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Systemic hemodynamic parameters for intravascular volume assessment during liver transplantation: central venous pressure versus stroke volume variation

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Abstract

The assessment of intravascular volume status in liver transplantation is of utmost importance for a reduction of intraoperative blood loss and transfusion rate and maintenance of a liver graft viability. Conventionally, maintenance of low central venous pressure, which is generally measured from the junction of the superior vena cava and right atrium or lower third of the superior vena cava close to the hepatic veins, had been advocated to facilitate hepatic venous outflow for the prevention of bleeding from the hepatic sinusoids and hepatic veins during hepatic resection surgeries, although the use of central venous pressure for the assessment of intravascular volume status was recommended to be abandoned in general. Despite a significant difference in physiological milieu between non-transplant hepatobiliary surgery and liver transplantation, low central venous pressure was maintained even in liver transplantation and yielded inconsistent results (presence versus absence of renal compromise) from study to study. For the better assessment of intravascular volume status, the use of stroke volume variation, which measures a dynamic change in stroke volume during a respiratory cycle of mechanical ventilation, was widely accepted. Despite the absence of physiologic vascular compliance and resistance in cirrhotic patients undergoing liver transplantation, the stroke volume variation successfully predicted fluid responsiveness better than central venous pressure. However, the use of stroke volume variation in liver transplantation could not produce better clinical outcomes than the use of central venous pressure. Moreover, the effects of stroke volume variation on a reduction in intraoperative blood loss have not been evaluated to date. In conclusion, the usefulness of central venous pressure and stroke volume variation in liver transplantation has not been clearly determined and needs to be evaluated further in the future.

Introduction

The goal of anesthetic management for liver transplantation (LT) is to maintain viability of a liver graft and perfusion of the major organs. Therefore, the assessment of intravascular volume status followed by the maintenance of optimal intravascular volume is mandatory to achieve the goal. In this review, clinical implications of 1) central venous pressure (CVP), which is the traditional static preload parameter and has been commonly used in LT, and 2) stroke volume variation (SVV), which is a newly developed dynamic preload parameter and became popular since the introduction of FloTrac/Vigileo system (Edwards Lifesciences, Irvine, CA, the United States) in the United State in April of 2005, in LT will be discussed.

Central venous pressure

Literally, CVP, a key physiologic estimate of preload for helping to determine intravascular fluid status, is measured from central veins, such as the superior or inferior vena cava. In clinical practice, the superior vena cava accessed through the internal jugular or subclavian vein is more frequently used for the measurement of CVP rather than the inferior vena cava, which can be catheterized from the femoral vein, because the CVP measured from the femoral vein might be affected by the manipulation of the liver during LT, leading to its misinterpretation of intravascular volume status, because the lower third of the superior vena cava close to the junction of the superior vena cava and right atrium, where the tip of a central venous catheter is usually located [1], is closer to the hepatic vein than the femoral vein, and because the internal jugular and subclavian veins are more accessible compared to the femoral vein for monitoring of CVP.

Although using CVP to guide fluid therapy was recommended to be abandoned in general medical practices because it does not correlate with changes in stroke volume index or cardiac index [2], it has been used for a reduction in blood loss during hepatic surgery [3-7]. Theoretically, lowering CVP augments hepatic venous outflow, thereby leading to the prevention of hepatic congestion that increases intraoperative blood loss. However, this physiological background based on non-transplant hepatobiliary surgery should not be generalized to patients undergoing LT for end-stage liver disease, who bear little resemblance to those undergoing hepatic resection. The patients with severe liver disease have impaired autoregulation of a variety of organ systems, which causes changes in mesenteric, renal, and cerebral blood flow [8-10] and have relatively higher model for end-stage liver disease (MELD) score compared to patients undergoing elective liver resection whose average MELD score is 6.5 [11].

In addition, clinical usefulness of low CVP during LT has not been clearly determined. According to the results of a retrospective study, maintenance of CVP below 5 mmHg increased peak postoperative serum creatinine, incidence of postoperative need for hemodialysis, and mortality at 30 days after LT despite a larger number of acutely ill

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patients in the normal CVP group [12]. On the contrary, 2 subsequent studies, one of which compared historical control with prospective group whose CVP was maintained lower than 5 mmHg and the other one of which was a prospective randomized study, demonstrated beneficial effects of a low CVP (a reduced amount of blood transfusion, no renal compromise, and protected liver function) [13,14].

Stroke volume variation

Under mechanical ventilation, stroke volume fluctuates according to a periodic change in intrathoracic pressure during a respiratory cycle. The increased intrathoracic pressure during an inspiratory phase squeezes the blood from the pulmonary vessels to the left ventricle, leading to an increase in stroke volume secondary to an increase in left ventricular preload. Concurrently, right ventricular filling is hindered by the enhanced right ventricular afterload. Then, an expiratory phase ensues with a decrease in intrathoracic pressure, which compensates for the blood flow distributed into the left ventricle during the inspiratory phase by restoring right ventricular filling that results in a reduction in stroke volume by decreasing pulmonary blood flow to the left ventricle [15]. Based upon the above physiologic mechanism, SVV is calculated by a discrepancy between maximum and minimum stroke volumes divided by mean stroke volume during an interval of 20 seconds. Stroke volume for SVV calculation can be measured using an intra-arterial catheter connected to FloTrac/Vigileo system.

Cirrhotic patients have an altered pattern of circulation, i.e., increased cardiac output and decreased peripheral vascular resistance [16,17], which may affect the accurate estimation of vascular compliance and resistance by FloTrac/Vigileo system. Nevertheless, The SVV calculated by the FloTrac/Vigileo system successfully predicted fluid responsiveness in mechanically ventilated patients undergoing LT comparable to pulse pressure variation [18] and SVV calculated using the stroke volume measured from Doppler echocardiography [19]. In addition, SVV obtained from FloTrac/Vigileo system predicted a change in stroke volume due to application of positive end-expiratory pressure after LT [20]. Despite significant discrepancy between radial and femoral arterial pressures, high incidence of hemodynamic instability and hypothermia, and systemic vasodilatation, which might influence the accuracy of the data provided by FloTrac/Vigileo system, its performance to adjust for difference in waveform from different arteries was maintained with no significant difference between SVVs obtained from the radial and femoral arteries during LT [21]. Particularly, femoral SVV performed best for the prediction of fluid responsiveness during an anhepatic phase compared to CVP and pulmonary artery occlusion pressure (PAOP) [22]. Furthermore, SVV showed a better correlation with right ventricular end-diastolic volume index, which was known to be the best filling pressure that represents preload during LT [23,24], than other filling pressures, such as CVP and PAOP [25].

However, despite the better performance for fluid management, SVV did not reduce incidence of acute kidney injury, 30-day and 1-year mortality in living donor liver transplant recipients, when compared to CVP [26]. Recently, it was found that SVV was not helpful to monitor portal hyperperfusion of a liver graft in a postoperative period despite the presence of an optimal range of CVP for the prevention of portal hyperperfusion injury [27]. Moreover, unlike CVP, the effects of SVV on a reduction in blood loss during LT have not been evaluated to date. However, in hepatic resection, SVV was found to achieve comparable outcomes, in terms of blood loss and parenchymal transection time, to those of CVP [28]. Even in patients undergoing living right donor hepatectomy, SVV, but not CVP, was a significant independent predictor of blood loss [29].

Conclusion

Generally, SVV became a better hemodynamic parameter for preload compared to CVP in LT. However, the clinical benefits of using the SVV for a reduction in blood loss have not been clearly determined in LT. Therefore, great efforts to establish evidences for the clinical usefulness of SVV with regard to decrease in intraoperative hemorrhage and maintenance of viability of liver grafts should be made in the future.

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