# Multivessel Revascularization in Shock and High-Risk Percutaneous Coronary Intervention

Sandeep K. Krishnan, MD, Robert F. Riley, MD, MS, Ravi S. Hira, MD, William L. Lombardi, MD*

**KEYWORDS**

• Multivessel revascularization • Percutaneous coronary intervention • Coronary artery disease • Cardiogenic shock • Revascularization

**KEY POINTS**

- Current guidelines support multivessel percutaneous coronary intervention in the setting of cardiogenic shock.
- It remains to be determined if those benefits extend consistently to patients with stable coronary artery disease.
- Trials underway, including COMPLETE and OPEN-CTO—along with many others, may help to address these questions.
- It is clear that clinicians must remain vigilant to do what is best for each patient on a case-by-case basis after discussing all options.

## MULTIVESSEL CORONARY ARTERY DISEASE AND CARDIOGENIC SHOCK

Approximately 40% to 60% of patients undergoing percutaneous revascularization have multivessel coronary artery disease (CAD), defined as 70% or greater stenosis in 2 or more coronary arteries or involving the left main.[1] Coronary artery bypass grafting (CABG) continues to carry a class I recommendation for many of these patients from the latest American College of Cardiology/American Heart Association guidelines statement,[2] but as many as 30% to 40% of patients with multivessel disease (MVD) and class I indications for CABG undergo percutaneous coronary intervention (PCI) despite its class II recommendation in most patients with MVD.[3] The use of PCI in these patients may be partially explained by patient preference, anatomic factors, or comorbidities that preclude surgical candidacy.[4] Technical advancements in PCI and recent studies comparing PCI with CABG may justify multivessel PCI as a reasonable alternative for certain patients.[5–8] Moreover, in the setting of cardiogenic shock (CS), most guidelines tend to support the use of PCI as a therapeutic alternative to CABG in this setting.[2,9]

CS is a result of end-organ hypoperfusion owing to left ventricular (LV), right ventricular, or biventricular myocardial dysfunction resulting in systolic and/or diastolic myocardial pump failure.[10] Acute myocardial infarction accounts for approximately 75% of all patients with CS.[11,12] CS complicates 8.6% of ST-segment elevation myocardial infarctions (STEMI)[13] and 2.5% of non-STEMI,[14] is associated with a 60% to 70% mortality rate, and remains the leading cause of death in patients hospitalized with myocardial infarction in the era of reperfusion.[15] The only therapy found to improve outcomes in this

Division of Cardiology, University of Washington, School of Medicine, 1959 Northeast Pacific Street, Seattle, WA 98195, USA

* Corresponding author. Division of Cardiology, University of Washington Medical Center, 1959 Northeast Pacific Street, Seattle, WA 98103.

*E-mail address:* lombaw@cardiology.washington.edu

Intervent Cardiol Clin 6 (2017) 407–416
http://dx.doi.org/10.1016/j.iccl.2017.03.009

patient group remains revascularization, particularly complete revascularization.[9] However, given the potential negative outcomes associated with PCI, especially in the setting of shock (increased risk of stent thrombosis, ongoing ischemia, contrast-induced nephropathy, and longer radiation exposure[16]), multivessel revascularization during the initial presentation of CS continues to remain an infrequent practice despite its broad acceptance in the literature. Further, although the highest risk cases have the greatest incremental mortality benefit from treatment, they are simultaneously the least desirable to treat owing to the increased risk of adverse outcomes. This likely leads to errors of omission with patients being less likely to undergo cardiac catheterization in the setting of CS.

## CARDIOGENIC SHOCK

CS is defined by hemodynamic and clinical parameters. Hemodynamic parameters include persistent hypotension (systolic blood pressure <80–90 mm Hg or mean arterial pressure 30 mm Hg lower than baseline) for longer than 30 minutes, a cardiac index of less than 1.8 L/min/$m^2$ without support or less than 2.0 to 2.2 L/min/$m^2$ with support, and elevated filling pressures (LV end-diastolic pressure >18 mm Hg or right ventricular end-diastolic pressure >10–15 mm Hg). Clinically, signs and symptoms of hypoperfusion (ie, cool extremities, nausea, decreased urine output, and/or altered mental status) help to diagnose CS.[10] Decreased perfusion and end-organ dysfunction leads to lactic acidosis, catecholamine and neurohormonal release, along with activation of systemic inflammatory and coagulation cascades. This eventually results in a downward spiral (Fig. 1) with further myocardial depression and hypoperfusion.[17,18]

CS presents with a wide clinical spectrum ranging from "preshock" (significant risk of developing CS), "mild" CS (responsive to low-dose inotropes/vasopressors), "profound" CS (responsive to high-dose inotropes/vasopressors and intraaortic balloon pump [IABP]), and "severe refractory" CS (unresponsive to high-dose inotropes/vasopressors and IABP). The aim is to restore adequate perfusion and prevent end-organ dysfunction thus breaking the downward spiral of untreated CS. Given that many patients with CS present with acute coronary syndromes (ACS), these diagnoses and their pathologies are inextricably linked. Moreover, a large majority of these patients also have MVD as their etiology for CS.[19]

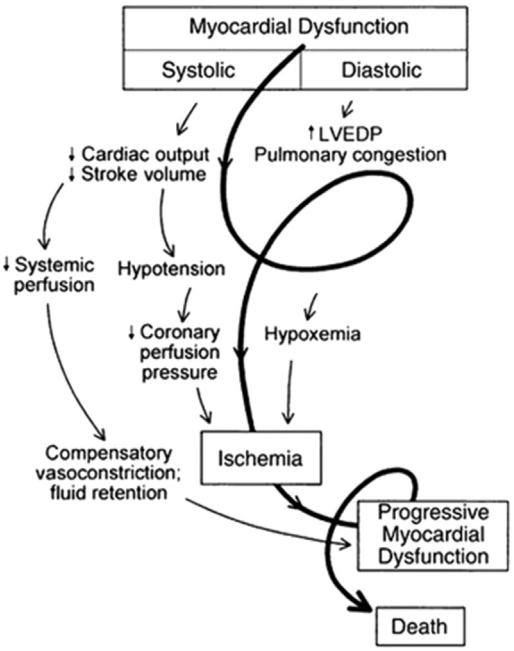


Fig. 1. The downward spiral in cardiogenic shock. LVEDP, left ventricular end-diastolic pressure. (*Reprinted from* Hollenberg SM, Kavinsky CJ, Parrillo JE. Cardiogenic shock. Ann Intern Med 1999;131:49; with permission.)

### Patients with Multivessel Disease Presenting with Acute Coronary Syndrome

Patients with MVD can be divided into 2 broad categories: those presenting with and those presenting without concurrent CS. Those patients with ACS, multivessel CAD, and also CS have a class I guideline recommendation from both American and European cardiac societies for CR (either surgical or percutaneous). Data supporting these recommendations were initially shown in the SHOCK trial (Should We Emergently Revascularize Occluded Coronaries for Cardiogenic Shock) and more recently in the IMPRESS in Severe Shock (IMPella vs IABP REduces mortality in STEMI patients treated with primary PCI in Severe cardiogenic SHOCK) trial. Those patients with ACS and multivessel CAD who do not present in CS are addressed by the PRAMI (Preventive Angioplasty in Acute Myocardial Infarction) and CvLPRIT (Complete vs Lesion-Only Primary PCI) data, currently with a class IIb recommendation in the guidelines for CR.[2,9]

The SHOCK trial, published in 1999, provided a modicum of clarity regarding the revascularization of patients presenting with CS. This study was a randomized trial comparing 2 treatment strategies—emergency revascularization and