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Cardio Vascular Assessment in Vasoplegia Following Cardiac Surgery

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ABSTRACT

Severe vasodilatation during cardiac surgery has been known for many years. It is characterized by a low systemic vascular resistance, a high cardiac index, and a low blood pressure, which can be resistant to even high doses of vasoconstrictors. This condition is associated with a high mortality. After cardiac surgery, there can be cardiac dysfunction as well. However with a low systemic vascular resistance, the cardiac function curve would be shifted upwards and to the left, giving the impression of good contractility. The ejection fraction, which is the commonly used measurement of cardiac contractility, cannot therefore be relied upon for a correct assessment under these circumstances. This can affect fluid and inotrope management. Therefore a measurement which does not depend on the afterload, but measures pure myocardial contractility is required. Ventricular elastance is such a measure. A measurement of arterial elastance which reflects the flow characteristics and pulsatility of flow may be more appropriate than systemic vascular resistance to measure arterial tone. Ventricular-arterial coupling has to be maintained for optimum myocardial performance. The severe vasodilatation, the possible myocardial impairment and also its treatment with high doses of vasoconstrictors can affect the relationship between the ventricular and arterial systems. Therefore measurement of ventricular arterial coupling is also important.

Keyword

Arterial elastance, Ejection fraction, Vasoplegia, Ventricular arterial coupling, Ventricular elastance.

Introduction

Vasoplegic shock is associated with severe hypotension, and tissue hypoperfusion. Vasoplegic syndrome is a fairly frequent occurrence during cardio pulmonary bypass. The incidence ranges from 9% to 44% [1]. Vasoplegic shock is characterized by a low blood pressure, the requirement for vasopressors and fluids, a high cardiac output, and a low systemic vascular resistance [2-4]. This is similar to septic shock [5]. Severe vasodilatation with the above features is termed vasoplegia [6]. In this situation the inability to achieve adequate organ perfusion can lead to multiple organ failure and death.

Pathophysiological alterations in the cardiovascular system in vasoplegia after cardiac surgery

The main issues would be due to,

- Vasodilatation because of vasoplegia
- Myocardial depression may occur during cardiac surgery.

Vasodilatation

Extreme vasodilatation or vasoplegia causes persistent and severe hypotension.

The causes for vasoplegia are

- Activation of adenosine triphosphate sensitive potassium channels
- Activation of inducible forms of nitric oxide synthase
- Deficiency of vasopressin [3].

Fischer GW et al. [3] identified several aetiological factors, for the vasoplegic syndrome after cardio-pulmonary bypass.

- Pre-operative use of angiotensin-converting enzyme inhibitors
- Beta blockers
- Higher euro score
- Heart failure surgery
- Decline in Mean Arterial Pressure after initiation of Cardio-pulmonary bypass

Because of the vasodilation, there is a reduction in the total peripheral vascular resistance. With vasoplegia, despite, adequate

fluid resuscitation and a high cardiac index, there is a low blood pressure and a low systemic vascular resistance even with the administration of vasopressors [7].

Myocardial dysfunction after cardio-pulmonary bypass

Myocardial dysfunction can occur after cardio pulmonary bypass to various degrees. Myocardial impairment occurs because of ischaemia, reperfusion and the systemic effects of cardio pulmonary bypass [8,9].

Issues in the cardiac assessment in vasoplegic shock

As described above the two main cardiovascular dysfunctions are vaso dilatation and cardiac depression. These can have an impact on the assessment of cardiac function.

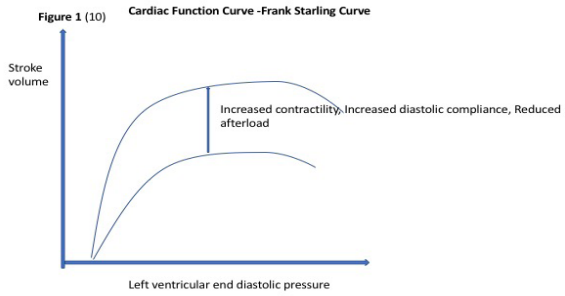


Figure 1: Cardiac function curve -Frank-Starling Curve.

The Frank Starling curve, demonstrates the changes in stroke volume in response to changes in end diastolic pressure. End diastolic pressure depends on end diastolic volume. As shown, when the volume is increased the output will also increase, but only to a certain point, after which the curve plateaus. Therefore any fluid challenge will increase the output, only if the heart is functioning on the ascending part of the curve. Volume resuscitation is a major component of management of shock [11,12]. Volume resuscitation should be done only in fluid responsive patients.

The Frank Starling curve is affected by the afterload and the inotropic state of the heart [10]. A decrease in the afterload would mean that the heart pumps against a lowered resistance and therefore a given volume would have an increased output when compared to a normal situation.

Therefore with the severe drop in afterload in vasoplegic shock, the Frank Starling curve will be shifted up and to the left [13]. This can occur even if the myocardium is dysfunctional. The severe drop in afterload may compensate for the reduced contractility of the myocardium. This would give the impression that cardiac function is satisfactory. The shift in the curve upward and to the left, will also indicate a greater fluid responsiveness. An associated right ventricular dysfunction will also show a false fluid responsiveness [11]. Inappropriate fluid resuscitation can occur under these circumstances. There may be a delay in diagnosing and treating myocardial impairment.

The cardiac function curve would therefore be an inappropriate method to assess cardiac function in vasoplegic shock.

This would also mean that the left ventricular ejection fraction may appear normal despite an actual depression of contractility. The left ventricular ejection fraction is not a reliable index of LV systolic function in septic shock [14]. This may be the situation in vasoplegic shock after cardiac surgery as well, where the pathophysiology is similar [15].

The left ventricular ejection fraction depends on contractility and afterload. When the afterload is low, ejection fraction could appear to be satisfactory as described earlier. However, with the use of vasoconstrictors, the actual cardiac contractility would become apparent when the mean arterial pressure is adequate.

A method of assessment of intrinsic myocardial contractility and arterial resistance would be required to properly assess the cardiovascular system in these circumstances. These measurements have to be independent of loading factors so that only the intrinsic myocardial function is measured.

The Pressure Volume Curve

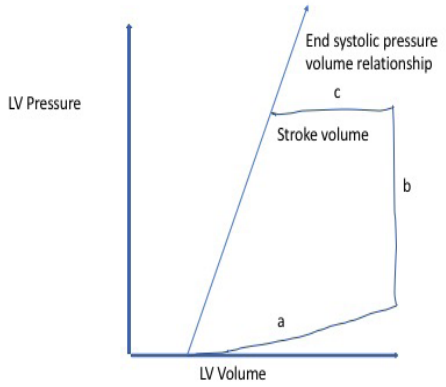


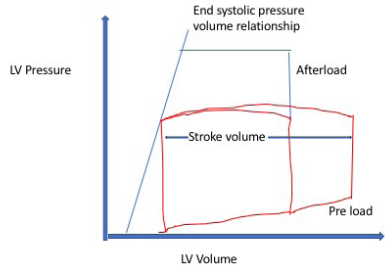
Figure 2: Left ventricular, Pressure-Volume curve.

The pressure volume curve of the left ventricle is shown in this simplified diagram (Figure 2). It shows the left ventricular pressure in relation to the volume contained within. With the left ventricular volume filling up from end systolic point, there is a slight rise in pressure (a). When left ventricular systole begins the aortic and mitral valves are closed, therefore the volume remains constant. (iso volaemic contraction). As the left ventricle at this stage is a closed chamber, left ventricular pressure rises steeply. (b) When the pressure exceeds the pressure keeping the aortic valve closed, the aortic valve opens (b) and the volume is ejected, with a further slight rise in pressure. The volume ejected is the stroke volume. (c) The line (a) is extrapolated to where it meets the volume axis. This point and the end systolic pressure point when joined will give the ventricular end systolic pressure volume relationship. The slope of the line is the left ventricular elastance [10,16].

As the pressure volume curve demonstrates, provided the state of inotropy remains the same, changing the preload or afterload, would not affect the end systolic pressure volume relationship and the ventricular elastance [10]. Therefore ventricular elastance would give an assessment of pure myocardial contractility which

is independent of loading factors.

Figure 3: Pressure-volume relationships.



The ventricular elastance is measured by dividing the end systolic pressure by the end systolic volume [17]. The end systolic volume is obtained by echocardiography. The average normal value is about 4.3 [18].

Afterload

As said before, the decrease in afterload is the main issue in vasoplegic shock. This is generally measured by the systemic vascular resistance. However this ignores the pulsatile nature of both pressure and flow. Therefore alternative measurements have been sought. Arterial elastance is such a measure [19].

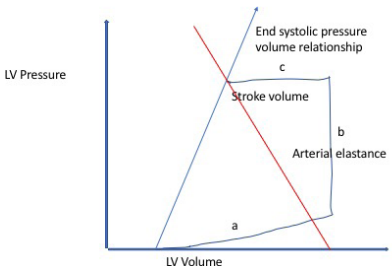
Fluid resuscitation on its own may not be sufficient to improve the perfusion pressure. The low afterload has to be corrected. Therefore the afterload has to be measured. It can be measured in a functionally useful manner by the

- Arterial elastance
- Dynamic arterial elastance/ Afterload reserve

Arterial Elastance

The arterial elastance takes into account the peripheral resistance, the vascular compliance, impedence and systolic and diastolic time intervals [19].

Figure 4: Arterial elastance.



In the pressure volume curve, the arterial elastance is obtained by a line joining the end systolic pressure point and the end diastolic point on the volume axis (Figure 4) [20].

The arterial elastance can be measured as a single measurement by dividing the end systolic pressure by the stroke volume [19]. Normal values are in the region of about 2.3- mmHg/ml 18).

Dynamic arterial elastance / Afterload reserve

This is another method to assess arterial tone, using pulse pressure variation and stroke volume variation during positive pressure ventilation [21-23]. This is measured by PPV/SVV and should approach unity. A ratio of <1 indicates vasodilatation and >1 vasoconstriction.

Ventricular-arterial coupling

An adequate cardiac output and a sufficient pressure are required to maintain organ perfusion. This is brought about by the interaction between the heart and the vascular system. The vascular system should be able to handle the blood pumped out by the heart [21]. This matching between cardiac function and the arterial system is termed ventricular -arterial coupling. It is an indicator of cardio vascular efficiency [21]. In the presence of matching or coupling, myocardial energy usage is optimal. Decoupling of the ventricular -arterial system can lead to myocardial failure.

Ventricular-arterial coupling is measured by the ratio between arterial elastance and ventricular elastance [24]. The system is said to be decoupled when this ratio is above 1 [21]. Guarracino et al. state that the assessment of the ratio between arterial elastance and ventricular elastance, during management of patients in shock, would be useful to guide therapy [21]. The measurement of the ventricular-arterial coupling during administration of vasopressors would prevent over correction of the arterial elastance.

Clinical Implications

The clinical implications of these issues can be summarized as

- Left ventricular ejection fraction as a measure of cardiac function may be misleading in the presence of vasoplegia. The actual cardiac dysfunction may be revealed only after the vasodilation has been corrected to a certain extent with a vasopressor [8].
- Intrinsic left ventricular function should be assessed, to correctly assess left ventricular function. This can be assessed with measurement of left ventricular elastance.
- The presence of vasodilation can be identified by measuring arterial elastance.
- Measurement of ventricular-arterial coupling is important to have optimum function and also to prevent further deterioration of cardiac function.
- When using vasopressors and inotropes it may be important to measure ventricular-arterial coupling, to keep it matched [21].

Complicating Issues

Ventricular- arterial coupling involves multiple physiological issues, which are not captured in the pressure-volume plane. (25). The arterial elastance does not reflect the pulsatile left ventricular load and is not dependent only on arterial properties [25]. In vivo, the end systolic pressure volume relationship is nonlinear [26]. The peak value of end systolic pressure volume relationship decreases with the left ventricular volume [27]. Baan et al. demonstrated that the end systolic pressure volume relationship has an afterload dependency [28]. These factors may have an effect on the accuracy of the measurements of ventricular and arterial elastance.

Conclusion

The cardio vascular involvement in vasoplegic shock after cardio-pulmonary bypass is severe vasodilatation and there maybe cardiac

dysfunction as well. The severe vasodilatation will influence the cardiac function curve and make the measurement of left ventricular ejection fraction inaccurate. Therefore a measurement of intrinsic ventricular contractility, such as left ventricular elastance is required. Measurement of arterial elastance and ventricular-arterial coupling will give a more complete and useful assessment, which will also be beneficial in terms of fluid resuscitation, the use of vasopressors and inotropes.

Although errors in the method have been identified, the measurement of ventricular and arterial elastance and the ventricular-arterial relationship, maybe useful clinically to assess ventricular function, the presence of vasoplegia and to guide therapy.

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