EDITORIAL COMMENT

Right Versus Left Ventricular Shock

A Tale of Two Ventricles*

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Fifty years ago, the management of acute myocardial infarction (MI) consisted of prolonged bed rest and futile observation of electrical and mechanical complications, resulting in 40% mortality (1). Over the ensuing five decades, proactive interventions, including coronary care units, defibrillators, temporary pacemakers, pharmacologic support, and reperfusion therapy, slowly whittled mortality to the gratifying 2% achieved in the present era of stents and potent antiplatelet agents (2,3). Although patients developing cardiogenic shock continue to suffer excess mortality, the landmark SHOCK trial (4) was the first randomized study of acute MI to document that early reperfusion exerts beneficial effects on survival in patients with left ventricular (LV) shock. In this issue of the Journal, Jacobs et al. (5) report the fate of patients with predominant right ventricular (RV) shock entered into the registry component of the SHOCK study (4). In order to interpret the results of this study, it is important to draw distinctions between cardiogenic shock attributable to predominant acute RV ischemia (RVI) compared with that resulting from LV infarction. These two forms of hemodynamic compromise differ dramatically with respect to pathophysiology, natural history, and management.

Pathophysiology of hemodynamic compromise with right heart ischemia. Right heart ischemia occurs in 40% to 50% of patients with acute transmural inferior-posterior LV infarction, and its presence is associated with striking increases in the incidence of hemodynamic compromise, arrhythmias, and in-hospital mortality (6,7). Although hemodynamic manifestations develop in less than half of cases, RVI may result in cardiogenic shock characterized by disproportionate elevation of right heart filling pressures and hypotension-low output despite preserved LV systolic function (6).

The pathophysiologic mechanisms contributing to hemodynamic compromise with severe RVI are now well documented (6,8–13). Right coronary artery (RCA) occlusions proximal to the RV branches reduce RV free wall perfusion, resulting in depressed global RV performance, which diminishes transpulmonary delivery of LV preload, leading to decreased cardiac output despite intact LV contractility. Acute RV dilation results in elevated intrapericardial pressure, which, together with increased RV diastolic pressure shifting the interventricular septum toward the volume deprived left ventricle, conspires to further limiting LV filling.

There are important compensatory and aggravating factors that modulate the hemodynamic manifestations of RVI. When RV free wall contraction is depressed, RV systolic pressure is generated by LV septal contractions mediated through mechanical displacement of the septum into the RV cavity (9–12). The critical compensatory contributions of LV septal contraction to global RV systolic performance are emphasized by the deleterious effects of depressed LV septal function (6). Augmented right atrial (RA) contraction is another important compensatory mechanism that enhances RV performance and optimizes cardiac output. Loss of this booster pump function, due to atrioventricular dyssynchrony or ischemic depression of RA contractility resulting from very proximal RCA occlusions, further impairs RV performance and is associated with more severe hemodynamic compromise and higher mortality (9,12).

Differential responses of the ventricles to ischemia and reperfusion. The right and left ventricles differ in their architecture, mechanical performance, metabolism, and coronary blood flow (6). Thus, it should not be surprising that they differ markedly in their oxygen supply-demand characteristics and, accordingly, their responses to ischemia and reperfusion. Although acute ischemic RV dysfunction may result in hemodynamic compromise associated with higher in-hospital morbidity and mortality, most patients manifest spontaneous early hemodynamic improvement and later recovery of RV function, even in the absence of reperfusion of the infarct-related artery (10,14). Furthermore, global RV performance typically recovers over several weeks, with subsequent return of RV ejection fraction to near normal levels within 3 to 12 months. Moreover, chronic unilateral right heart failure secondary to RVI is rare. Observations from experimental animal studies document that the spontaneous recovery of RV function despite chronic RCA occlusion is attributable to the more favorable oxygen supply-demand characteristics of the RV in general and the beneficial effects of collaterals in particular (10). This dramatic spontaneous recovery of RV function and trivial infarction contrasts sharply with the response of the left ventricle to equivalent ischemic insults.

The salutary effects of timely reperfusion on myocardial function and infarct size in the ischemic left ventricle are well documented. Given the more favorable oxygen supply-demand characteristics of the right ventricle and the strength of observations supporting the notion that the
preponderance of acutely dysfunctional RV myocardium is viable, it would be expected that reperfusion would have salutary effects on recovery of RV function and clinical outcome. Observations from our laboratory have demonstrated that successful mechanical reperfusion of the RCA (including the major RV branches) results in prompt improvement in and later complete recovery of RV performance and in hemodynamics associated with excellent clinical outcome (11,15,16). In contrast, reperfusion failure is associated with lack of recovery of RV compromise, refractory hemodynamic compromise, and higher inhospital mortality (16).

In aggregate, these results support the concept that, under conditions of ischemia and reperfusion, the right ventricle is more resilient than the left ventricle. Moreover, the term RV “infarction” is to a great extent a misnomer, for the acutely ischemic dysfunctional right ventricle appears to represent predominantly viable myocardium, which responds favorably to successful reperfusion, even late after the onset of occlusion.

**RV versus LV shock.** Acute LV cardiogenic shock is characterized by pulmonary edema and low output-hypotension attributable to LV systolic and diastolic dysfunction resulting from acute ischemia-infarction often superimposed on prior infarction (2). In contrast to the resilience of the ischemic right ventricle, LV function rarely improves spontaneously and recovery is often disappointing even after successful reperfusion. Based on these considerations, it would be expected that the salutary effects of reperfusion interventions on survival would be greater in patients with RV shock compared to those with LV shock. In this issue of the Journal, Jacobs et al. (5) compare the fate of patients with predominant RV versus LV shock entered into the registry component of the SHOCK trial. The main component of the landmark SHOCK trial consisted of a multicenter, randomized study of early revascularization versus initial medical therapy in patients with LV shock complicating acute MI; results of that study showed that early revascularization lowered six-month mortality (4). The present report of the registry arm (933 shock patients ineligible for the main trial or eligible but not randomized) compared outcomes in 49 (5.3%) patients with predominant RV shock to 884 (94.7%) others with LV shock. Results of this study demonstrate that, compared with patients with LV shock, those with RV shock were younger and had a lower prevalence of both multivessel disease and prior MI. Despite this more favorable clinical profile, there were similar high mortality rates with both RV and LV shock (53% vs. 61%, p = NS), with revascularization imparting equivalent modest survival benefits to both groups.

Given the wealth of previous data documenting the resilience of the ischemic RV and the beneficial effects of reperfusion on recovery of right heart performance, hemodynamics, and survival, the high mortality in the present RV shock group and the lack of a differential survival benefit with reperfusion compared with the LV shock group is disappointing. However, in order to interpret these findings, there are several methodologic issues to consider. Cardiogenic shock is a difficult problem to subject to the rigors of a randomized, multicenter study, thereby emphasizing the impressive accomplishments of the SHOCK trial in demonstrating the beneficial effects of early revascularization in such patients (4). The present nonrandomized registry cases were identified by discharge diagnosis-related group (acute MI complicated by shock); therefore, evaluation and treatment were not controlled. Right ventricular ischemia was established as a “clinical diagnosis” based on electrocardiographic, echocardiographic, hemodynamic, or angiographic parameters. Unfortunately, echocardiographic data (the “gold standard” for delineation of the presence and extent of RV dysfunction) were not provided; hemodynamic evaluation by right heart catheterization was performed in only 61% of cases, and determination of LV function was available in only one-third of cases. Therefore, the magnitude of RV and LV dysfunction are difficult to discern. Selection bias may have influenced the results of this study. It is noteworthy that of 993 patients enrolled, only 49 (5.3%) constitute the RV shock group. Because the incidence of anterior and inferior MI are nearly equivalent and approximately 25% of those with inferior MI develop hemodynamic compromise, the number of RV shock patients is unexpectedly low. In a similar regard, many patients with RVI rapidly stabilize with volume, pharmacologic support, and successful revascularization and enjoy favorable outcomes; such cases may have been underrepresented in the present study. Furthermore, the hemodynamic characteristics of the RV shock group also suggests potential selection bias toward the most severely decompensated patients. The observed striking elevations of RA pressure (mean = 23 mm Hg) reflect the most extreme end of the hemodynamic spectrum of RVI and are consistent with a subset of patients many of whom likely suffered concomitant severe right atrial as well as RV ischemic dysfunction, a condition typically attributable to very proximal RCA occlusions and associated with more severe hemodynamic compromise than in RVI without right atrial involvement and excess mortality (9).

It is important that the present disappointing revascularization results be interpreted within the context of the study time frame (1993 through 1998), a period during which the more widespread application of primary angioplasty together with the advent of stents and potent antiplatelet agents resulted in dramatic improvement in reperfusion success. It should be noted, as well, that previous studies of RVI documenting the salutary effects of reperfusion on clinical outcome included all patients with RVI regardless of hemodynamic status, not just those, as in the present study, with shock. Furthermore, previous studies documenting the benefits of reperfusion on the ischemic RV have emphasized the importance of restoration of flow to the main RCA, as well as the major RV branches. In contrast, in the present RV shock group, angiography was performed in 63% of
patients, only 49% underwent percutaneous transluminal coronary angioplasty, and intraaortic balloon pump support was employed in just half of cases. Additionally, thrombolitics were administered in half of these RV shock patients, who are at higher risk of thrombolytic failure attributable to proximal RCA occlusion with extensive clot burden and impaired coronary fibrinolytic delivery owing to hypotension (17,18). Unfortunately, because angiographic data are not provided regarding culprit vessel flow before or after intervention, it is not possible to determine thrombolytic or angioplasty success rates. If the present registry study achieved reperfusion rates similar to those observed in the randomized SHOCK trial (70% percutaneous transluminal coronary angioplasty success), the present revascularization results would be difficult to extrapolate to MI patients in the present era, in whom primary angioplasty achieves TIMI 3 flow in up to 98% of cases. In aggregate, these considerations limit firm conclusions regarding the benefits of successful reperfusion in patients with RVI and hemodynamic compromise.

Despite these methodological considerations, the present observations are important and have implications for patients with RVI. These findings are consistent with and support those of prior studies documenting that patients with RVI, particularly those with the severe hemodynamic compromise, are at risk for adverse outcome. Based on the strength of previous observations documenting the resilience of the ischemic right ventricle and demonstrating the beneficial effects of primary angioplasty on RV performance and clinical outcome, reperfusion should be considered as a fundamental pillar of therapy in patients with RVI, particularly in those with severe hemodynamic compromise.

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REFERENCES


