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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.

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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):

- 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, aluminium phosphide poisoning
3. Mechanical complications: valve stenosis and regurgitations
4. Ischemic: NSTEMI, STEMI, cardiogenic shock
5. High-risk PCI

Course of Hemodynamic Compromise

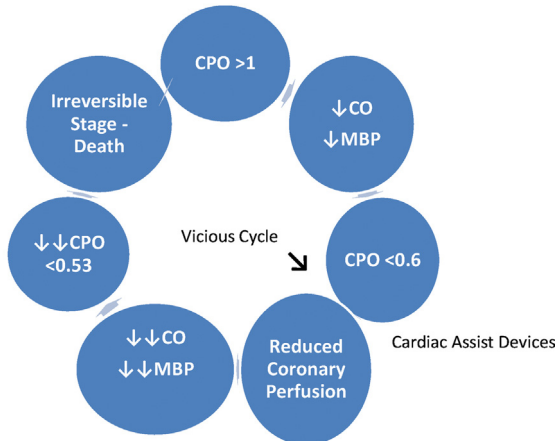


Fig. 1 – The course of hemodynamic compromise.

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
<ul style="list-style-type: none"> • Poor oxygen delivery Reduced coronary blood flow: difference between diastolic (mean) coronary arterial BP – LVEDP • Increased myocardial oxygen demand 	<ul style="list-style-type: none"> • Cardiac power product = $CO \times MBP/451$ • 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
<ul style="list-style-type: none"> • Improve coronary blood flow (difference between diastolic coronary arterial BP – LVEDP) • Deliver oxygen directly • Administer food products 	<ul style="list-style-type: none"> • Decrease heart rate • Decrease contractility • 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.

Course of Hemodynamic Compromise

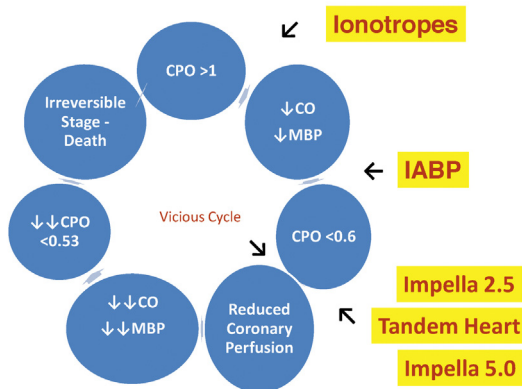


Fig. 2 – Timing of strategies providing hemodynamic support.

3.2. Role of ionotropes

The major mechanism of effectiveness of these drugs is by elevation of BP (MBP) but the other component of CPO i.e. cardiac output is not affected much (as there is no increase in stroke volume), and so overall CPO is only marginally elevated. On the other hand, \uparrow LVEDP caused by these drugs worsens ischemia and thus the balance of action is that on short term they may transiently stabilize hemodynamics at the cost of worsening ischemia, but with long-term deleterious effects. Further, this therapy may be associated with complications, including arrhythmia, worsening ischemia, etc. Understandably, when the cause of decompensation is ischemic, these drugs are practically useless.

3.2.1. Role of IABP

In situations, where ischemic mechanism is more important than hemodynamic e.g. high risk PCI hemodynamic compromise follows the ischemic insult but starts the vicious cycle causing more ischemic insult (because of decreased coronary perfusion). Here stabilization of ischemia is more important, or in other words, techniques, which increase myocardial oxygen delivery or reduce its requirements, are mandatory. IABP is one such a device. Here the mechanism of stabilization is indirect LV unloading and consequent reduction in LVEDP and augmentation of stroke volume (and therefore CO) and elevation of MBP. Improved coronary energetic is as a consequence of increased coronary delivery: increased diastolic coronary pressure (as a consequence of increased MBP) and reduced LVEDP. The effect on hemodynamics is also salutary: increased MBP and increased CO (CPO improves by 10%). Finally, as a result of unloading of LV, the oxygen requirement of heart also decreases by shifting the PVA curve slightly to left. Thus the effect of IABP is beneficial but modest. Further as shown in Fig. 2 the window of opportunity is small, before CPO reaches 0.6. Thus the timing is critical. A study by Mishra and co-workers has actually shown that once the destabilization becomes manifest very little can be done. On the other hand there is a role of prophylactic ally supporting with IABP in high risk situations.³ The bottom line is that to be effective, the degree of support both ischemic and hemodynamic

should co-relate with degree of insult and device chosen within appropriate time.^{4,5}

3.2.2. Tandem heart

The main mechanism of beneficial action is indirect LV unloading (connecting left atrium to common iliac artery) and the effect predominantly hemodynamic: increased MBP and increased CO (4 L/min) so that overall CPO improves by 80%. There is some ischemic benefit as well, which operates via indirectly unloading LV (and so reducing LVEDP). However, during shock, there is a paradoxical worsening of ischemia due to increasing oxygen requirement because of shifting of the PVA curve to the right. Further, there are several issues with this device: technical – the implantation technique is very complex and involves obtaining double access and trans-septal puncture (21F venous for LA and 15F arterial). It takes at least 30 min for implantation. Further, the risk of limb ischemia is the highest among all and so is the bleeding risk and requirement of transfusion. In addition, there is risk of residual ASD. It is contra-indicated in VSD, peripheral vascular disease and RV Failure. The magnitude of hemodynamic benefit is good, but it may worsen ischemia in shock. This device is effective, when the CPO is >0.53 (Fig. 2).

3.2.3. Impella device

Here, the mechanism of action is by direct unloading of LV into aorta and as a consequence, providing huge hemodynamic support – increased Mpp and increased CO (2.5 L/min with Impella 2.5 and 5 L/min with Impella 5), which leads to CPO improvement of 50% in Impella 2.5 and 100% in Impella 5. Further, there is ischemic benefit as well again operative via direct LV unloading, which shifts PVA curve to left even during shock as also reducing LVEDP. Thus the hemodynamic support provided is as follows: Impella 2.5 $<$ tandem heart $<$ Impella 5. The new Impella CP, which can be inserted percutaneously provides a CO of 4 L/min (like Tandem Heart) but which is coupled with a good ischemic benefit as well. Here the communication is between LV \rightarrow ascending aorta, which directly unloads LV (providing ischemic benefit as well). The technique involves single arterial puncture (Impella 2.5 – 13F, Impella CP – 14F). The technique is less difficult compared to Tandem Heart, the ease of implantation intermediate and it takes about 10 min for percutaneous procedure. The implantation carries a moderate risk of limb ischemia but a minimal risk of bleeding and requirement of transfusion. Its use is contra-indicated in presence of LV thrombus, VSD, severe AS and RV failure. Thus the only problem with this device is requirement of relatively large arterial access.^{6,7} This device is also effective when the CPO is >0.53 (Fig. 2).

3.2.4. ECMO

It is essentially a mini-cardiopulmonary bypass. It involves right atrial-aorta connection and leads to indirect LV unloading. It is useful in very sick patients.⁸⁻¹⁰

4. Conclusions

Advanced decompensated heart failure is a heterogenous condition with a high mortality. It is important to understand

the etiology, mechanism, and degree of decompensation. Choice of right strategy drug or device at the right time is critical in successful outcome.

Conflicts of interest

The author has none to declare.

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Sundeep Mishra

Department of Cardiology, AIIMS, New Delhi, India

E-mail address: sundeepmishraj@gmail.com

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