The final common pathway

D.R. Holmes*

Mayo Clinic, School of Medicine, 221 Fourth Avenue SW, 55905 Rochester, MN, USA

Received 26 August 2002; accepted 28 August 2002

See doi:10.1016/S1095-668X(02)00429-3, for the article to which this editorial refers.

Cardiogenic shock remains the final common pathway to death in a substantial number of patients with acute myocardial infarction. We have learned a great deal about this syndrome which at its bottom line is characterized by a state of severe tissue hypoperfusion.

Much of what we know has come from single centre observational experiences, from multicentre registries, from subsets of patients in randomized trials of acute myocardial infarction therapy, and now from one specific trial of cardiogenic shock.1–4 Data from these sources have advantages as well as disadvantages. A prominent problem has been patient selection. For some of these trials patients with profound shock have been excluded which makes interpretation difficult. This issue of patient selection is perhaps best illustrated by the quote from Galen ... ‘all who drink of this remedy recover in a short time, except those whom it does not help, who all die. Therefore, it is obvious that it fails only in incurable cases’. Population-based data such as that from the TRACE Registry overcome some of the aforementioned disadvantages, although not all of them.

We have learned about definition, pathophysiology, epidemiology, clinical setting and outcome.

1. Cardiogenic shock has been variably defined but includes hypotension with a systolic blood pressure <90 mmHg or a reduction in blood pressure >30 mmHg below basal levels, adequate filling pressures, but systemic hypoperfusion with altered mental status, oliguria and cool extremities either singly or in combination.
2. We know that typically cardiogenic shock arises from the loss of a large amount of myocardium often >35–40% of the left ventricle. In some patients, this may result from a single large infarction, while in others from cumulative damage from prior infarctions or recurrent infarction after the initial event. There are other contributors which are variable, including extension of infarction, reperfusion injury, variable neurohumoral responses and mechanical complications.5
3. We know the setting in which cardiogenic shock occurs which is typically during acute myocardial infarction. The incidence has varied but is typically 6–9% and has been quite stable over the course of time. We know that although usually ST-segment elevation myocardial infarction is present and most often involves the anterior wall, that non-ST-segment elevation myocardial infarction may be present in approximately 15–30% of patients.6
4. Finally, we know that identification of shock portends a grim prognosis. In patients with shock, intensive measures are required.7–12 In general, more intensive measures are associated with improved outcome. In multiple series, both observational as well as the single randomized clinical trial, early or urgent revascularization has been associated with improved outcome. However, even with urgent revascularization, 30-day mortality is 30–40% so there is great room for improvement.

Against this background knowledge base, what does the data from the TRACE study group tell us.13
In this nationwide prospectively collected registry, the incidence of shock was 6.7%, similar to what has been found in other studies and appears to be representative of modern practice. The group of patients missing are those who died with shock before they could reach the hospital. This study importantly extends our knowledge of the timing of shock; of the 444 patients with shock, 59% developed it early within the first 48 h of presentation, but 30% developed it ≥5 days after the index infarction. The timing of the shock was of great importance in terms of its association with outcome. Patients developing shock within the first 48 h had a 30-day mortality of 45% vs those who developed it later on, in whom the mortality was >80%. There were important differences between these two patient groups—late shock patients had more reinfarction, more diabetes mellitus and more congestive heart failure; significantly, they had received lytic therapy less frequently. This is important because it has been documented in some series that thrombolysis may decrease the incidence of development of cardiogenic shock related to myocardial infarction.

An area of great interest has been the effect of revascularization either with percutaneous coronary intervention or with coronary bypass graft surgery. The TRACE study evaluated patients admitted from 1990–1992. These patients were not treated invasively so we cannot make any inference as to the effect of revascularization on mortality. This study, however, does offer significant insight into the long-term outcome of these patients. Although the number of patients is small, review of Figs. 1 and 2 documents the incredibly dismal outlook, particularly in those patients who develop shock late. As the authors note "in absolute numbers, only 3 and 1, respectively, of the patients with intermediate or late cardiogenic shock development were alive after 5 years". Reviewing the figures documents the inexorable decline with continuing hazard. This is somewhat different from other series, e.g. GUSTO I, in which the 1-year survival after hospital discharge was substantially better than seen by the TRACE investigators. Whether this difference was a function of length of follow-up, more intensive medical therapy, patient selection for entry or utilization of a more aggressive invasive approach remains unclear.

The bottom line remains that cardiogenic shock is often the final common pathway to a fatal outcome. Even with the most modern therapy, as the TRACE investigators identify 'medical therapy alone does not result in an acceptable outcome for patients with cardiogenic shock complicating acute myocardial infarction'. While revascularization strategies have been found to improve outcome, we still have a ways to go. It remains works in progress.

References

