World Journal of Hepatology

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DOI: 10.4254/wjh.v7.i10.1302

World J Hepatol 2015 June 8; 7(10): 1302-1311
ISSN 1948-5182 (online)

REVIEW

Hemodynamic monitoring during liver transplantation: A state of the art review

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Author contributions: De Marchi L and Plotkin JS designed research; Rudnick MR performed research; Rudnick MR and Plotkin JS analyzed data; Rudnick MR wrote the paper.

Conflict-of-interest: The authors have no conflict of interest to report.

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Received: September 19, 2014
Peer-review started: September 20, 2014
First decision: December 17, 2014
Revised: March 25, 2015
Accepted: April 8, 2015
Article in press: April 9, 2015
Published online: June 8, 2015

Abstract

Orthotopic liver transplantation can be marked by significant hemodynamic instability requiring the use of a variety of hemodynamic monitors to aide in intraoperative management. Invasive blood pressure monitoring is essential, but the accuracy of peripheral readings in comparison to central measurements has been questioned. When discrepancies exist, central mean arterial pressure, usually measured at the femoral artery, is considered more indicative of adequate perfusion than those measured peripherally. The traditional pulmonary artery catheter is less frequently used due to its invasive nature and known limitations in measuring preload but still plays an important role in measuring cardiac output (CO) when required and in the management of portopulmonary hypertension. Pulse wave analysis is a newer technology that uses computer algorithms to calculate CO, stroke volume variation (SVV) and pulse pressure variation (PPV). Although SVV and PPV have been found to be accurate predicators of fluid responsiveness, CO measurements are not reliable during liver transplantation. Transesophageal echocardiography is finding an increasing role in the real-time monitoring of preload status, cardiac contractility and the diagnosis of a variety of pathologies. It is limited by the expertise required, limited transgastric views during key portions of the operation, the potential for esophageal varix rupture and difficulty in obtaining quantitative measures of CO in the absence of tricuspid regurgitation.

Key words: Intraoperative monitoring; Physiologic monitoring; Liver transplantation

Core tip: Accurate hemodynamic monitoring is essential to safely navigate orthotopic liver transplantation. Although specific indications for pulmonary artery catheters exist, their use has slowly been replaced by newer technologies which offer less invasive and more accurate measurement. The latest evidence on the strengths and limitations of arterial pulse wave form analysis, intraoperative transesophageal echocardiography, peripheral *vs* central arterial blood pressure monitoring and pulmonary arterial catheters are discussed.

Rudnick MR, De Marchi L, Plotkin JS. Hemodynamic

monitoring during liver transplantation: A state of the art review. *World J Hepatol* 2015; 7(10): 1302-1311 Available from: URL: http://www.wjgnet.com/1948-5182/full/v7/i10/1302.htm DOI: http://dx.doi.org/10.4254/wjh.v7.i10.1302

INTRODUCTION

Orthotopic liver transplantation (OLT) has been performed for the past three decades with significant improvement in patient and graft survival. Despite improvements in the anesthetic and surgical techniques, it still is a challenging procedure, requiring dedicated, specifically trained providers and a collection of monitors not common to other operations.

Most classic hemodynamic monitors like radial and femoral arterial lines and a pulmonary artery catheter (PAC) are still part of the protocol at many institutions[1], but new technology has been emerging. These new devices and techniques along with evidence of the limitations of some of the classic monitors, are reshaping the way in which hemodynamics are monitored during anesthesia for liver transplantation in the 21st century.

HEMODYNAMICS DURING LIVER TRANSPLANTATION

Liver transplantation can be thought of as having 3 distinct stages: the dissection or pre-anhepatic phase, the anhepatic phase, and the neohepatic phase. Each stage has its own hemodynamic concerns.

The pre-anhepatic phase is when all the dissection occurs, and is marked by significant fluid shifts from drainage of ascites to the potential for significant blood loss in the presence of varices from portal hypertension. Additionally, manipulation of the liver and downward retraction of the inferior vena cava may intermittently obstruct venous return causing hemodynamically significant changes in preload[2].

The anhepatic stage is defined as the cessation of blood flow to the native liver until the time of reperfusion of the transplanted liver. With cross clamping of the portal vein and IVC, cardiac output (CO) may decrease by up to 50%[3]. To avoid this sudden loss of preload, volume loading should occur prior to crossclamping. An alternative is use of the "piggyback" technique by the surgeons where the inferior vena cava is only partially occluded. Other alternatives include the use of a temporary portocaval shunt or venovenous bypass. Some centers make use of one of these techniques routinely while others employ them as clinically indicated[4].

The neohepatic stage is defined as the beginning of reperfusion until the end of the case. Reperfusion is often marked by significant hemodynamic instability due to the rapid return of blood from the previously obstructed portal system and newly transplanted liver. This blood tends to be acidotic, hyperkalemic, cool, and contains a variety of inflammatory and vasodilatory mediators[3]. The result is often a transient but significant decrease in myocardial contractility, chronotropy and systemic vascular resistance[5]. Postreperfusion syndrome, defined as a decrease in mean arterial pressure by 30% for at least 1 min within 5 min of reperfusion, has been reported to occur in 12.1%-42% of patients[6,7]. After overcoming the instability of reperfusion, the remainder of the neohepatic stage tends to have relatively stable hemodynamics.

BLOOD PRESSURE

Invasive blood pressure monitoring is the standard of practice during liver transplantation. The number and location of these lines varies by center[1]. In healthy individuals, radial artery pressures have a higher systolic pressure as compared to femoral or more central pressures. This difference has been attributed to pulse amplification as a result of the impedance and harmonic resonance of the vasculature. However, the central and distal mean arterial pressures remain relatively unchanged[7,8].

Specific circumstances can create significant discrepancies between central and radial mean arterial pressures. When measuring radial arterial pressure, the presence of a proximal obstructive vascular lesions, rewarming after hypothermic cardiopulmonary bypass[9,10], and high dose vasopressor therapy in critically ill patients[7] are all known to underestimate central pressures. The cause for this discrepancy in obstructive vascular lesions is self-evident, but controversy exists regarding the etiology in cardiopulmonary bypass or vasopressor therapy. In the latter, it has been theorized vasoconstriction of the extremity conductance vessels, which are more sensitive to vasopressors than the central vasculature, significantly reduces the flow to the radial artery[10]. Regarding the use of cardiopulmonary bypass, it is theorized to cause an extreme vasodilatory state leading to proximal shunting, possibly in the splanchnic beds, in combination with distal vasoconstriction which both contribute to lower peripheral pressures[11].

Similarly, it has been suspected in OLT that radial arterial and more centrally measured pressures may not correlate well[12]. Our observations and demonstrated in unpublished data, over many years, have shown a consistent decrease in systolic, diastolic and mean pressures in radial *vs* femoral arterial pressures, most pronounced immediately after reperfusion. The theory behind this, similar to post-cardiopulmonary bypass physiology, is extreme peripheral vasodilation, seen especially during reperfusion, decreases distal pressures disproportionately to central pressure[13,14]. Studies attempting to demonstrate this effect have had conflicting results. Acosta *et al*[15] found no difference in mean arterial pressure at any point during OLT, while Arnal *et al*[16] found systolic but not mean arterial pressures differed during reperfusion which was exag-

gerated in patients receiving vasopressors. A study looking at pediatric OLT, on the other hand, did show a discrepancy is both systolic and mean arterial pressures when comparing femoral to radial pressures throughout most of the operation[17]. Interestingly, a separate study showed noninvasive blood pressure measurements of the upper extremities more closely correlated with femoral artery pressures than radial pressures 3-10 min after reperfusion. Presumably this is due to the more proximal location of the blood pressure cuff[18].

Larger trials are required to definitively determine the reliability of radial pressure monitoring during OLT, but currently the literature suggests central monitoring from femoral or brachial locations and mean arterial pressures should be followed over peripheral radial systolic pressures.

PAC

According to the Frank-Starling law, as end-diastolic volumes increase, myocardial fiber length increases which in turn increases the number of myosin-actin connections resulting in increased CO. Classically, central venous and pulmonary artery occlusion pressures (PAOPs) have been used as surrogates for volume measurements. However, numerous studies have established that these static preload measurements are poor predictors of end diastolic volume and fluid responsiveness in a wide variety of medical and surgical patients[19-26]. The compliance of the heart and vasculature, intrathoracic pressures, cardiac contractility and valvular pathologies significantly affect the pressure measured for a given preload making static pressure measurements an unreliable indicator of end diastolic volume[27,28].

Specifically during liver transplant, Costa *et al*[29] found stroke volume index did not correlate with either central venous pressure (CVP) or PAOP. Similarly, Rocca *et al*[28] looked at pre-anhepatic dissection, the anhepatic phase, and after reperfusion and found no correlation between cardiac index and CVP or PAOP during liver transplantation. A separate study by Rocca *et al*[30] again found in 244 patients undergoing liver transplantation that CVP and PAOP correlate poorly with stroke volume index.

PACs also allow for the measurement of CO *via* intermittent thermodilution. Although often used as the gold standard for CO measurements, its accuracy depends on several user-dependent techniques such as the speed, volume and temperature of the injectate as well as its timing with respect to the respiratory cycle[27]. Significant tricuspid regurgitation or intracardiac shunts also limit the accuracy of thermodilution[31,32]. Additionally, during liver transplantation, the rapid return of cooled blood during reperfusion and administration of large volumes of intravenous fluids generate thermal noise which may result in an underestimation of the true CO[33,34].

A new generation of PACs allow for continuous CO monitoring. This technology uses heat instead of cold thermodilution *via* a thermal filament connected to a specialized PAC and a distal thermistor. CO values are then continuously calculated. Several studies have shown continuous CO measurements correlate well with intermittent thermodilution in a variety of patient populations[35-37]. Although also seen to be accurate during liver transplantation[33], it has similar limitations to intermittent thermodilution with less accuracy during reperfusion and cross-clamping[34].

Mixed venous oxygen saturation (SvO_2) can be measured *via* a PAC and used as an indirect measure of CO. However, changes in SvO_2 are not very specific to CO and may be the result of changes in oxygen content, oxygen consumption or, in the case of reperfusion, return of desaturated blood[38]. Specifically, in liver transplantation SvO_2 has shown to poorly correlate with CO[39].

Perhaps the most important use of PAC during liver transplantation is in patients with portopulmonary hypertension. Patients with mean PA pressures above 50 have typically been denied liver transplantation due to an unacceptably high risk of mortality ranging from 71%-100%[40,41]. While mild (mean PA pressures 25-35 mmHg) and moderate (mean PA pressures 35-45) pulmonary hypertension are not strict contraindications for liver transplantation[42,43], these patients still do have an elevated perioperative mortality rate as high as 33%-35%[40,41]. When pulmonary artery pressures are responsive to treatment, the mortality risk significantly decreases[44-46]. Preoperative workup demonstrating increased PA pressures on echocardiography suggest portopulmonary hypertension. Additionally, hypoxemia or exaggerated respiratory alkalosis also may suggest increased PA pressures[47]. However, a PAC is the only modality that directly measures pulmonary arterial pressures. At the start of the procedure, it may unveil worse pulmonary hypertension than suspected during preoperative evaluation leading to cancellation of the procedure. Intraoperatively, the sudden volume shifts and release of vasoactive mediators seen, especially during reperfusion, may result in significant right heart strain and failure[48]. Treatment of elevated PA pressures during OLT includes the use of venovenous bypass during the anhepatic phase, phosophodiesterase-5 inhibitors, endothelin receptor antagonists, and prostacyclins[48-50].

The use of PACs does have significant diagnostic limitations however they are still one of the most accurate tools to assess CO and an essential monitor in patients with significant pulmonary hypertension.

CO WAVEFORM ANALYSIS

Beginning in 1904, Joseph Erlanger and Donald R Hooker theorized that CO could be derived from the arterial pulse pressure[51]. Logically, as more blood is ejected from the heart, there should be a greater pressure transmitted to the arterial tree. This correlation

is limited, however, due the vascular resistance which determines the runoff of blood from the conductance vessels into the arterioles. Even more complex is the nonlinear compliance of the arterial tree where a given volume of blood will decrease compliance more at higher pressures as compared to lower pressures[52]. Only recently has the technology been available to not only precisely analyze pressure waveforms but also the sufficient knowledge to create algorithms which account for the complex physiology of pulse wave morphology.

There are currently several commercially available products which calculate continuous CO from arterial waveform analysis. The PiCCO system (Pulsion Medical System, Munich, Germany) requires the placement of a thermodilution catheter into the axillary or femoral artery. A solution is then injected into any central venous catheter and CO is then determined by the arterial temperature probe. After this initial calibration, the system then continuously calculates CO based on arterial waveform analysis. Additionally, static preload indices such as global end diastolic volume and intrathoracic blood volume can be calculated. The LiDCO system (LiDCO Plus, Cambridge United Kingdom) is similar to the PiCCO system but uses lithium indicator dilution rather than thermodilution to calibrate the pulse wave form to the CO[53]. The Flowtrac/Vigileo system (Edwards Lifesciences, Irvine, CA United States) uniquely does not require intermittent CO bolus calibration. It accounts for changes in arterial compliance and resistance using a conversion factor Khi which factors in the standard deviation of the mean arterial pressure and the analysis of the skewness and kurtosis of the arterial waveform. Large vessel compliance is then estimated using patient demographics of age, gender and body surface area[54].

In patients undergoing liver transplant, PiCCO derived CO measurements were found to agree with the gold standard of PAC thermodilution CO[55]. Similarly Nissen *et al*[56] looking at dissection, anhepatic, early and late reperfusion phases during OLT also found arterial pulse wave CO measurements to correlate with thermodilution CO.

Despite a few reassuring studies, significant concerns have been raised concerning the validity of arterial waveform analysis in particular patient populations whose physiology may not be well represented by the algorithms. CO by waveform analysis was found to correlated poorly with PAC thermodilution CO in patients on a significant dose of vasopressor[57,58]. There was also poor correlation during cardiac surgery[59] and abdominal aortic aneurysm operations[60] with the uncalibrated FlowTrac system performing worse than the PiCCO system.

The hyperdynamic circulation in liver cirrhosis is characterized by low systemic vascular resistance, elevated CO and possible underlying cardiomyopathy. The transplant operation then adds large changes to preload and afterload, vasoactive mediators during reperfusion, myocardial contractility changes and the possibility of significant hemorrhage[14]. Likely as a result of this altered physiology and dynamic intraoperative changes, several studies have found poor correlation between waveform analysis CO calculations when compared to the PAC thermodilution. Krejci *et al*[59] found, in Child-Pugh class B and C cirrhotics, both FlowTrac and LiDCO systems correlated poorly with PAC thermodilution. Two separate studies found that in Child-Pugh B and C cirrhosis, the degree of FlowTrac's inaccuracy was proportional to the patient's SVR with lower resistances showing less correlation to reference thermodilution values[61,62]. Della Rocca *et al*[63] examined the effect of the high output cardiac state and found Flowtrac underestimated CO in liver transplant patients whose CO exceeded 8 L/min.

A recent software update to FlowTrac has been released (third generation) whose aim was to improve CO accuracy specifically in low systemic vascular resistance states. Some improvements have been seen in septic patients[64] and those undergoing cardiac surgery[65]. Despite these improvements, in liver transplantion the accuracy of FlowTrac CO measurements remain unreliable. Tsai *et al*[66] found a 55% discrepancy between the third generation software of FlowTrac COs as compared to PAC thermodilution. Likewise, Su *et al*[67] found a percentage error of 75% which was inversely related to the patient's systemic vascular resistance index. CO derived from waveform analysis depends on the intrarterial peripheral catheter reflecting systemic conditions. CO in cirrhosis and during liver transplantation, however, is not evenly distributed[29]. Peripheral arterial waveform analysis, therefore, cannot be recommended for liver transplant intraoperative monitoring or in Child-Pugh class B or C patients.

With the currently available technology, arterial waveform analysis cannot reliably measure CO during OLT.

STROKE VOLUME AND PULSE PRESSURE VARIATION

Although CO by waveform analysis has proven unreliable in liver transplant, the technology also allows for the continuous measurements of stroke volume variation (SVV) and pulse pressure variation (PPV).

Pulse pressure changes proportionally to left ventricular stroke volume[25]. During positive pressure ventilation, blood return to the right heart decreases. After this lower volume of blood passes through the pulmonary vasculature, left ventricular end diastolic volume decreases. The overall result is lower stroke volumes and a smaller pulse pressure after positive pressure ventilation. The magnitude of this difference is proportional to preload. Patients whose CO would be supplemented by increased intravascular volume will have a larger difference in pulse pressure during positive pressure ventilation and exhalation. A review of twenty-nine studies studying this phenomenon found SVV less than 11.6% ± 1.9% and a PPV less than 12.5% ±

1.6% predicted volume responsiveness in critically ill patients[26].

Arterial waveform analysis of the SVV has been shown to predict fluid responsiveness during general anesthesia[68] and the use of vasoconstrictors does not change the variation[69]. In liver transplantation, poor SVV was found to be a better predictor of right ventricular end diastolic volume index than CVP[70]. Kim *et al*[71] confirmed these findings and found a PPV of greater than 9% predictive of lower RVEDVI which would likely be fluid responsive. Biais *et al*[72] found, in liver transplant, a SVV of 10% discriminated fluid responders from non-responders with a 93% sensitivity and 94% specificity.

The accuracy of SVV or PPV in predicting fluid responsiveness depends on the patient meeting a specific set of criteria. Stroke volume is less predictably dependent on preload during spontaneous breathing. Breathing effort causes irregularity in both pulse rate and intrathoracic pressures[73]. Not only must the patient be mechanically ventilated with no breathing efforts, but the pressure must be adequate to decrease preload. The degree of SVV is linearly related to tidal volume with tidal volumes less than 10 mL/kg showing a lesser degree of SVV[74]. Likewise, when driving pressures (defined as the difference between the plateau and positive end expiratory pressure) are less than 20 cm H_2O, a PPV less than 13% does not rule out fluid responsiveness[75]. De Backer *et al*[76] found that PPV only reliably predicted fluid responsiveness at tidal volumes above 8 mL/kg. Finally, during cardiac arrhythmias, SVV is more dependent on the irregular diastolic time than on intravascular volume[77].

Although waveform analysis has limited utility in quantitative measurements of CO in cirrhosis and liver transplantation, it is a minimally invasive option of monitoring end diastolic volume and fluid responsiveness.

TRANSESOPHAGEAL ECHOCARDIOGRAPHY

Echocardiography has a variety of intraoperative uses. In experienced hands, it has the capability of diagnosing right ventricle (RV) or left ventricle (LV) systolic or diastolic dysfunction, volume overload, global or regional wall abnormalities, and intracardiac air or thrombosis[78-80]. In liver cirrhosis, numerous case reports have reported its usefulness in diagnosing porto-pulmonary hypertension[81], ischemic heart disease, cirrhotic cardiomyopathy[82], and intraoperative thromboembolic events[83]. More rare conditions such as pericardial tamponade[84], cardiomyopathy secondary to undiagnosed hemochromatosis[85], and Takosubo cardiomyopathy[86,87] have also been reported during OLT intraoperative transesophageal echocardiography (TEE).

One of the greatest strengths of TEE is its ability to directly visualize in real-time the preload of both the right and left sides of the heart. As previously discussed, the PAOP is a known poor measure of LV preload. The PAOP may differ dramatically despite no change in left end-diastolic volume (LVEDV) and fails to detect hypovolemia when compared to direct measurement of LVEDV by TEE[88]. The LVEDV can be directly visualized from the transgastric mid-short axis view (TG mid-SAX). Additionally, TEE has been shown to accurately determine stroke volume and left ventricular changes[88]. In a multicenter study of 244 patients undergoing OLT, stroke volume index was found to be more strongly correlated with right ventricular end diastolic volume index than either CVP or PAOP[30].

Similarly, CVP can be an unreliable indicator of stroke volume and intravascular volume[20,23,89]. During increases in intravascular volume, the RV dilates which results in no significant change in the CVP despite large increases in volumes[90]. The resulting fluid overload can result in congestion and injury to the newly transplanted liver. SVV can indicate the need for additional fluid, but depends on a regular heart rate and adequate tidal volumes without respiratory effort. These are not limitations for TEE as it directly measures end diastolic volume. Determination of preload by TEE has significant clinical consequence. Hofer *et al*[82] found intraoperative use of TEE influenced fluid therapy in 50% of OLT patients.

Hypotension in the presence of adequate preload may be a result of myocardial dysfunction. Coronary artery disease (CAD) is not uncommonly encountered in patients undergoing OLT, with one study reporting up to 32% of patients over the age of 50 having moderate to severe disease[91]. Patients with known CAD undergoing OLT have a mortality of 50%[92]. Due to this high mortality, reversible ischemia seen on stress testing is a contraindication to proceeding with OLT[93]. Intraoperative TEE allows for the real time detection of myocardial ischemia manifested by regional wall motional abnormalities. A study by Smith *et al*[94] found intraoperative TEE was more sensitive in detecting myocardial ischemia than EKG changes. A separate study showed TEE may be more sensitive than conventional monitors as 73% of patients with regional wall abnormalities had no detectable change in heart rate, blood pressure or PA pressures[95].

Pulmonary embolization can be a real and significant risk during OLT[96]. Paradoxical embolization to the systemic circulation may result in stroke and can occur as a result of a patent foramen ovale. Cirrhotic patients are at particular risk for paradoxical embolization as dilated pulmonary vasculature can allow the free passage of emboli to the systemic circulation. TEE can not only identify patients with right to left shunts but also distinguish between intracardiac and transpulmonary etiologies, thereby identifying those patients who are at higher risk for paradoxical embolism[97]. Furthermore, TEE Doppler is the most sensitive monitor for the detection of air embolism, able to detect 0.1 mL/kg of air[98]. The diagnosis of an acute pulmonary embolism, however, is more difficult with TEE, typically manifesting

Table 1 Comparative summary table of hemodynamic monitors in orthotopic liver transplantation

Monitor	Benefits and uses	Limitations
Invasive blood pressure monitoring	Beat-to-beat monitoring of blood pressure	Peripheral arteries possibly underestimate the central mean arterial pressure especially during reperfusion or use or high dose vasopressors
Pulmonary artery catheter	Accurately determines cardiac output *via* intermittent thermodilution	Invasive
	Directly measures PA pressures	Static pressure measurements are imperfect indicators of fluid status or stroke volume
Arterial pulse wave analysis - CO	Less invasive option to calculate CO	Does not reliably calculate CO in advanced cirrhosis or during OLT
Arterial pulse wave analysis - SVV	Predicts fluid responsiveness in OLT population	Requires sinus rhythm
		Requires patient does not make any spontaneous respiratory efforts
		Most accurate during tidal volumes of 8-12 mL/kg
Transesophageal echocardiography	Direct assessment of cardiac filling	Requires advanced training
	Monitors myocardial ischemia and strain	Limited views intraoperatively
	Potentially can diagnose pulmonary embolisms, shunts, effusions, and valvular pathologies	Risk of esophageal varix rupture or esophageal injury

CO: Cardiac output; SVV: Stroke volume variation; OLT: Orthotopic liver transplantation; PA: Pulmonary artery.

with signs of RV dysfunction such as RV dilation, hypokinesis and possible pulmonary hypertension[99]. However, a large burden of pulmonary embolism is required to see these effects, especially those over 30% of the pulmonary artery area, are more likely to show RV dysfunction[100]. The classic "McConnell sign" refers to hypokinesis of the RV with preservation of RV apical contractility[101]. This sign has been reported to be very specific for acute pulmonary embolism but with a sensitivity of only 19%[102,103].

Despite the many advantages of TEE, like every monitor, there are limitations and risks. The proficient use of TEE requires significant training and expertise with the American Society of Echocardiography recommending 300 transthoracic echocardiograms, 20 esophageal intubations and 50 transesophageal examinations within a 6 mo period[103]. However, the performance of "limited-scope examinations" by non-credentialed anesthesiologists is not uncommon with 88% of users lacking echocardiography certification[104].

Additionally, the presence of esophageal varices creates concern for rupture. However, while grade four esophageal varices may be a true contraindication[27], TEE has been safely performed in patients with grade Ⅰ and Ⅱ varices[105]. TEE is also limited in its ability to assess pulmonary artery pressures in the absence of tricuspid regurgitation with the far majority of centers using a PAC with or without the use of TEE[1]. As previously discussed, TEE is a very sensitive monitor for ischemia, however, the transgastric short axis view which best assesses the circumference of the left ventricle is largely unavailable during the operation due to posterior retraction of the stomach[27].

CONCLUSION

A variety of hemodynamic monitors are an essential part of the successful intraoperative management of patients undergoing OLT but each has their own indications and limitations (Table 1).

Invasive blood pressure monitors is currently the standard of care during transplantation, however, the evidence suggests that peripheral measurements are possibly not representative of central perfusion pressures especially in instances of significant vasopressor use or in patients with unequal vasodilation as is the case in significant cirrhosis or during reperfusion. When possible, more central invasive monitors at the femoral or brachial artery have a theoretical advantage of representing central perfusion pressures.

PACs continue to have controversial indications in and outside the operating room. The available evidence is clear that static cardiac pressure measurements such as CVP and PAOP are imperfect predictors of fluid responsiveness and CO. However, CVP allows for the monitoring of the backpressure of the IVC into the newly transplanted liver and may still guide the transplant anesthesiologist in fluid management or need for vasodilators to prevent injury to the liver. When using a combination of data such as the heart rate, blood pressure, CVP, urine output, a more clearly picture of the patient's hemodynamic status may emerge.

A role still exists for PACs for the accurate measurements of CO *via* intermittent or, more recently, continuous thermodilution which remains the current gold standard. In OLT, PACs also play an important role in the monitoring of patients with PA pressures in patients with pulmonary hypertension which, even in the setting of mild hypertension, carries a significant risk of morbidity and mortality.

Pulse pressure analysis is a newer monitoring technique. Although the promise of accurate and continuous CO analysis has not been delivered in the OLT patient population, continuous PPV monitoring does appear to predict fluid responsiveness and may serve as an invaluable guide.

TEE is the most direct measurement of cardiac filling that currently exists allowing for the real-time assessment of fluid status during these dynamic operations. Additionally, it also offers the unique benefit of diagnosing a variety of other intraoperative complications such as myocardial ischemia, pulmonary embolism, and pulmonary or pericardial effusions. It currently is limited by the expertise required to interpret the images, but as more anesthesiologists are trained in this technology it stands to supplant many of the indirect monitors currently in use.

The perfect hemodynamic monitor would be non-invasive, precise and accurate, and provide continuous data at all stages of transplantation. Until this device exists, adept intraoperative management requires knowledge of the applicability and known limitations of available technology. Perhaps the current best monitor is the experienced provider who can adeptly integrate the various pieces into a complete but adaptable perioperative treatment plan.

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P- Reviewer: Shehata MMM, Shi ZJ **S- Editor**: Tian YL
L- Editor: A **E- Editor**: Liu SQ