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REVIEW

To give or not to give fluid challenges!

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S U M M A R Y

Fluid challenges are used to improve cardiac output and oxygen delivery. This is done in the presence of hypovolaemia. Hypovolaemia is generally diagnosed on static haemodynamic parameters, such as Central Venous Pressure, Pulmonary Capillary Wedge Pressure etc. Only about half of patients administered fluid in this manner, have benefitted. Recently interest has been directed at functional haemodynamic parameters. These are based on the relationship between mechanical ventilation and venous return due to the cyclical changes in intra thoracic pressure. These cause variation in the stroke volume, systolic blood pressure and pulse pressure during the respiratory cycle. There are factors which can affect the accuracy and the interpretation of these parameters. Spontaneous respiration, the tidal volumes used to ventilate, PEEP, lung and chest compliance, heart rhythm, right ventricular function are these factors. Passive leg raising can be used in the presence of these or in doubt.

This article attempts to set out, how to determine whether a fluid challenge will improve the cardiac output, and also to identify the problems in arriving at that decision.

1. Haemodynamics and functional haemodynamics

Haemodynamics is the physiology concerned with the movement of blood and the forces and pressures associated with that circulation. Haemodynamics measures oxygen delivery to tissues. For this, static parameters such as Central Venous Pressure, Pulmonary Capillary Wedge Pressure, Systemic Vascular Resistance, Cardiac Output are measured. They do not tell us, however, whether increasing the volume status will improve the cardiac output and the oxygen delivery or compromise the chances of survival. Only about 50% of patients responded to fluid administration based on these parameters.[1] Functional Haemodynamics attempts to address this dilemma, with the use of dynamic parameters.[2] Therefore it is necessary to measure the haemodynamics to know the oxygen delivery and to monitor the functional haemodynamics to assess whether the oxygen delivery can be improved.[3]

1.1. Cardiac output

$$DO_2 = CI(Hb^*1.34^*SaO_2 + .003PaO_2)$$

DO_2-Oxygen delivery to tissues, CI-Cardiac Index, Hb-Haemoglobin concentration, SaO_2-Arterial oxygen saturation, PaO_2-Arterial oxygen tension.

The oxygen delivery depends on the cardiac output, amount of haemoglobin, the saturation and the dissolved oxygen.

The cardiac output is affected by,

1. The pre load, this is the volume status of the patient.
2. The myocardial contractility
3. The after load or the resistance against which the left ventricle pumps, which is measured by the systemic vascular resistance.

Of the above the Pre load or the fluid volume would be focused upon, as attempting to improve the fluid status, and thereby increasing the cardiac index and thus the oxygen delivery to tissues is very often the first line of management when it is required to improve the cardiac output.

The haemodynamic parameters available to measure the fluid status of a patient are,

Static parameters-A parameter measured under a single loading condition

- Pulmonary capillary wedge pressure
- Central venous pressure
- Right ventricular end diastolic volume
- Left ventricular volume
- Global end diastolic volume

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However these are all measurements taken under single loading conditions.

They, therefore do not indicate whether the patient is normo volaemic, hypovolaemic or over loaded. They are static parameters. These static indices of preload have a low predictive ability for hypovolaemia.[2,4] It has been shown that the CVP does not accurately indicate the preload and also does not predict fluid responsiveness.[2,10,16]

An inappropriate fluid administration, where the heart is unable to increase the cardiac output with a fluid bolus, can lead to oedema in both tissues and lungs causing further hypoxia. Therefore it becomes important to identify those patients who will have a beneficial effect with a fluid administration.[5] For this purpose functional haemodynamics are required.

Functional haemodynamics depend on the heart–lung interaction.

Dynamic parameters - Heart–lung interaction on preload indices

Variations in
- Stroke volume
- Systolic pressure
- Pulse pressure

With mechanical ventilation

With mechanical ventilation the pre load will change with each cycle and therefore, there will be cyclical variations in stroke volume, systolic pressure and pulse pressure.[18]

With mechanical ventilation in a patient with normal left ventricular function, during inspiration, the intra thoracic pressure rises. The venous return is therefore decreased, and cardiac output will fall. In expiration, the intra thoracic pressure falls and the venous return will increase, thereby increasing the cardiac output.[6] Because of this heart–lung interaction during mechanical ventilation, it is possible without administering extraneous fluid to obtain two points on the Frank-Starling curve. One, when the preload is decreased and the other when it is increased. The changes in the cardiac output will manifest as variations in Stroke Volume, Systolic Blood Pressure, and Pulse Pressure with inspiration and expiration. If these variations are present it means that the patient is on the steep part of the curve and if there is no variation on the plateau section. With a fluid challenge the cardiac output can increase only if the patient is on the steep part of the Frank-Starling curve.

As shown in the diagram (Fig. 1), in a person with normal cardiac function, who will be on the rising steep part of the curve, when there is a reduction in the venous return from A to B, the stroke volume will also fall from 1 to 2. This will manifest as a fall in stroke volume, systolic blood pressure or pulse pressure. In a person with impaired left ventricular function, who will therefore be on the plateau part of the Frank Starling curve, a corresponding change in venous return from C to D, will not show a reduction in stroke volume. There is hardly any difference between 3 and 4 in the figure. Therefore a patient with ventricular dysfunction, who will be on the plateau part of the curve will not show any changes in stroke volume, systolic blood pressure or pulse pressure.

The CVP measurement on the other hand, on such a patient, will just indicate the fluid volume of the patient, but not whether it is adequate or not. It will also not predict, as it is just one measurement under static conditions, how the patient will respond to a fluid challenge.[10] Marik et al. in a literature survey in 2008, have said the CVP does not indicate the blood volume or the fluid responsiveness.[8] Use of CVP measurements to assess whether or not a patient's cardiac output will increase significantly in response to an infusion of intravenous fluid cannot therefore be recommended.[9,10,19]

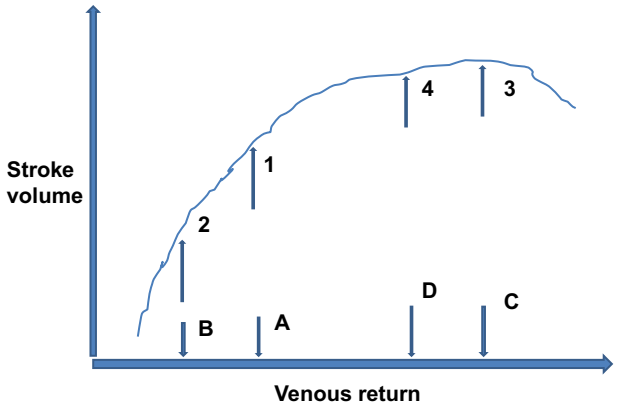

Fig. 1. Frank Starling curve.

Stroke volume variation can be easily observed with transoesophageal echocardiography.[5,7,11]

The picture shows the continuous wave, with transoesophageal echo, at the aortic valve. The variation is easily observed. The peak velocity at the aortic valve is an accurate indicator of predicting haemodynamic effects of volume expansion.[17] This is because the stroke volume is the product of the aortic valve area and the velocity time integral at the aortic valve. The aortic valve area can be assumed to remain constant during the respiratory cycle.[17] The peak velocity at the aortic valve is easily observed with transoesophageal echocardiography as shown in Fig. 2. There is a variation in the peak velocity (stroke volume) in Fig. 2.

During mechanical ventilation, at inspiration, the venous return falls thereby making the stroke volume less, and during expiration the venous return increases thus making the stroke volume more. This patient is therefore in the steep part of the Frank-Starling curve and will be able to increase the cardiac output following a fluid challenge. These patients are termed responders.[2]

This is a picture of the continuous wave transoesophageal wave form at the aortic valve of another patient (Fig. 3). As explained before, with mechanical ventilation, there is cyclical change in the venous return, however, there is no corresponding variation in the stroke volume in this patient. This means that this patient is on the flat part of the Frank Starling curve and is unable to increase the cardiac output in response to a fluid challenge. This is a non-responder.

The above two figures show the peak velocity, at the aortic valve, with mechanical variation. As discussed before, this is an indication of the stroke volume. The arterial blood pressure, when monitored with an arterial cannula and wave form will also show, whether the systolic blood pressure varies with mechanical ventilation. Responders and non-responders can be identified in this manner too.

As explained above heart-lung interactions enable two points to be determined on the Frank Starling curve, with the venous return at two different levels without the administration of extraneous fluid.

However this same heart-lung interaction is subject to interference by other factors. These will cause difficulties in interpretation and may have an effect on the accuracy of the estimations and assessments.

Respiratory system issues

1. Tidal volume
2. PEEP