Mechanical Circulatory Devices in Acute Heart Failure

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KEYWORDS

• Cardiogenic shock • Acute heart failure • Mechanical circulatory support
• Ventricular assist device

KEY POINTS

- Cardiogenic shock can complicate acute myocardial infarction, occur after cardiac surgery, and develop in the setting of chronic heart failure.
- Temporary ventricular assist devices (VAD) provide rapid hemodynamic stabilization and can improve survival in certain causes of cardiogenic shock.
- Indications for temporary mechanical support are bridge to decision in patients whose candidacy for advanced heart failure therapies is uncertain; bridge to transplant or durable VAD; or bridge to recovery of native cardiac function.
- Common complications of temporary ventricular device therapy are bleeding, infection, thromboembolism, and limb ischemia.

INTRODUCTION

Cardiogenic shock is defined as myocardial dysfunction causing decreased cardiac output and tissue hypoxia despite adequate intravascular volume.[1] It is determined by various clinical and hemodynamic parameters. Hemodynamic criteria include sustained hypotension (systolic blood pressure [SBP] less than 90 mm Hg for greater than 30 minutes or the need for supportive measures to maintain SBP >90 mm Hg) or reduced cardiac index (<2.2 L/min/m^2) with an elevated pulmonary capillary wedge pressure (>15 mm Hg).[2] Clinical criteria include evidence of end-organ

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hypoperfusion, such as cool extremities, low urine output (less than 30 mL/h), altered mental status, pulmonary congestion, elevated lactate, and mixed venous saturation less than 65%.[3]

Causes of Cardiogenic Shock

The most common cause of cardiogenic shock is acute myocardial infarction (MI). It can also be caused by mechanical complications of MI, such as acute mitral regurgitation or rupture of the interventricular septum or the free wall. Nonischemic causes of shock include acute decompensation of a chronic cardiomyopathy, myocarditis, postcardiotomy syndrome, and cardiac allograft dysfunction. The various causes of cardiogenic shock are summarized in **Table 1**. Although left ventricular (LV) dysfunction is the most common cause of cardiogenic shock, right ventricular failure (RVF) (whether isolated or contributing to biventricular failure) can also produce shock and is discussed in subsequent sections.

Continuum of Cardiogenic Shock

The severity of cardiogenic shock covers a wide spectrum (**Fig. 1**). Patients in mild shock can be adequately treated with low-dose inotropes and vasopressors, such as dobutamine, dopamine, and norepinephrine. Patients in more profound shock refractory to additional inotropes and vasopressors require mechanical circulatory support (MCS) to maintain adequate systemic perfusion.[4] With worsening cardiac failure, cardiac output decreases, LV filling pressures increase and if the shock becomes profound then a vasodilatory state ensues. The most common initial device used in shock is the intra-aortic balloon pump (IABP), however the IABP only modestly augments cardiac output and reduces LV filling pressures. It functions as a volume-displacement device, with IABP deflation lowering aortic pressure and, thus, afterload resulting in improved forward flow. Extracorporeal membrane oxygenation (ECMO) can provide total cardiopulmonary support; it has significant drawbacks, including significant bleeding risk, activation of inflammatory cascades, potentially inadequate ventricular unloading when placed peripherally, and vascular injury or insufficiency from the large-bore peripheral catheters required during implantation.[5] A temporary ventricular assist device (VAD) provides the ventricular unloading and hemodynamic support needed in patients with severe refractory cardiogenic shock.

BENEFITS OF MCS

The main goal of MCS is to decompress the failing ventricle and augment systemic perfusion.[6] Mechanical unloading of the LV decreases pulmonary congestion, reduces pulmonary arterial pressure, and improves RV function. These hemodynamic benefits are also associated with favorable cellular changes. Normalization of LV pressure reduces neurohormonal activation, which decreases catecholaminergic excess that is myotoxic.[7] LV decompression reduces myocardial oxygen consumption and promotes myocardial recovery.[8] Cardiac myocytes from VAD-supported patients have less fibrosis and collagen content in the cardiac extracellular matrix when compared with nonsupported patients, indicating a reduction in reverse remodeling.[9] They also have improved myocyte contractile properties, such as increased magnitude of contraction with shortened time to peak contraction, and reversed downregulation of beta-receptors with increased response to beta-adrenergic stimulation.[10,11] These findings suggest that mechanical support may help reverse some cardiac dysfunction in heart failure.