FLUID RESPONSIVENESS IN CRITICALLY ILL PATIENTS

An Essay

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Presented By

Hussein Rabee Mohammed Ahmed

Under supervision of

Prof. Dr. Gamal Fouad Saleh Zaki

Professor of Anesthesia and intensive care
Faculty of Medicine, Ain Shams University

Dr. Hazem Mohamed Fawzi

Assistant professor of Anesthesia and intensive care
Faculty of Medicine, Ain Shams University

Faculty of Medicine
Ain Shams University
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INTRODUCTION

Fluid resuscitation for hypovolaemia is the mainstay of the medical management of critically ill patients, whether as result of trauma burns, major surgery or sepsis. Although recent studies have suggested that the timing of volume replacement deserves careful consideration, when it comes to selecting the resuscitation fluid clinicians are faced with a range of options. At one level the choice is between colloid or crystalloid solutions. Crystalloid are widely used having been recommended in a number of resuscitation guidelines and intensive care management **(Perel et al., 2009)**.

Over the last few years, the concept of fluid responsiveness has become popular in Europe and South America, likely because it is a pragmatic approach to fluid therapy. Indeed, we have a clear idea of the normal total blood volume (800–1,000ml/m^2), and of normal right and left ventricular end-diastolic volumes (90–110ml/m^2 and 60–80ml/m^2, respectively) in healthy subjects. However, it is much more difficult to determine which level of pre-load is optimal in an 'abnormal' situation, e.g. vasodilation induced by sepsis. Therefore, a practical approach to determine fluid therapy consists of detecting patients who will be able to turn fluid loading into a significant increase

in SV and cardiac output. Of course, clinical end-points of fluid therapy are usually different, e.g. increasing blood pressure or urine output, but will be achieved only if the physiological effect (an increase in SV and cardiac output according to the Frank-Starling mechanism) occurs first. If not, fluid administration is useless or even potentially harmful, e.g. leading to a worsening in pulmonary edema **(Michard et al., 2002).**

In spontaneously breathing patients (with or without mechanical ventilation), the predication of volume responsiveness can be a difficult challenge, In particularly in those whom have already been resuscitated in the preceding hours or days and when continuation of fluid infusion carriers risks of pulmonary edema. In the cases, static markers of cardiac preload are generally in the normal range are rarely helpful for determination of volume responsiveness. Since absolute measures of preload cannot be used effectively to assess volume responsiveness more dynamic tests need to be employed to improve the utility of these measure. Because of the presence of spontaneous breathing as indices of volume responsiveness that use heart-lung interaction such as respiratory variation in atrial pressure and in stroke volume are no longer reliable. Carful analysis of hemodynamic consequence of passive lag raising using real time aortic blood flow monitoring may be

helpful for predicting the beneficial effect of volume administration. Fluid challenge strategy can still be applied provided that clinicians carefully follow the recommended rules in term of the type of fluid, rate of infusion, clinical end-point and safety limits in order to minimize the risk of fluid overload **(Teboul et al., 2007).**

Mechanical ventilation induces cyclic changes in left ventricular (LV) stroke volume which are related to the expiratory decrease in LV preload due to the inspiratory decrease in right ventricular (RV) filling and ejection. There are recent clinical data demonstrating that respiratory changes in arterial pulse (or systolic) pressure and in Doppler aortic velocity (as surrogates of respiratory changes in LV stroke volume) can be used to detect biventricular preload dependence, and hence fluid responsiveness in critically ill patients **(Michard and Louis, 2000).**

Aim of the Work

Review the recent medical literature with respect to:

- Assessment of advantages and disadvantages of fluid therapy.
- Predict the ideal fluid choice in resuscitation.
- Prediction of fluid responsiveness in critically ill patients.
- Monitoring of volume expansion in critically ill patients.
- Study of the recent medical techniques to evaluate fluid responsiveness in critically ill patients.

Basic Cardiovascular Physiology

Determinants of Cardiac Performance:

The discussion of ventricular dysfunction, it is most often in reference to the left ventricle, however it is important to understand that the same basic principles apply to the right ventricle. The left and right sides of the heart exist in a series, and are therefore interdependent; in normal physiology, the right and left ventricle will have the same output **(Blitt et al., 1995).**

Cardiac function is the volume of blood pumped each minute, and is expressed by the following equation:

CO = SV x HR

Where:
CO is cardiac output expressed in L/min (normal ~5 L/min)
SV is stroke volume per beat
HR is the number of beats per minute.

(Blitt et al., 1995)

How Determinants of Cardiac Function Interact:

There are two ways to examine of looking at the relationship between the multiple determinants of cardiac

function, and how changes in the separate factors affect the overall cardiac output:-

1. **Frank – Starling Principle:**

The Frank–Starling law of the heart (also known as Starling's law or the Frank–Starling mechanism) states that the stroke volume of the heart increases in response to an increase in the volume of blood filling the heart (the end diastolic volume). The increased volume of blood stretches the ventricular wall, causing cardiac muscle to contract more forcefully (the so-called Frank-Starling mechanisms). The stroke volume may also increase as a result of greater contractility of the cardiac muscle during exercise, independent of the end-diastolic volume. The Frank-Starling mechanism appears to make its greatest contribution to increasing stroke volume at lower work rates, and contractility has its greatest influence at higher work rates. This allows the cardiac output to be synchronized with the venous return and arterial blood supply without depending upon external regulation to make alterations **(Costanzo and Linda, 2007).**

Physiology

As the heart fills with more blood than usual, the force of cardiac muscular contractions increases. This is a

result of an increase in the load experienced by each muscle fiber due to the extra blood load entering the heart. The stretching of the muscle fibres augments cardiac muscle contraction by increasing the affinity of troponin C for calcium, causing a greater number of actin-myosin cross-bridges to form within the muscle fibers. The force that any single cardiac muscle fiber generates is proportional to the initial sarcomere length (known as preload), and the stretch on the individual fibers is related to the End Diastolic Volume of the left and right ventricles. In the human heart, maximal force is generated with an initial sarcomere length of 2.2 micrometers, a length which is rarely exceeded in the normal heart. Initial lengths larger or smaller than this optimal value will decrease the force the muscle can achieve. For larger sarcomere lengths, this is the result of less overlap of the thin and thick filaments; for smaller sarcomere lengths, the cause is the decreased sensitivity for calcium by the myofilaments **(Mikhail et al., 2004).**

2. Cardiac and Vascular Function Curves:

These are simultaneous plots of cardiac output and venous return as a function of end diastolic volume or (right atrial pressure).

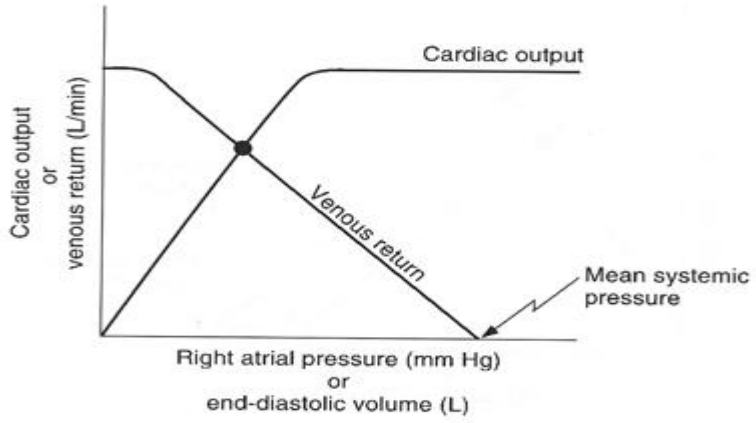

Figure (1): Simultaneous Plots of the cardiac and vascular function curves. The two curves cross at the point of equilibrium for the cardiovascular system **(Miller et al., 2000)**

- **Cardiac Function Curve**: This is simply the Frank-Starling curve for the ventricle showing the relationship of cardiac output as a function of end diastolic volume.

- **Venous Return Curve**: This is the relationship between blood flow in the vascular system (venous return) and right atrial pressure.

- **Mean Systemic Pressure**: This is the point where the venous return curve intersects the X axis. The mean systemic pressure reflects the right atrial pressure when there is 'no flow' in the system. At this point the pressure is equal throughout the circulatory system.

- **Equilibrium**: This is the steady-state where the two curves intersect; it reflects the point where cardiac output is equal to venous return.

(Miller et al., 2000)

Cardiac output can increase or decrease by altering the Frank-Starling curve, the venous return curve, or both; some predictions can be made by examining how the curves shift as the variables change **(Miller et al., 2000).**

Mean Systemic Pressure Changes: The mean systemic pressure is affected by blood volume as well as venous compliance. Changes in the mean systemic pressure will shift the vascular function curve left or right.

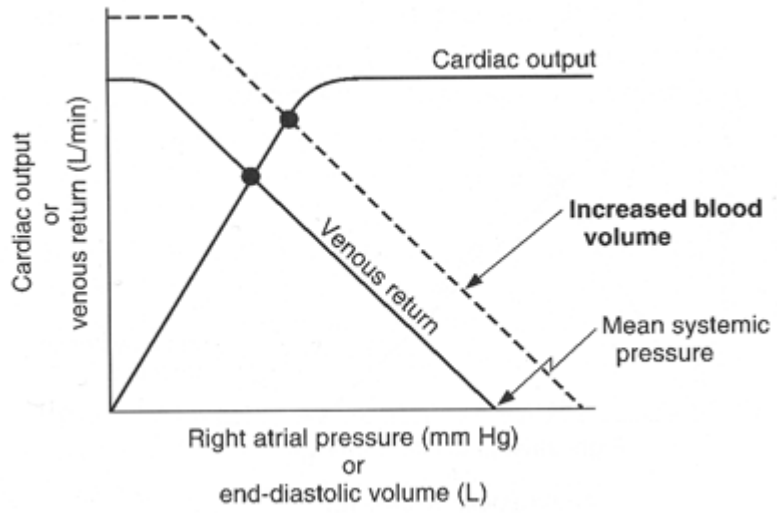

Figure (2): Mean Systemic Pressure Changes **(Miller et al., 2000)**

- Mean systemic pressure is increased by an increase in blood volume and/or a decrease in venous compliance (as shown above). This will act to shift the vascular function curve to the right, illustrating an increase in both cardiac output and right atrial pressure. Conversely, mean systemic pressure is decreased by a decrease in blood volume and/or an increase in venous compliance (not shown). This will shift the vascular function curve to the left, illustrating a decrease in both cardiac output **(C.O)** and right atrial pressure **(Miller et al., 2000).**

Inotropic Changes: Contractility is determined by various autonomic mechanisms and certain drugs (such as digitalis). Inotropic changes will alter the slope of the cardiac curve up or down (as discussed above).

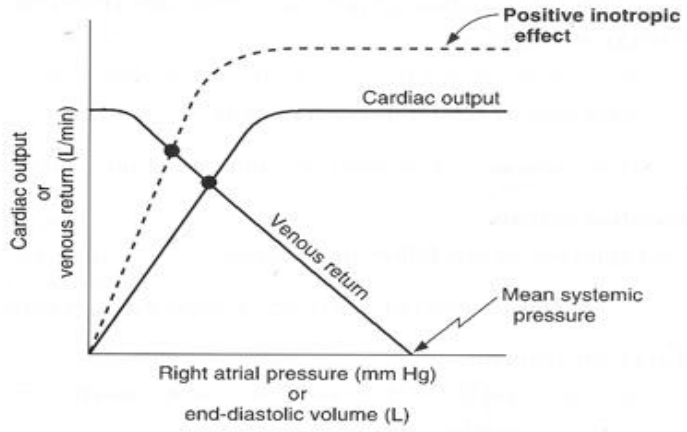

Figure (3): Intropic changes alter the slope of cardiac curve **(Miller et al., 2000)**

- Positive inotropic agents, such as digoxin, will increase contractility and therefore increase the cardiac output (as shown above). This new equilibrium point now reflects an increased cardiac output and a lower right atrial pressure (more blood is now being ejected from the heart with each beat).
- Negative inotropic agents have the opposite effect, decreasing contractility and cardiac output, and increasing right atrial pressure (not shown).

(Miller et al., 2000)

Total Peripheral Resistance Changes: (**TPR)** is determined by the resistance of the arterioles. Changes in **(TPR)** will change the slope of both the cardiac function curve and the venous return curve.

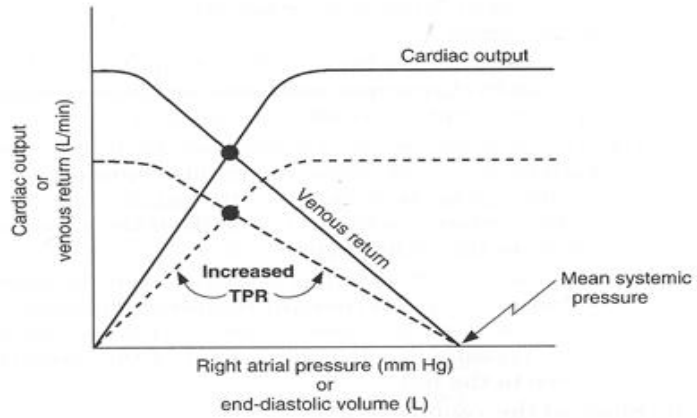

Figure (4): Changes in the total preipheral resistance change the slope of both cardiac function curve and the venous return curve **(Miller et al., 2000)**

- An increase in total peripheral resistance **(TPR)** (shown above) will cause blood to be retained on the arterial side of circulation and will increase the aortic pressure against which the heart must pump. This will act to shift both slopes downward. As a result of this simultaneous change, both the cardiac output and the venous return are decreased, however the right atrial pressure remains the same.

- A decrease in **(TPR)** (not shown) will allow more blood to flow to the venous side of circulation and will lower the aortic pressure against which the heart must pump. This will shift both slopes upward. Both cardiac output and venous return will be simultaneously increased; again, right atrial pressure will remain the same **(Miller et al., 2000).**

Fluid Resuscitation

Fluid distribution in the body

To understand fluid resuscitation, it is important to appreciate the distribution of the fluids in the body. In a normal human body, 60% of body weight is composed of water in males and 50% in females. Body water is further divided into intracellular (66%) and extracellular (34%) compartments. Included in extracellular compartment is the transcellular fluid. This comprises 2.5% of total body water and includes aqueous humor and fluids produced by the salivary glands, pancreas, tracheo-bronchial tree, cerebrospinal fluid and genitourinary tract **(Shahid and Mehra, 2009).**

Extracellular fluid is distributed in the intravascular compartment (25%) and in the interstitial compartment (75%). Utilizing a typical example, in a 70 kg male, the total body water **(TBW)** is 42L. Of the 42L, approximately 66% or 28L is in the intracellular compartment **(ICF)** and 34% (14L) is in the extracellular compartments **(ECF)**. The extracellular component is further divided into interstitial (75 % of **ECF** =10.5L) and intravascular (25% of **ECF**= 3.5L). Distribution of resuscitative fluid in the body depends on the solute concentration of the administered fluids. Resuscitative fluid with no solutes will be distributed in all fluid compartments. If 1L of 5%

dextrose **(D5W)** solution is administered, the total body water will increase to 43L and the intravascular volume will be increased by approximately 83 mL. Almost 2/3 of the fluid will distribute into the intracellular compartment. On the other hand, 1L of normal saline infusion increases the intravascular volume 250 mL and no fluid will be distributed into the intracellular compartment. Thus, in a hypotensive patient, **D5W** is not the resuscitative fluid of choice **(Shahid and Mehra, 2009).**

Types of crystalloids

Crystalloids are the most commonly used fluids for fluid resuscitation in various types of shock. They replenish the extracellular fluid losses.

Normal saline

Normal saline (0.9%sodium chloride) contains 154 mEq/L 0f both Na and Cl, it has an osmolarity of 308mOsm /L and pH of 5.0. It has the advantage of being the only crystalloid that can be mixed with blood. Because of its chloride content, it can lead to hyperchloremic metabolic acidosis, which can be corrected by chloride excretion by the kidneys. When 5% dextrose is added to 1L normal saline, the osmolality increases to 586mosm/L **(Shahid and Mehra, 2009).**