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Editorial

Upscaling cardiac assist devices in decompensated heart failure: Choice of device and its timing

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ABSTRACT

Advanced heart failure is a heterogeneous condition unified by a very high mortality unless right treatment is instituted at the right time. The first step is understanding the mechanism leading to instability: hemodynamic or ischemic. Right kind of therapy; drugs (ionotropic) or IABP or other cardiac assist devices should be chosen according to mechanism of insult as well as degree of insult. Drugs such as ionotropes are effective only in very early course but if the decompensation has progressed beyond a certain point device such as IABP may be effective but again only early in the course when CPO? 0.6. Beyond a certain point, even IABP may not be effective: here only Impella (2.5, CP or 5) or Tandem Heart may be effective. However, beyond a certain point CPO < 0.53, even these devices may not be effective. Thus crux of the matter is choice of a right device/drug and timing of its institution.

1. Introduction

Acute decompensated heart failure represents a heterogeneous group of the cardiac conditions with some of the worst acute outcomes among all medical conditions. The etiology ranges from idiopathic pump failure, to mechanical causes, ischemic etiology or during the course of high-risk PCI (Table 1). The reason for decompensation originates either in sudden hemodynamic compromise or ischemic damage but in most cases there is a simultaneous occurrence of both ischemic damage and hemodynamic compromise. However, the relative contribution of these two mechanisms may differ in different conditions. While ischemic damage is most important contributor in high-risk PCI, and acute coronary syndromes; NSTEMI, STEMI and cardiogenic shock, hemodynamic compromise is the most important contributor in other types of decompensations. In any case, it is a vicious cycle: one leading to another (Fig. 1). The underlying mechanism contributing to decompensation is very important to recognize, because treatment depends on ability to address the relevant mechanism.

2. Mechanism of destabilization

There are two mechanisms of destabilization: ischemic damage and hemodynamic compromise.

2.1. *Ischemic damage*

Reduced oxygen delivery to the heart is essentially a question of "demand supply mismatch". In other words ischemic damage happens when the coronaries are unable to deliver enough blood as required by the myocardium. Thus this mismatch can happen in two ways (Table 2):

1. **Inadequate oxygen delivery**: (i) Reduced coronary blood flow due to atherosclerosis, plaque rupture, and thrombotic occlusion or any other cause leading to compromise of blood flow. Technically it is calculated as the difference between diastolic (mean) coronary arterial BP – LVEDP. Thus not only reduced blood flow but even raise in LVEDP can cause a situation of inadequate oxygen delivery. (ii) Direct inadequacy of oxygen delivery in situations like

Table 1 – Etiology of advanced decompensated heart failure.

1. Idiopathic
2. Pump failure: myocarditis, hypertension, alumunium phosphide poisoning
3. Mechanical complications: valve stenosis and regurgitations
4. Ischemic: NSTEMI, STEMI, cardiogenic shock
5. High-risk PCI

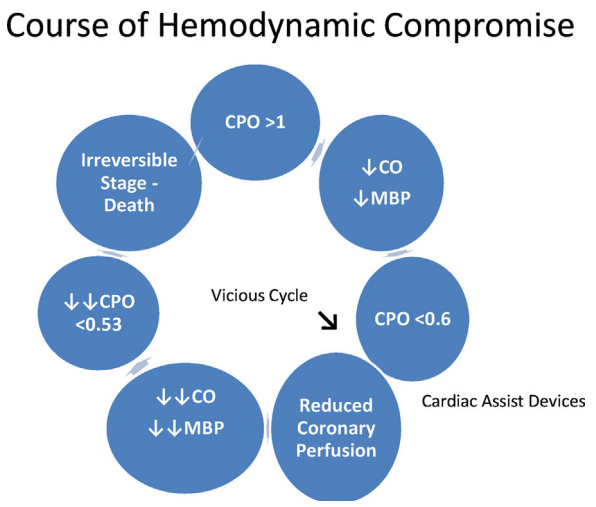

Fig. 1 – The course of hemodynamic compromise.

anemia or pulmonary congestion and edema can also worsen this process.

2. **Increased myocardial oxygen demand**: Increased heart rate, contractility, preload, after-load and muscle mass can all increase the oxygen consumption. However, the most important co-relate of myocardial oxygen demand is pressure volume area (PVA). A shift of this curve to right increases the oxygen demand and destabilizes, whereas a shift to left reduces it. Thus any cardio-protective mechanism, be it drugs or mechanical assist devices, essentially shifts this curve to left. Paradoxically, ionotropes and other stimulants (although they increase mean blood pressure (MBP) initially) push this curve to right. The various mechanisms to decrease ischemic insult and push the curve to left are given in Table 3.

Table 2 – Mechanism of decompensation.

Mechanism of decompensation	
Ischemic damage	Hemo-dynamic compromise
• Poor oxygen delivery Reduced coronary blood flow: difference between diastolic (mean) coronary arterial BP – LVEDP	• Cardiac power product = CO × MBP/451
• Increased myocardial oxygen demand	• CPO is direct co-relate of end-organ perfusion

Table 3 – Modalities to decrease the ischemic damage.

How to reduce ischemic insult	
Increase oxygen delivery	Reduce myocardial oxygen demand
• Improve coronary blood flow (difference between diastolic coronary arterial BP – LVEDP)	• Decrease heart rate
• Deliver oxygen directly	• Decrease contractility
• Administer food products	• Reduce preload
	• Reduce afterload
	• Reduce muscle mass
	• Shift pressure volume area (PVA) curve to right

2.2. *Hemodynamic compromise*

This is the second and more obvious mechanism of destabilization manifest as not only symptoms such as weakness, sweating and even collapse but also drastic fall in BP. It correlates with both forward delivery of blood as well as pressure head. Technically, it is measured as cardiac power output (CPO), which is derived from the equation:

$$CPO = CO \times MBP/451$$

A value <0.6 is indicative of hemodynamic compromise, whereas a value <0.53 is incompatible with life. As can be seen, there is a very small ''window of opportunity'', once hemodynamic compromise starts, and the management has to be instituted rather quickly and effectively.

3. Management of ischemic and hemodynamic support

Be it initial ischemic damage or hemodynamic compromise, the decompensation ensues with fall in CPO < 1. For a very short duration of time, when the CPO hovers around 1, the patient may be befitted by use of drugs, which increase the cardiac output (milrinone/amrinone or levosimendan) or increase systemic MBP (intravenous ionotropes). However, very soon this window of opportunity passes away and the use of these drugs may actually become counter-productive (Fig. 2).

3.1. *Drugs improving cardiac output*

Milrinone/amrinone and levosimendan act by increasing the cardiac output (or at least by preventing a fall in CO) predominantly by reducing the after-load.[1] However, these drugs paradoxically worsen the energy balance of the heart, by increasing the oxygen consumption; milrinone more than levosimendan.[2] Thus the overall benefit of this strategy is very small and that too very early in the course of decompensation. Further, when ischemic damage is the initial etiology (by worsening the energy kinetics) these drugs may not benefit at all.