peripheral resistance which will further increase L-V



Clinical correlation: Sweating; Pallor; Cold periphery; Restless, drowsy, confused.

Fig. 2: Effects of cardiogenic shock on central nervous system.

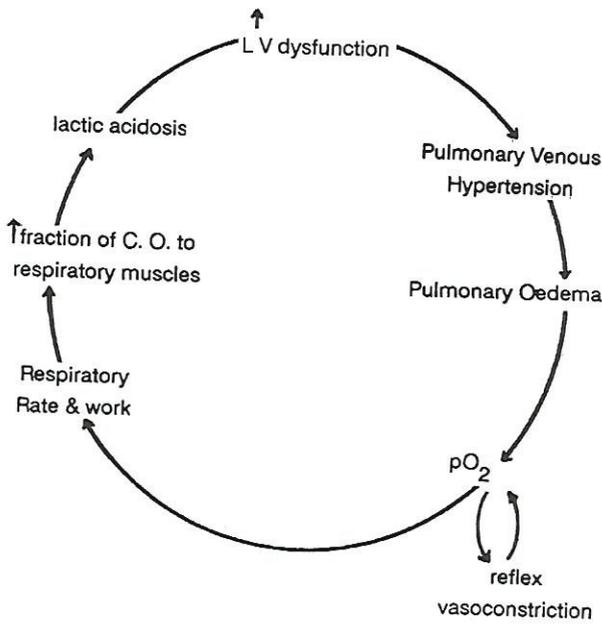
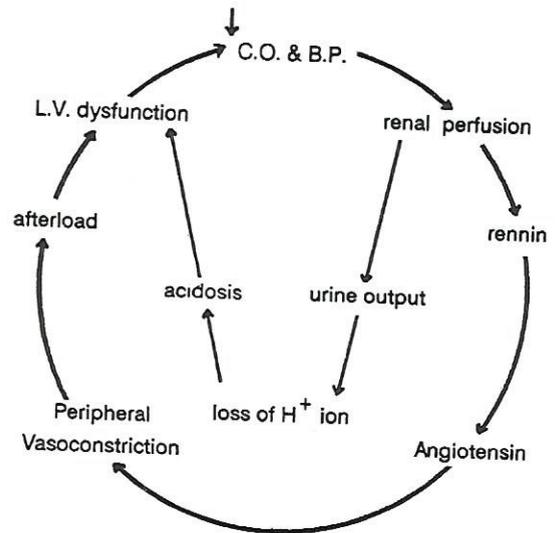


Fig. 3: Changes in the lungs in cardiogenic shock.



Clinical Correlation: Low hourly urine output.

Fig. 4: Renal changes in cardiogenic shock.

dysfunction causing further decrease in cardiac output and renal perfusion (Fig. 4). Clinically there will be low hourly urine output.

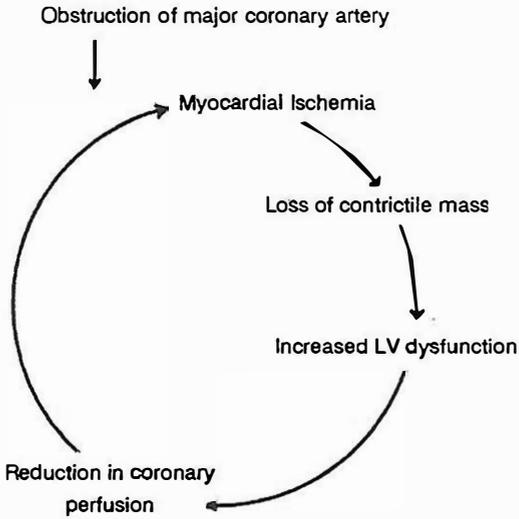


Fig. 5: Obstruction of major coronary artery.

### CONCLUSION

Obstruction of a major coronary artery causes myocardial ischemia due to loss of contractile mass and leads to left ventricular dysfunction. This in turn further reduces the coronary perfusion causing more ischemia and worsening the degree of left ventricular dysfunction. The cycle is thus perpetuated and compounded leading ultimately to death (Fig. 5).

### PATHOPHYSIOLOGY

