Fluid Therapy and Pulse Pressure Variation in Renal Transplantation

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Abstract

Optimal fluid therapy is considered as very crucial to decrease delayed graft function after renal transplantation. The intraoperative period of renal transplantation is divided into two phases. The first phase is the one prior to the reperfusion and the second is after reperfusion of the graft. Prior to reperfusion, physicians should not consider the patients as hypervolemic as they are usually adequately dialyzed. The role of fluid therapy in these patients is very critical, hypovolemia might lead to brain hypoperfusion and over-transfusion might lead increased intracranial tension. All these factors make fluid management in these procedures complex and challenging. Evidence on the optimum protocol for intraoperative fluid management in is Goal-directed therapy (GDT) in the operating room is a term used to describe the use of cardiac output or similar parameters to guide intravenous fluid and inotropic therapy.

Keywords: Fluid Therapy, Pulse Pressure Variation

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Introduction

Resuscitation fluids are used during hypovolemia to expand the intravascular volume, for improving cardiac output and perfusion pressures. The characteristics of ideal intravenous fluid for resuscitation include the following: 1) should tend to stay in the vascular compartment. 2) should be isotonic to plasma 3) solutes' concentration as same as of human plasma 4) Inexpensive [1].

Fluid solutions are generally classified as colloids and crystalloids:

Crystalloid

Crystalloids are the first-line of choice in fluid therapy. They are solutions that contain small molecules, cheap, and easy to use. These solutes are freely permeable through capillary membranes [14]. Crystalloids have tonicity as same as of plasma, with most of the solutions

having osmolality between 240 and 340mOsm/kg. Crystalloids include normal saline and balanced crystalloids (Ringer's solution, Plasma-Lyte (PL) and Sterofundin) [12].

Colloid

Colloids are solutions containing high molecular weight particles that increase oncotic pressure and are added to a crystalloid. Colloid stays in the intravascular compartment for a longer time and it minimizes the volume infused during resuscitation compared to crystalloids. The volume of expansion of colloids is 5 times than crystalloids studying its properties, whereas in the clinical setting, the expansion is just 1.2–1.4 times [18]. The endothelial glycocalyx model has suggested that at low capillary filling pressures (hypovolemia), crystalloids tend to stay in the intravascular compartment and colloid is not superior in expanding the intravascular volume [21].

Choice of Fluid in Renal Transplantation

In the second phase of surgery, reperfusion presents metabolites from the graft, leading to acidosis in the recipient. Moreover, pre-existing acidosis in the recipients leads to the careful selection of crystalloid. Normal saline has traditionally been the resuscitation fluid of choice in the perioperative period of kidney transplantation. However, problems such as acidosis, hyperkalaemia, hyper-chloraemia and acute kidney injury were arisen [10].

Plasma-Lyte in the perioperative period is safe in renal transplantation and is associated with a favorable biochemical profile, including a reduced incidence of hyperkalaemia, better diuresis and less frequent use of renal replacement therapy early after surgery [15]. Plasma- Lyte is also associated with better MAP compared to the normal saline Group [19].

Combination of balanced crystalloids and normal saline crystalloid solutions are associated with lower serum chloride, lower serum creatinine levels at day 2, 3, and 7 and higher urine output when compared with patients who got normal saline alone [20].

Colloids are not recommended to be used in kidney transplantation. Colloids are associated with clotting disorders that may cause persistent renal damage, mainly observed with the use of hydroxy-ethyl starches [17]. Furthermore, in the perioperative period bleeding and insensible losses can reduce the extracellular volume and activate the inflammatory cascade, with consequent damage of the glycocalyx, which increases capillary permeability and loses of intravascular fluids. Therefore, the effectiveness of colloids was found to be less than crystalloids in renal transplantation [9].

Phases of Fluid Therapy in Renal Transplantation

Fluid management in renal transplantation includes resuscitation, optimization, stabilization, and evacuation (deresuscitation). Each phase is distinct and dynamic [11].

Resuscitation: In renal transplantation, factors such as adequate dialysis and morning dose of antihypertensives together with the vasodilatory action of anesthetic lead to a transient hypovolemic and hypotensive state, therefore, Resuscitation is for the first hit of hypotension or hypovolemia is advised [11].

Optimization: Post-reperfusion, volume status, and the perfusion pressure of the patient should be optimum to aid in the proper function of the neograft. Hemodynamic monitors can aid the physician in this phase and mostly it results in a slight positive balance.

Stabilization: it involves fluid therapy for maintenance and replacement of ongoing losses (if present).

Evacuation (deresuscitation) is as important as resuscitation because the excess positive fluid balance might hinder renal function by edema and might hinder renal perfusion by increasing CVP and renal venous pressure.

The markers and tests that guide fluid administration -which are now available at the bedside- are also helpful for guiding fluid removal (Deresuscitation strategies); the latter can be safely performed when tests have verified that there is no preload responsiveness [2].

Definition of fluid responsiveness:

Fluid responsiveness is defined as the ability of the left ventricle to increase its stroke volume after fluid administration which lead to an increase in the stroke volume by 10 to 15% after 200-500mL of crystalloids infusion over 10 to 15min. A positive response is only given by an average of 50% of patients [13, 16]. The rationale behind predicting fluid responsiveness is to identify patients on the ascending portion of the Frank-Starling curve, as these Patients have 'preload reserve' and are able to increase cardiac output in response to fluid administration. Previously we depended on Clinical signs like tachycardia and urine output, which were neither sensitive nor reliable predictors of fluid responsiveness [16].

Whereas fluid responsiveness is a continuous variable, there are many methods examining this characteristic. Applicability on both spontaneously breathing patients and those who are mechanically ventilated is now a prerequisite for these methods [18].

Volume responsiveness assessment methods are classified into static and dynamic measurements, depending on how and what surrogate to the preload or preload change is measured, into static and dynamic measures. They include:

A. Pressure based volume therapy (static): arterial blood pressure, central venous pressure (CVP), and (PAOP).

B. Stroke volume-based volume therapy (dynamic): stroke volume variation (SVV), pulse pressure variation (PPV), or pleth variability index (PVI) and volume therapy based on physiologic testing (dynamic)-Passive leg raising (PLR), End-expiratory occlusion test (EEOT) [21].

A. Static measures of fluid responsiveness

Considering the Frank–Starling relationship, the response to volume infusion is more likely to occur when the cardiac preload is low than when it is high

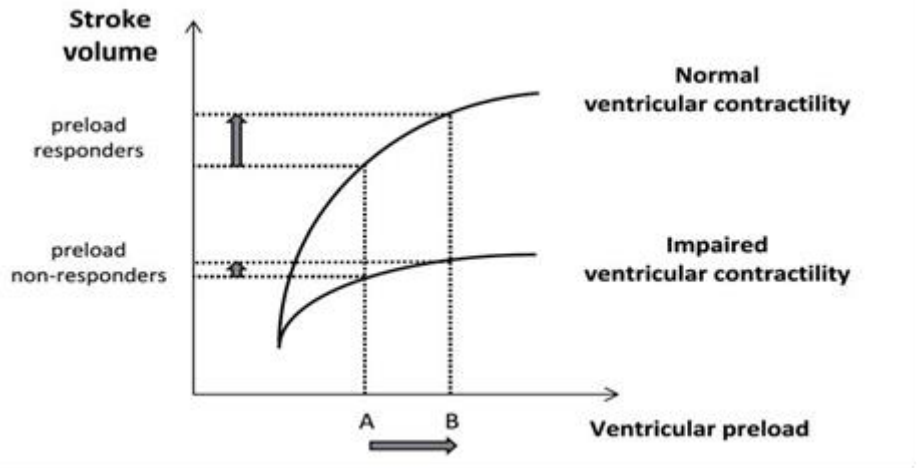

Figure (1): Frank–Starling relationship for assessing preload responsiveness [11].

Pressure measures depend on 3 determinants: the breath cycle, the cardiac cycle, and the anatomical and physiological properties of the heart. An assessment of preload by pleural pressure measurements should be carried out at the end of expiration, as pressure is near to 0 and intravascular pressure approaches transmural pressure. In regard to cardiac cycle, the measurement of the CVP should be made at the end diastole when the tricuspid valve closes and just before the ventricular systole. This point is represented by the R wave on the electrocardiogram [3].

Arterial blood pressure

As revealed before, any given arterial blood pressure cannot be used to decide whether additional fluid should be provided to a patient to increase cardiac output. Nevertheless, hypotension, in conjunction with the wider clinical picture, can help to find an indication to give fluid [13].

Central venous pressure (CVP)

Pulmonary artery occlusion pressure (PAOP)

Pulmonary artery catheters (PACs) measure the pulmonary artery occlusion pressure (PAOP) which is equivalent to the left-ventricular end-diastolic pressure (LVEDP) assuming normal pulmonary vascular resistance and absence of mitral.

But, like CVP, PAC is influenced by many other factors that are not related to the fluid status or fluid responsiveness such as cardiac compliance, intra-abdominal pressure, airway pressure and positive end- expiratory pressure (PEEP), pulmonary vascular resistance, and cardiac pathologies such as mitral/tricuspid regurgitation and congestive heart failure. In addition, PAC insertion and removal carry serious risks including arrhythmias, pulmonary infarction, and catheter knotting/ entanglement [13].

Extensive researches have concluded that PAOP should not be used to decide whether to give additional fluids [21].

Global end-diastolic volume (GEDV)

Global end-diastolic volume (GEDV) and global end-diastolic volume index (GEDI) are estimates of the total amount of blood in the cardiac chambers, detected by a transpulmonary thermodilution method. In this context, The PiCCO system uses transpulmonary thermodilution via a thermodilution-tipped arterial catheter and a central venous catheter through which a small bolus of saline is administered [14].

Inferior vena cava diameter:

The diameter of the inferior vena cava (IVCd) is assumed to change with preload. Theoretically, an increase in the IVCd signifies an increase in preload and right atrial filling. The IVC is visualized using bedside ultrasound in the subcostal plane [12].

Its accuracy is operator- dependent and prior training is required to develop proficiency in image acquisition and interpretation. Multiple patient related factors such as obesity, lung hyperinflation due to obstructive lung disease, presence of pneumothorax, and abdominal gaseous distention may cause inadequate sonographic window rendering this method unusable. The diameter of the IVC has been shown to correlate with RAP [16].

Left ventricular end-diastolic area:

Left ventricular end-diastolic area (LVEDA) can be measured using transthoracic or trans-esophageal (TEE) echocardiography [23].

B. Dynamic measures of fluid responsiveness:

Dynamic measure is defined as a change in preload (either by fluid challenge or physiological testing simulating volume loading manuvers), and observing the change in stroke volume (SV).

Dynamic measures depending on fluid challenge.

A fluid challenge is a maneuver in which a defined bolus of fluid is given within a short time frame. In most cases, this is an artificial colloid. It is indicated that the bolus is relatively standardized within the goal- directed hemodynamic therapy (GDT) literature, and is 250ml [22]. One of the simplest algorithms is to measure stroke volume, give a fluid challenge, and repeat this until the stroke volume no longer increases by more than 10% [21].

The bolus shouldn't be small or given too slowly, so that an acute increase of the right ventricular end-diastolic volume is not reached, there is a risk of a false negative result [21].

Mini fluid challenge (MFC) with concomitant monitoring of stroke volume is one of the most robust methods for preload challenge to avoid the risk of volume overload. The minimum volume of fluid to cause a significant hemodynamic effect has been demonstrated to be 4ml/kg. The main foreshortening of MFC method is the need for a real-time cardiac output (CO) surrogate (the first problem in fluid challenge maneuver) to trace the effect of MFC, as the longer the duration of the fluid challenge, the lower the proportion of fluid responders. This would make the test more applicable without the need for advanced hemodynamic monitors [21, 22].

The main limitation of the 'mini' fluid challenge is that it induces only small changes in cardiac output so that the technique used to measure these changes must be very precise.

Optimum heamoglobin status for patients under this challenge test is a must, as unsuccessful fluid challenge does not significantly increase stroke volume and, therefore, might decrease oxygen delivery due to inherent hemodilution if blood is not used with fluid challenge [21].

The dynamic preload parameters like Pulse contour analysis, SVV and PPV, are based on changes in the arterial pressure waveform due to changes in stroke volume in relation to positive pressure ventilation.

Pleth variability index (PVI)

The PVI is an algorithm that allows for the continuous and automatic estimation of respiratory variations in the pulse oximeter plethysmographic variation (ΔPOP) to assess fluid responsiveness. To use these parameters for GDT, it is mandatory to continuously measure the blood pressure or the pulse oximeter waveform amplitude [21].

The ΔPOP is calculated as follows:

ΔPOP (%) = 100 × (amplitude max – amplitude min) / [(amplitude max + amplitude min) / 2].

The reliability of the PVI is limited, but the PVI can play an important role in bedside monitoring for mechanically ventilated patients who are not undergoing surgery. Patients who are expanded with colloid may be more suitable for PVI [13].

PVI is not an accurate predictor of fluid responsiveness during kidney transplantation [15].

Pulse pressure variation (PPV)

PPV represents the variation of the pulse pressure over the ventilator cycle. It is defined as the difference between Pulse Pressure (systolic minus diastolic pressure) Maximal (PP max) and minimal (PP min) values over mean pulse pressure were determined over the same respiratory cycle.

PPV = PP max-PP min/ PP mean

PPV (%) =100× (PP max - PP min)/ [(PP max +PP min)/2]]

Stroke volume variation (SVV)

SVV represents the variation of the stroke volume over the ventilatory cycle.

SVV= (SV max –SV min)/ SV mean

Data from recent researches reveales the following:

In prone and supine positions, if BMI is < 30 kg/m2 , PPV in the prone position can predict fluid responsiveness as good as PPV in the supine position [1].

Both PPV and SVV are useful to predict cardiac response to fluid loading. In both responders and non-responders, PPV has a greater association with fluid responsiveness than SVV.

During major abdominal surgery, fluid resuscitation prompted by a PPV value higher than 13% (preload dependence state) led to optimization of both macrocirculation and sublingual

microcirculation, therefore, it is important to apply immediate correction of preload dependence to prevent reduced microcirculation and stresses the importance of PPV to guide fluid management during surgery [20]. Dynamic measures based on physiologic testing

Passive leg raising (PLR)

PLR test has been demonstrated to be reliable in detecting preload responsiveness in many studies through the evaluation of PLR-induced changes (volume expansion) on cardiac output (CO) [18].

In critically ill patients on mechanical ventilation, the diagnostic value of PLR for predicting fluid responsiveness depends on cardiac systolic function. [22].

The principle of PLR is that a certain volume of blood from the lower extremities and abdominal compartment increases preload and mimics a fluid challenge. During a PLR, an average around 300 mL of blood from rising the legs and mesenteric splanchnic pool is auto- transfused to the central circulation. Compared to a fluid bolus, it carries the benefit of not adding additional fluids in case the patient would not be fluid responsive. The PLR increases the mean systemic filling pressure (Pmsf) and, in case of preload responsiveness, venous return [6].

There are five rules that should be taken into account, when performing a PLR, semi-recumbent position ,direct measurement of COP by any tool like PPV and SVV ,short-term and transient , measured after and before during PLR, and finally, pain, cough, discomfort, and awakening could provoke adrenergic stimulation, resulting in erroneous interpretation of cardiac output changes [18].

PLR is not suitable for fluid responsiveness evaluation in patients undergoing kidney transplantation due to the unsuitable positions required by the PLR.

Fluid Therapy and Hemodynamic Monitoring in Renal Transplantation

CVP, pulse contour analysis, and transesophageal Doppler have been used in renal transplant surgeries [9].

CVP more than 12 mmHg is the target in renal transplantation and CVP less than 8 mmHg was strongly associated with graft dysfunction in adult data trial, [47,98,153]. However, it was indicated that CVP measurement should be abandoned in renal transplantation since it may be misleading; moreover, it was recommended the using intra-operative and post-operative cardiac output monitoring devices for guiding fluid therapy in renal transplant recipients [4].

During kidney transplant operation, the recipient is exposed to many intraoperative factors which may alter the CVP reading, hence, can be misleading in decision making. These factors can be summarised in the following points:

During the operation, the position of the patient is not always in flat supine position. The surgeon may be tilting the table in a different direction, commonly head down while elevating the left or the right side to improve the access to the iliac vessels. The effect of posture changes on CVP reading was documented since a long time [14].

Transplant surgery always requires the use of abdominal retractors. These retractors cause a pressure effect on the viscera and subsequently affect the venous return. Moreover, the tension created by the retractors will resist movement of the diaphragm and will eventually affect the intrathoracic pressure that lead to false CVP reading [24].

The target intra-operative CVP remains elusive. Aggressive hydration ensures good allograft perfusion. However, overhydration carries the risk of pulmonary congestion, pulmonary oedema, and prolonged intubation especially in patients with pre-existing cardiac conditions [5].

There is high risk of central vein stenosis due to the placement of central venous catheters. Central vein stenosis could threaten the future of arteriovenous fistula and arteriovenous graft in the ipsilateral extremity when the renal graft fails, and the patient returns to dialysis [10].

In renal transplantation, SVV predicts and correlates better with the volume status of the patient better than CVP in adult data [23].

Transesophageal Doppler (TED) is another tool to predict the volume status of the patient. In a randomized trial, there was no difference in the volume of intraoperative fluid administered between the TED group and conventional fluid management group and no difference in other complications [7].

In another randomized controlled trial, both TED and FloTrac devices (a minimally invasive system for advanced heamodynamic monitoring including SV and SVV) can be used effectively to guide goal directed fluid therapy in renal transplantation, However, lesser total fluid was required in the FloTrac group, which may lead to a lesser number of fluid-related postoperative complications [13].

Mean arterial pressure (MAP) targets after reperfusion of the graft are still a debate. The lower limit of MAP that must be maintained differs from 70 to 93mmHg according to different observations on graft dysfunction, however, it is indicated that the renal blood flow changes may not always follow the changes in MAP. Moreover, increasing the systemic vascular resistance may or may not improve renal perfusion [8]. Good early graft function was expected if MAP > 95mmHg [5].

Pulse Pressure Variation

The role of fluid therapy in these patients is very critical, hypovolemia might lead to brain hypoperfusion and over-transfusion might lead increased intracranial tension. All these factors make fluid management in these procedures complex and challenging. Evidence on the optimum protocol for intraoperative fluid management in is Goal-directed therapy (GDT) in the operating room is a term used to describe the use of cardiac output or similar parameters to guide intravenous fluid and inotropic therapy [11].

Intraoperative goal directed fluid therapy (GDT) have been reported to improve patient outcome in high risk surgical patients [25].

GDT aims to optimize oxygen delivery through various strategies. The main three GDT strategies are:

Stroke volume optimization with fluids.

Oxygen delivery index with fluids and inotropes

Pulse pressure variation (PPV) and stroke volume variation (SVV) optimization with fluids.

[9, 11]

Pulse pressure: is the difference between systolic and diastolic blood pressure, or it is the change in blood pressure seen during a contraction of the heart. (Pulse pressure = the systolic pressure - the diastolic pressure).

The systemic pulse pressure is direct proportional to stroke volume and inversely proportional to the compliance of the aorta. [22].

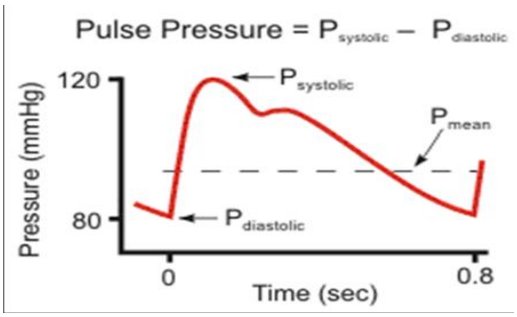

Figure (2): Arterial wave showing pulse pressure [22].

Values and variation

• Systemic pulse pressure = Psystolic- Pdiastolic = 120mmHg - 80mmHg = 40mmHg

• Pulmonary pulse pressure= Psystolic - Pdiastolic = 25mmHg - 10mmHg = 15mmHg

Low values (narrow) In trauma a low or narrow pulse pressure suggests significant blood loss. In an otherwise healthy person a difference of less than 40 mmHg is usually an error of measurement. If the pulse pressure is actually low, e.g. 25 mmHg or less, the cause may be low stroke volume, as in Congestive Heart Failure and/or shock. This interpretation is reinforced if the resting heart rate is relatively rapid, e.g. 100-120. [19].

High values (wide) may be physiological: As in pregnancy and shortly after exercise or pathological:As in • Atherosclerosis • Chronic aortic regurgitation • Thyrotoxicosis • Fever • Anaemia • Pregnancy • Anxiety • Heart block • Aortic dissection • Endocarditis • Raised intracranial pressure [19].

Pulse pressure variation (PPV) is a naturally occurring phenomenon in which the arterial pulse pressure falls during inspiration and rises during expiration due to changes in intra-thoracic pressure secondary to negative pressure ventilation (spontaneously breathing). While on controlled mechanical ventilation, arterial pressure rises during inspiration and falls during expiration due to changes in intra-thoracic pressure secondary to positive pressure ventilation. Its normal value is less than 13%. The principles underlying the PPV and SVV are based on simple physiology that the inferior vena cava (IVC) is a compliant blood vessel subject to abdominal

pressure and acts as a reservoir .Its caliber is altered by respiration, blood volume and right heart function [12].

Dynamic changes of arterial waveform-derived variables, such as SVV and PPV, during mechanical ventilation are highly accurate in predicting volume responsiveness in critically ill patients with an accuracy greater than that of traditional static indices of volume responsiveness. This technique, however, is limited to patients who receive controlled ventilation and who are not breathing spontaneously [21].

Pulse pressure variation (PPV) is a famous dynamic method of fluid responsiveness. PPV is simply calculated by dividing the largest pulse pressure (PPmax - PPmin) by the average pulse pressure (PPmax + PPmin /2) and expressed as percentage [22] . PPV was previously used in GDT in major abdominal surgery with good performance. [21].

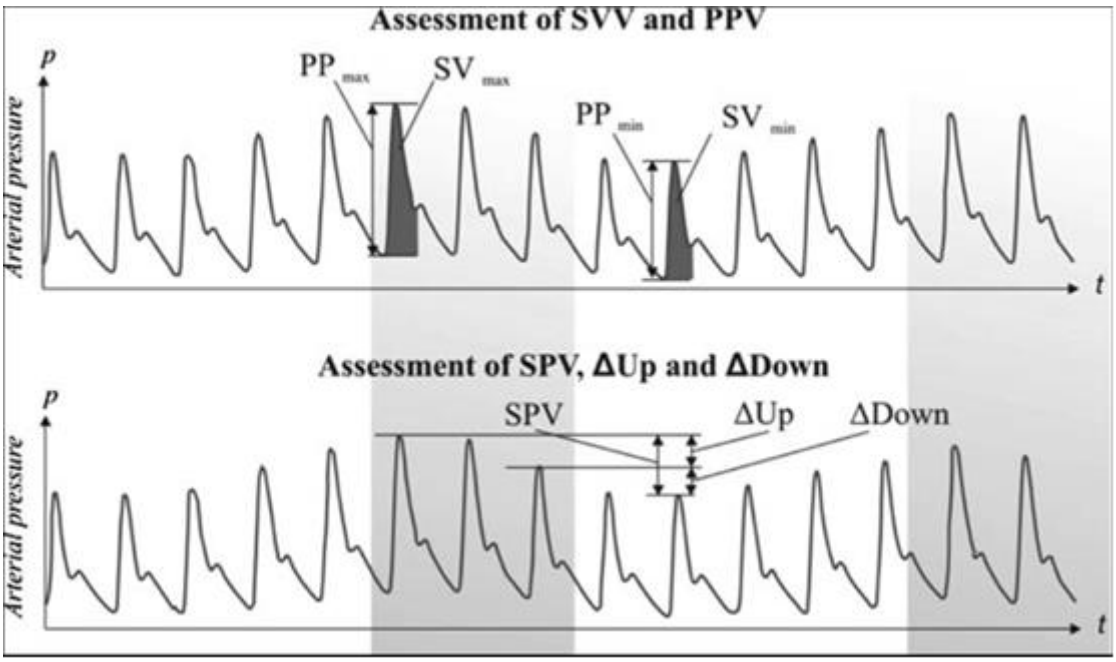

Figure (3): Assessment of SVV and PPV [12].

Preload and cardiac output relationship

SV is determined by three factors: preload, afterload, and contractility. The preload gives the blood volume for the ventricle to pump, as well as the end diastolic length of the muscle. The contractility is the force that the muscle can create at the given length, and the afterload is the arterial pressure against which the muscle will contract. These factors establish the volume of blood pumped with each heartbeat.

The direct relationship between end-diastolic fiber length and contractile force was first demonstrated experimentally by Frank Starling. This Frank-Starling principle illustrates the relationship between CO and LVEDV. It is based on the length-tension relationship within the ventricle. If ventricular end diastolic volume (preload) is increased , resulting in an increased 'tension' of the muscle. In this way, cardiac output is directly related to venous return, the most important determining factor of preload. When heart rate is constant, CO is directly related to preload (up to a certain point.). An increase in preload will increase the CO until very high end-

diastolic volumes are reached. At this point CO will not increase with any further increase in preload, and may even decrease after a certain preload is reached [8].

The use of goal-directed therapy (GDT) has been investigated in different situations, including the emergency, ICU, and operating room. The typical patient population that have been chosen for evaluating the potential benefits of GDT, include patients suffering from septic shock. and In the surgical setting, higher risk procedures, such as

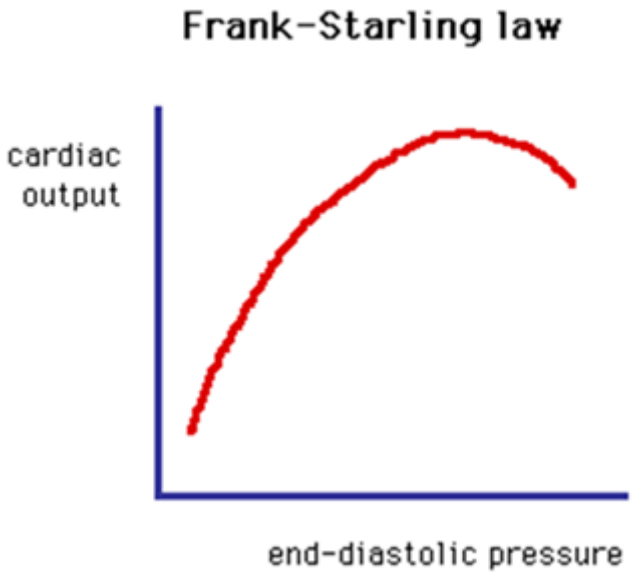

Figure (4): A schematic drawing of Frank-Starling curve. [21].

Gastrointestinal, vascular, and cardiac surgery patients, these patients are often considered at higher risk for a poor outcome regardless of the type of surgery. Therefore strategies(such as GDT), which may improve complications, , mortality and morbidity are frequently discussed by clinicians and in an attempt to guide clinical care.

The use of GDT forces physicians to stick to a defined algorithm (that potentially improves outcomes), and also helps to reduce variation in care. Patient outcome is always the top priority for any doctor, but until now studies aiming to define the optimal hemodynamic parameter(s) and intervention(s) for GDT, the use of GDT in the perioperative setting will not be accepted by the majority [4].

The implementation of GDT to clinical practice is not a new concept. It dates back many years, and remains a major focus in medicine especially in treating patients with septic shock. The use of GDT in septic shock has been accepted for many years. Recent trials such as ARISE, ProCESS, and also OPTIMISE, have not shown significant benefit of GDT with regards to morbidity or mortality [8].

No Conflict of interest.

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