Clinical Significance of Portal Hypertension Diagnosed With Bedside Ultrasound After Cardiac Surgery

André Y. Denault, MD, PhD,*† William Beaubien-Souligny, MD,‡ Mahsa Elmi-Sarabi, MSc,* Roberto Eljaiek, MD, MSc,§ Ismail El-Hamamsy, MD,|| Yoan Lamarche, MD, MSc,|| Alexandra Chronopoulos, MD, FRCPC,† Jean Lambert, PhD,¶ Josée Bouchard, MD, FRCPC,# and Georges Desjardins, MD*

BACKGROUND: Portal venous flow pulsatility detected by Doppler ultrasound is a sign of congestive heart failure in noncritically ill patients. The assessment of portal and splenic venous flows has never been reported in patients undergoing cardiac surgery.

METHODS: This is a case series performed in patients undergoing cardiac surgery between February 2014 and February 2015 in which portal and/or splenic venous flows were assessed by the attending anesthesiologist during surgery or by the intensivist after surgery using transthoracic echography in 9 patients or transesophageal echocardiography in 5 patients. Data collection was done retrospectively by reviewing intraoperative and postoperative monitoring documents. The technique of assessment is detailed in this article.

RESULTS: We report the abnormal portal and/or splenic venous flow pulsatility from 14 patients perioperatively. At the time of pulsatility detection, patients had a median cumulative fluid balance of 3.8 L (interquartile range: 0–4.6 L) and a median right atrial pressure of 14.0 mm Hg (interquartile range: 12.0–15.5 mm Hg). In some patients (4/14), signs of right ventricular dysfunction on echocardiography and/or right ventricular pressure monitoring were present.

CONCLUSIONS: Doppler evaluation of portal and splenic venous flow using transthoracic echography and transesophageal echocardiography may represent a promising modality to assess end-organ venous congestion in cardiac surgery patients. (Anesth Analg 2017;124:1109–15)

Appropriate use of intravenous fluid therapy is a critical component of patient management during and after cardiac surgery involving cardiopulmonary bypass. When hypovolemia is present, fluid administration increases cardiac output, which maintains systemic perfusion. However, overzealous administration of intravenous fluid coupled with right heart dysfunction can have a negative impact on organ perfusion by promoting, among others, venous congestion. The adverse effects of fluid overload on organ function may be responsible for significant morbidity in critically ill patients, resulting in prolonged mechanical ventilation, delirium, delayed wound healing, and impaired intestinal and hepatic functions.[1,2] In addition, numerous observational studies have linked positive fluid balance and acute kidney injury.[3–5]

Additional tools are currently needed to adequately assess venous congestion to develop preventive strategies. Among these tools, portal venous flow pulsatility has been described as a marker of portal hypertension resulting from congestive heart failure and correlates with elevated right atrial pressure (RAP) and worse New York Heart Association functional class.[6–11] This sign could represent a marker of clinically significant hepatic venous congestion.[12,13]

This article describes our preliminary findings using point-of-care transthoracic echography (TTE) and transesophageal echocardiography (TEE) to assess portal and splenic venous flow pulsatility as potential signs of venous congestion in patients undergoing cardiac surgery.

METHODS

Study Design and Patient Selection

This is a retrospective case study performed in patients undergoing cardiac surgery at the Montreal Heart Institute between February 2014 and February 2015. In this report, we included all cases where pulsatile portal and/or splenic venous flow were detected. The images included were recorded by TEE performed in the operating room by the attending anesthesiologist and by TTE in the intensive care unit by the attending critical care physician. We obtained written informed consent from all patients preoperatively for the use of clinical data and ultrasound images collected during the perioperative period. The research and ethics committee of the Montreal Heart Institute approved the use of the perioperative ultrasound database for this study. The study was performed in accordance with the Declaration of Helsinki and its later amendments. All the examinations were performed by physicians with National Board Certification in TEE and critical care ultrasound certification from the American College of Chest Physicians.

Doppler Ultrasound of the Venous Portal and Splenic Circulation

Assessment of the portal and splenic vein was performed with the patients in the supine position. The splenic vein is a main tributary of the portal vein, and we assumed its flow may provide similar information.

From the *Department of Anesthesia and †Intensive Care Unit, Research Centre, Montreal Heart Institute and Université de Montréal, Montreal, Quebec; ‡Department of Nephrology and §Department of Medicine, and Université de Montréal, Montreal, Quebec; ||Department of Surgery, Montreal Heart Institute and Université de Montréal, Montreal, Quebec; ¶Department of Social and Preventive Medicine, Université de Montréal, Montreal, Quebec; and #Department of Nephrology, Hôpital du Sacré-Coeur de Montréal, Montreal, Quebec.

Accepted for publication October 19, 2016.

Conflicts of Interest: See Disclosures at the end of the article.

Address correspondence to André Y. Denault, MD, PhD, Department of Anesthesia, Montreal Heart Institute, 5000 Belanger St, Montreal, Quebec H1T 1C8, Canada. Address e-mail to andre.denault@umontreal.ca.

DOI: 10.1213/ANE.0000000000001812

Assessment of the portal vein using TTE can be done using a phased array transducer. The technique and interpretation have been previously described.[14] Visualization of the portal vein is done using a phased array transducer positioned in a right posterior-axillary coronal view in the 9th to 11th intercostal spaces as shown in Figure 1A. The portal vein is identified by its position, in the caudal part of the liver, and by its hyperechogenic rim compared with other hepatic vessels as shown in Figure 1B. Confirmation of the portal vein position is done using pulsed-wave Doppler mode to differentiate portal venous flow signature (monophasic to biphasic) from the pattern seen in the hepatic artery (sharp systolic upstroke) and the hepatic veins (triphasic).[14] To obtain an adequate Doppler evaluation of the flow in the portal vein, the ultrasound transducer is positioned as parallel as possible in relation with the portal vein position in the liver parenchyma. Blood flow velocity in the portal vein usually ranges from 10 to 30 cm/s, so Doppler scale should be adjusted to obtain the best velocity differentiation with minimal noise (usually in the 20–40 cm/s or 0.2–0.4 m/s range).

Assessment of portal venous flow using TEE ultrasound can be done during surgery or at the bedside of the critically ill patient. The TEE probe is positioned in the transgastric position by advancing it further than 42 cm from the incisors in most patients. A transverse (short-axis) cut of the liver is obtained by turning the probe to the right side of the patient. A multiplane angle rotation of 90° to 110° leads to a craniocaudal plane of the liver as seen in Figure 1D. The liver parenchyma and vasculature will be inverted in comparison with the TTE examination. Confirmation is done using pulsed-wave Doppler as mentioned above for the TTE approach. As blood flow within the portal vein is directed away from the probe, the waveform seen by TEE ultrasound assessment is a mirror image of the pulsed-wave Doppler seen with TTE ultrasound.

Assessment of splenic vein flow is done with a phased array transducer positioned in the left posterior-axillary position in the 9th to 11th intercostal spaces to obtain a coronal view of the spleen as shown in Figure 2A. The spleen is under the left diaphragm, under and lateral to the stomach. In general, using a coronal view, it will be identified between the hyperechogenic aspect of the diaphragm and the kidney below. It has similar echogenicity as the liver. The position of the splenic vein in the hilum can be confirmed by pulsed-wave Doppler. The normal waveform is negative (blood directed away from the probe) and monophasic or biphasic. An image of the pulsed-wave Doppler waveform can be obtained after adjusting scale (20–40 cm/s) and gain to optimize velocity differentiation.

Assessment of splenic vein flow can be done with TEE via the transgastric approach. A view can be obtained in a fashion similar to the one described above for imaging the port vein, but turning the probe to the left side of the body and performing a multiplane angle rotation of 90° as shown in Figure 2D, to image the splenic hilum. In this view, splenic venous blood will travel in the direction of the probe, and the velocities measured during Doppler examination will be positive. An alternative view can be obtained by turning the probe toward the posterior aspect of the body, while the

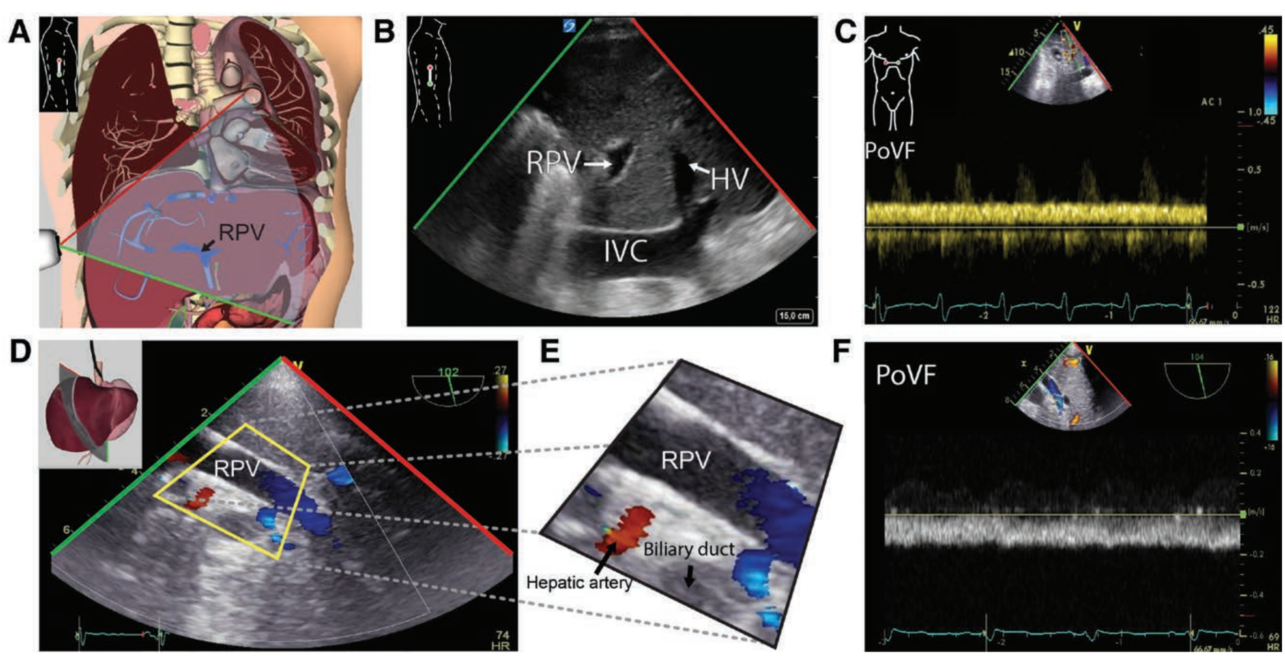

Figure 1. Portal venous flow (PoVF). A, Right portal vein (RPV) position obtained from a posterior axillary view using the Vimedix simulator (CAE Healthcare, Canadian Aviation Electronic (CAE) Healthcare, Saint-Laurent, Canada) and (B) Corresponding 2D transthoracic ultrasound image showing the relative position the RPV, inferior vena cava (IVC), and hepatic vein (HV). C, Doppler assessment of portal venous flow using pulsed-wave Doppler showing continuous monophasic flow with minimal variations. The normal portal venous velocities range from 10 to 30 cm/s and have a positive signal indicating that blood is directed toward the transducer. Note the background pulsatile higher velocity of the hepatic artery, which is in the same direction (Adapted with permission from Denault et al[15]). D, Transesophageal echocardiography (TEE) transgastric craniocaudal view of the liver shows (E) the main branch of the RPV on 2D ultrasound. F, Normal portal venous flow (pulsed-wave Doppler) examinations are away from the transducer (negative velocity).

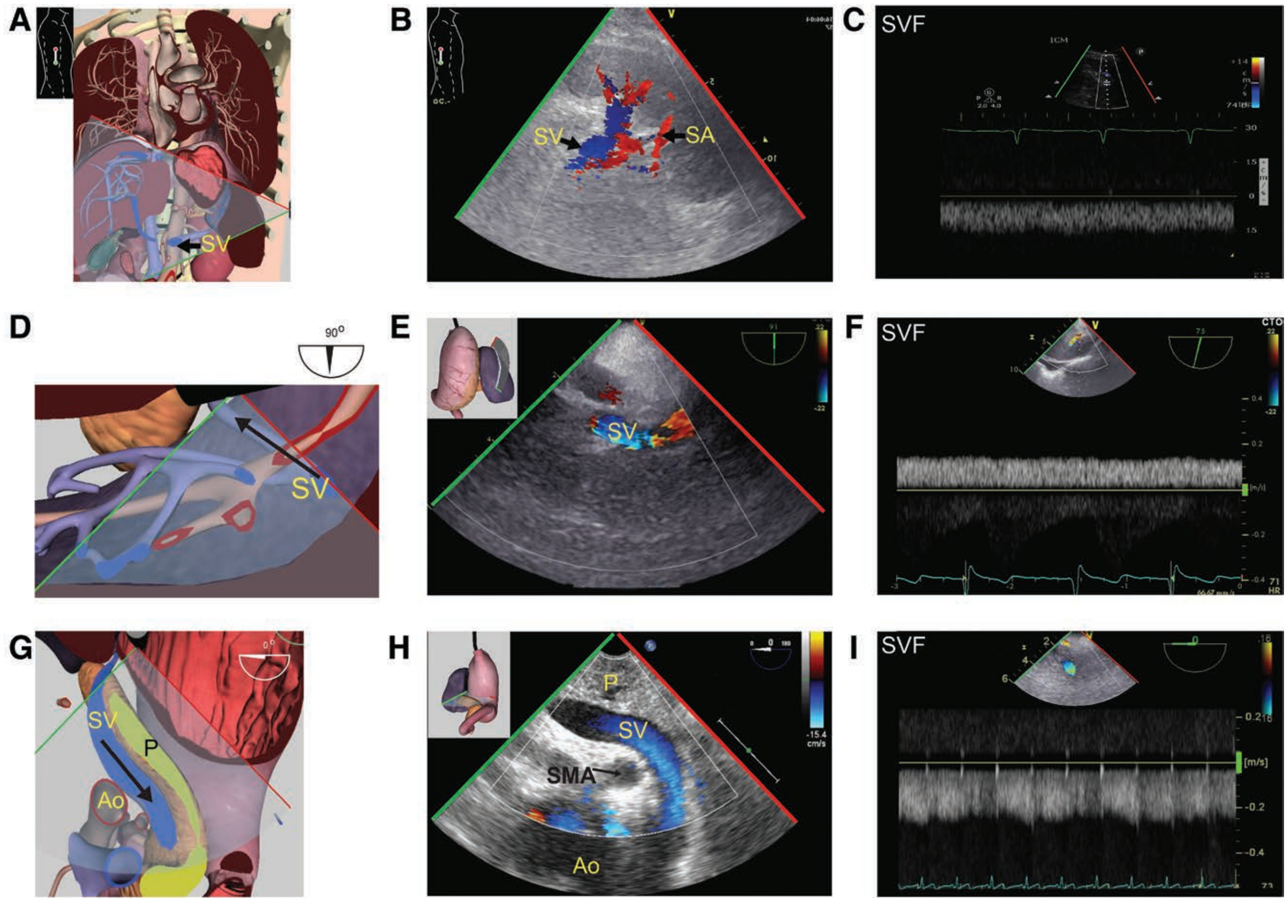

Figure 2. Splenic vein flow (SVF). A, Splenic vein (SV) position obtained from a posterior axillary view using the Vimedix simulator (CAE Healthcare) and (B) corresponding 2D transthoracic ultrasound image showing the relative position of the SV in relationship with the splenic artery (SA). C, Doppler assessment of splenic venous flow using pulsed-wave Doppler showing continuous monophasic flow with minimal variations. D, Transesophageal echocardiography (TEE) transgastric view at 90° with left-sided rotation shows the SV near the splenic hilum (Vimedix simulator; CAE Healthcare). E, Doppler assessment of splenic venous flow with TEE. F, Normal splenic vein flow is toward the TEE probe in this view. G, TEE transgastric view at 0° shows the splenic vein posterior to the descending aorta (Ao) (Vimedix simulator; CAE Healthcare). H, Doppler assessment of splenic venous flow with TEE. I, In this view, the normal splenic vein flow is away from the probe. P indicates pancreas; SMA, superior mesenteric artery (Adapted with permission of Denault et al[15]).

multiplane angle is maintained at 0°. In this position, the splenic vein is located anterior to the descending aorta as shown in Figure 2G. From this view, the venous flow will be directed away from the TEE probe and the velocities will be negative.

Normal portal and splenic venous flows assessed by pulsed-wave Doppler are hepatopetal and monophasic or biphasic.[16] Variations in the portal venous flow during the cardiac cycle are due to the transmission of hepatic venous pressure through the liver sinusoids.[17] When the RAP is increased, variation of portal venous flow during the cardiac cycle is more pronounced and becomes pulsatile. The pulsatility fraction (PF) is used to quantify the degree of pulsatility: PF (%) = 100 $[(V_{max} - V_{min})/(V_{max})]$.

Where V_{max} is the maximal blood velocity and V_{min} is the minimal blood velocity during the cardiac cycle. Abnormal portal venous flow pulsatility has been described as a PF of ≥50% in heart failure patients.[7,9–11,18]

It should be noted that simultaneous ECG tracing is often not available when using the factory preset for abdominal examination on point-of-care ultrasound devices as it is the case for some of the images presented in this report. Simultaneous ECG is available using the cardiac examination setting.

Splenic vein flow pulsatility has not been studied in patients with chronic heart failure but is expected to exhibit the same pattern because it is a major tributary of the main portal vein. A pulsatile flow pattern in the splenic vein is associated with increased risk of variceal bleeding in cirrhotic patients.[19]

Definitions and Statistical Analysis

Preoperative risk score EuroSCORE II was calculated using an online calculator.[20] Although the cutoff point to describe portal and splenic flow as pulsatile is not defined in the critical care population, we considered a PF of more than 50% as pulsatile, based on reports in patients with chronic heart failure. Continuous variables are presented as mean ± SD or median and interquartile range (IQR) and compared using *t* test or Mann-Whitney *U* test, where appropriate.

RESULTS

Portal or splenic vein pulsatility was observed in 14 cardiac surgery patients. Our sample included 8 males and 6 females with a median age of 58 years (IQR: 45–71 years). The median EuroSCORE II was 5.1 (IQR: 3.2–8.7), and the median cardiopulmonary bypass time was 104 minutes (IQR: 82–155 minutes).

Patients' characteristics are presented in Table. Abnormal portal or splenic vein pulsatility was detected intraoperatively or upon admission in 7 patients (50%) and up to day 11 in patient 7. The median of PF at the first detection was 73% (IQR: 51%–100%). The median number of days where portal or splenic venous flows were assessed per patient was 3 (IQR: 2.00–4.25) and 2 patients had 1 assessment only. Figure 3 shows an example of pulsatile portal venous flow in 2 consecutive assessments in patient 1 using TTE. Figure 4 shows the right ventricular pressure waveform and the corresponding pulsatile splenic vein flow in patient 4 using TEE.[21] The median cumulative fluid balance before the detection of portal or splenic venous pulsatility was 3.8 L (IQR: 0–4.6 L). Patients 5, 7, and 9 were known for pulmonary hypertension (mean pulmonary artery pressure ≥25 mm Hg) before surgery. Features of pulmonary hypertension, right ventricular dysfunction, and elevated RAP (Table) were observed in patients in association with an abnormal pulsatile portal/splenic venous flow. Portal and splenic venous flow measurements were done concomitantly in 7 assessments showing similarity in 6 of them, with less than 10% difference in PF, and 1 showing a 50% pulsatile portal venous flow but a 40% splenic venous flow.

Repeated assessment of portal venous flow identified significant changes in portal venous flow in 3 patients. In patient 2, portal venous flow was nonpulsatile after surgery (postoperative day 0) but became pulsatile on day 2 after a positive fluid balance of 1.9 L. In patients 5 and 6, portal venous flow was pulsatile during the first assessment and became nonpulsatile after a negative fluid balance of −2.4 L and −4.0 L, respectively.

DISCUSSION

This series represents our preliminary experience using intraoperative and postoperative ultrasound assessment of the portal and splenic venous flows for the evaluation of venous congestion in cardiac surgery. Portal venous flow pulsatility has never been reported using point-of-care ultrasonography in an intensive care unit setting. Furthermore, this report describes the use of extracardiac TEE ultrasound to assess portal and splenic veins Doppler signals during cardiac surgery.

Significant pulsatility in the portal venous circulation is suggestive of cardiogenic portal hypertension secondary to pulmonary hypertension, right ventricular failure, and tricuspid regurgitation.[14] A recent study by Styczynski et al[12] showed that a PF of 50% or more was the best predictive factor associated with an increase in serum bilirubin in heart failure patients. This suggests an impairment of hepatic function caused by venous congestion. Venous congestion of the digestive tract in congestive heart failure patients is thought to be responsible for the cardiointestinal syndrome characterized by a proinflammatory state.[22] Therefore, portal or splenic vein Doppler interrogation could be considered a potential echographic sign of cardiointestinal interaction.

Portal venous flow pulsatility has been described as a PF of ≥50% in heart failure patients.[7,9–11,18] In congestive heart failure patients, minimal portal velocity is found in systole approximately 0.2 seconds after the QRS complex.[23] This is consistent with the observed reduction of blood flow velocity in the hepatic veins during systole (systolic to diastolic ratio less than 1) observed in congestive heart failure because of impaired systolic filling of the right atrium.[24] The relationship between hepatic venous flow and portal venous flow has been also reported recently by several authors.[13,21,25] It must be noted that pulsatile portal venous flow has also been reported in healthy individuals with low body mass index (<20) and therefore must be interpreted carefully in thin patients.[16] The impact of expiration, inspiration, and Valsalva maneuver has been studied in 11 healthy individuals, and no differences were seen in the mean minimal/maximal velocity ratio during the respiratory phases and Valsalva maneuver.[23] It must be noted that the effect of atrial fibrillation and of positive pressure ventilation on the portal or splenic venous flows have not been described and might have influenced portal venous flow in our patients. Although the present study does not describe the normal mean portal or splenic venous flow after cardiac surgery, a PF of more than 50% could represent an abnormal finding based on the available literature.

Pulsatile portal or splenic venous flow is likely to be associated with fluid overload in the context of cardiac dysfunction leading to venous hypertension. Although repeated assessments showing changes in portal and splenic venous flows in relation with cumulative fluid balance support this hypothesis, it must be noted that other factors such as vasopressor/inotrope use, change in the position of the patient, and positive pressure ventilation could have affected portal and splenic venous flows.

Several echocardiographic and hemodynamic signs of right ventricular dysfunction were noted in our patients. These included the square root sign on right ventricular pressure monitoring (patients 4 and 12) and visualization of right ventricular hypokinesia and dilation (patients 4 and 12), elevation of PAP, and severe tricuspid regurgitation (patient 11).

Assessment of the portal and splenic venous flows may be useful in cardiac surgery patients. Assessment of venous congestion is currently limited in the clinical setting. The presence of edema on physical examination is known to be multifactorial.[26] Absolute RAP measurements in critically ill patients are known to be prone to technical caveats and are therefore misleading.[27] The development of new tools is needed to adequately assess the impact of venous congestion on end organs. Pulsatile renal venous flow has been recently described as a sign of renal congestion.[28] Interestingly, in this study involving 217 patients with heart failure, an abnormal monophasic renal venous Doppler signal predicted death or rehospitalization at 1 year better than RAP.[28] In the accompanying editorial, Tang and Kitai[13] suggested that portal venous flow pulsatility would correlate with those findings. Prevention and treatment of venous congestion based on markers of fluid overload and right heart failure could lead to significant improvements in overall tissue perfusion. The extent through which a restrictive fluid therapy in selected patients could help to prevent

Table. Clinical Context Associated With the Discovery of Portal and/or Splenic Venous Flow Pulsatility in the Reported Patients

Patient	Age/Sex	EuroSCORE II (%)	Procedure	LVEF (%)	Context	Modality Used/ Structure Studied	Detected at POD	PF (%)	Mean RAP Measured[a] (mm Hg)	Vasopressors and/or Inotropes	Fluid Balance[b] (mL)
1	50/M	4.0	Bentall	61	Prolonged CPB (273 min)	TTE-P/S	1	100	12	None	7347
2	44/M	NA	Cardiac transplant for DCM	N	Normal portal venous flow at ICU admission	TTE-P	2	100	12	I	1869
3	71/F	11.3	AVR, aortic thrombus removal	25	Clinical deterioration within 24 h of surgery	TTE-P/S	2	100	15	NE, E, MIL	1382
4	73/F	1.97	AVR	55	Patient-prosthesis mismatch, important clinical deterioration within 24 h of surgery. Appearance of a square root pattern on RV pressure monitoring and severe RV dysfunction. Death on POD 1	TEE-P/S	0	73	14	NE, E, V	3451
5	72/F	24.8	CABG	25	Pulmonary HTN (PAMP = 42 mm Hg) before surgery	TTE-P	0	100	12	NE	2651
6	63/M	6.1	AVR for endocarditis	35	Hemodynamic instability during surgery and prolonged CPB (286 min)	TTE-P	2	100	15	NE, D	2687
7	32/M	5.96	TVR	N	Pulmonary HTN (PAMP = 60 mm Hg) before surgery	TTE-P	11	50	14	None	9780
8	67/F	1.2	LVAD implantation for DCM	15	Cardiogenic shock and multiple episode of VT before surgery	TTE-P	0	50	15.5	NE, E, MIL	531
9	46/M	NA	Cardiac transplant HCM	N	Pulmonary HTN (PASP = 95 mm Hg before surgery) with RV dysfunction	TEE-P	OR	51	17.5	NE, E, MIL	−381
10	53/M	NA	Cardiac transplant for DCM	N		TTE-P	0	73	15.5	NE, E, V	8255
11	24/F	4.1	AVR, Ross procedure	56	Severe tricuspid regurgitation and pulmonary HTN (PASP = 55 mm Hg)	TTE-P	2	100	13.5	NE, MIL	4425
12	70/M	5.1	CABG	N	Episode of ventricular fibrillation during surgery, RV dysfunction	TEE-P	0	50	13.5	NE, E, V, MIL	1590
13	44/F	2.4	TVR	35	Superior vena cava stenosis	TEE-P/S	OR	51	16.5	NE, MIL	80
14	85/M	33.9	AVR, TVP, MVP, CABG	53	Tamponade after surgery needing surgical drainage, systolic LV dysfunction, and severe RV dilation	TEE-P	1	57	6	NE, E, V, MIL	8365

Abbreviations: AVR, aortic valve replacement; CABG, coronary artery bypass graft; CPB, cardiopulmonary bypass; D, dobutamine; DCM, dilated cardiomyopathy; E, epinephrine; HCM, hypertrophic cardiomyopathy; HTN, hypertension; ICU, intensive care unit; I, isoproterenol; LV, left ventricle; LVAD, left ventricular assist device; LVEF, left ventricular ejection fraction; M, male; MIL, milrinone; MVP, mitral valve plasty; N, LVEF reported as normal; NE, norepinephrine; OR, portal pulsatility was found during surgery; P, portal vein; PAMP, pulmonary artery mean pressure; PASP, pulmonary artery systolic pressure; PF, pulsatility fraction; POD, postoperative day; RAP, right atrial pressure; RV, right ventricle; S, splenic vein; TEE, transesophageal echocardiography; TTE, transthoracic echocardiography; TVP, tricuspid valve plasty; TVR, tricuspid valve replacement; V, vasopressin; VT, ventricular tachycardia.

[a]Mean RAP on the day of the assessment.

[b]Cumulative fluid balance before portal pulsatility was found.

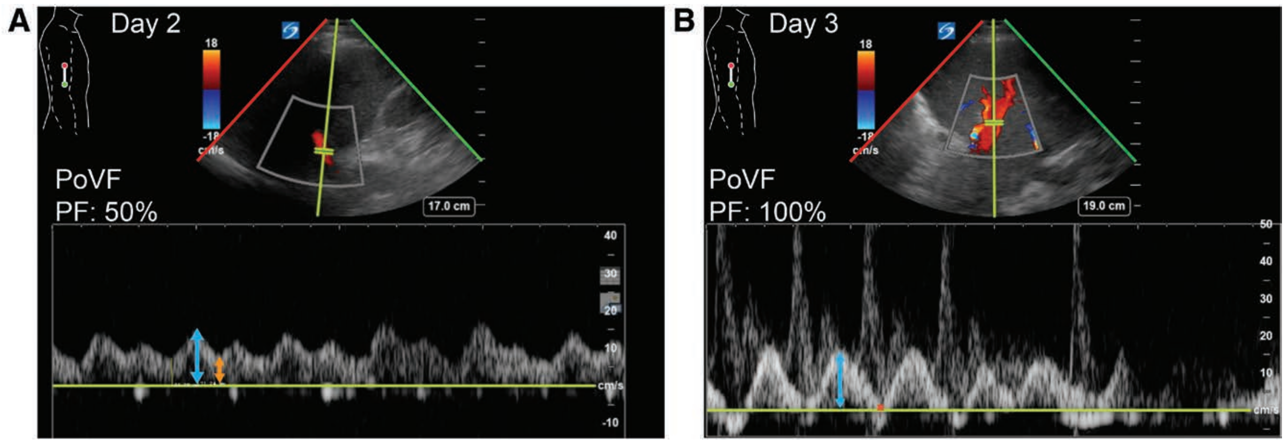

Figure 3. Pulsatile portal venous flow (PoVF). Patient 1 pulsed-wave Doppler of the portal vein (A) 2 days after intensive care unit (ICU) admission showing pulsatility (mean pulsatility fraction [PF] of 50%) and (B) 3 days after ICU admission showing severe pulsatility (PF of 100%). Note the Doppler signal of the hepatic artery visible in the background.

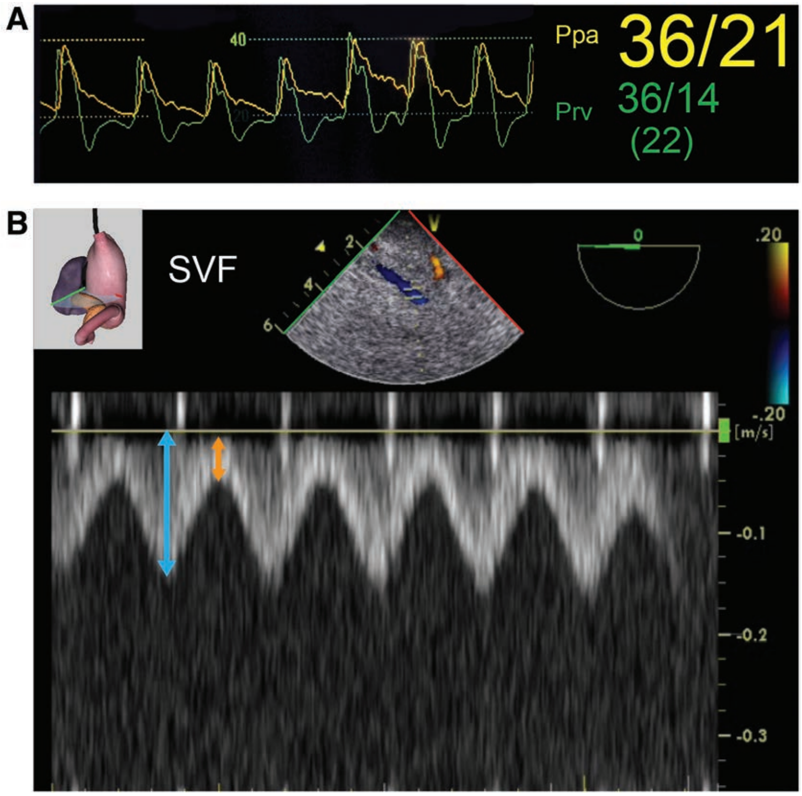

Figure 4. Signs of right ventricular dysfunction and portal pulsatility. A, Square root sign on right ventricular pressure (Prv) monitoring during surgery in patient 4 with (B) severe splenic venous flow (SVF) (pulsatility fraction [PF] of 73%) using transesophageal echocardiography. Ppa indicates pulmonary artery pressure.

related complications is supported by an increasing number of studies confirming the advantage of this strategy compared with the liberal use of fluids.[29]

The present report has several limitations. The small convenience sample without the use of a control group precludes any conclusions about how the presence of portal and/or splenic venous flow pulsatility could be a risk factor for adverse outcomes because of venous congestion. However, in patients with chronic heart failure, the severity of renal venous congestion correlated with mortality and rehospitalization.[28] Although right heart failure and a positive fluid balance could be contributing factors, other factors such as patient body habitus, position during assessment, and positive pressure ventilation could influence blood flow in these vessels and could thus lead to a misinterpretation of the Doppler waveform. Consequently, the assessment presented in this preliminary report should be used as an investigational tool at the present time and not to guide fluid balance management. The value of portal and splenic venous flow Doppler assessment should be studied in prospective studies before this assessment can be implemented in routine care.

DISCLOSURES

Name: André Y. Denault, MD, PhD.
Contribution: This author helped design the study, conduct the study, collect the data, analyze the data, and prepare the manuscript.

Conflicts of Interest: André Y. Denault was the Speaker for Covidien and CAE Healthcare.
Name: William Beaubien-Souligny, MD, FRCPC.
Contribution: This author helped design the study, conduct the study, collect the data, analyze the data, and prepare the manuscript.
Conflicts of Interest: None.
Name: Mahsa Elmi-Sarabi, MSc.
Contribution: This author helped prepare the manuscript.
Conflicts of Interest: None.
Name: Roberto Eljaiek, MD.
Contribution: This author helped prepare the manuscript.
Conflicts of Interest: None.
Name: Ismail El-Hamamsy, MD.
Contribution: This author helped prepare the manuscript.
Conflicts of Interest: None.
Name: Yoan Lamarche, MD, MSc.
Contribution: This author helped prepare the manuscript.
Conflicts of Interest: None.
Name: Alexandra Chronopoulos, MD, FRCPC.
Contribution: This author helped prepare the manuscript.
Conflicts of Interest: None.
Name: Jean Lambert, PhD.
Contribution: This author helped analyze the data.
Conflicts of Interest: None.
Name: Josée Bouchard, MD, FRCPC.
Contribution: This author helped prepare the manuscript.
Conflicts of Interest: None.
Name: Georges Desjardins, MD.
Contribution: This author helped prepare the manuscript.
Conflicts of Interest: None.
This manuscript was handled by: Nikolaos J. Skubas, MD, DSc, FACC, FASE.

REFERENCES

1. Prowle JR, Echeverri JE, Ligabo EV, et al. Fluid balance and acute kidney injury. *Nat Rev Nephrol*. 2010;6:107–115.
2. Beaubien-Souligny W, Bouchard J, Desjardins G, et al. Extracardiac signs of fluid overload in the critically ill cardiac patient: a focused evaluation using bedside ultrasound. *Can J Cardiol*. 2017;33:88–100.
3. Payen D, de Pont AC, Sakr Y, et al; Sepsis Occurrence in Acutely Ill Patients (SOAP) Investigators. A positive fluid balance is associated with a worse outcome in patients with acute renal failure. *Crit Care*. 2008;12:R74.
4. Bouchard J, Soroko SB, Chertow GM, et al; Program to Improve Care in Acute Renal Disease (PICARD) Study Group. Fluid accumulation, survival and recovery of kidney function in critically ill patients with acute kidney injury. *Kidney Int*. 2009;76:422–427.
5. Mullens W, Abrahams Z, Francis GS, et al. Importance of venous congestion for worsening of renal function in advanced decompensated heart failure. *J Am Coll Cardiol*. 2009;53:589–596.
6. Catalano D, Caruso G, DiFazzio S, et al. Portal vein pulsatility ratio and heart failure. *J Clin Ultrasound*. 1998;26:27–31.
7. Duerinckx AJ, Grant EG, Perrella RR, et al. The pulsatile portal vein in cases of congestive heart failure: correlation of duplex Doppler findings with right atrial pressures. *Radiology*. 1990;176:655–658.
8. Hosoki T, Arisawa J, Marukawa T, et al. Portal blood flow in congestive heart failure: pulsed duplex sonographic findings. *Radiology*. 1990;174:733–736.
9. Shih CY, Yang SS, Hu JT, et al. Portal vein pulsatility index is a more important indicator than congestion index in the clinical evaluation of right heart function. *World J Gastroenterol*. 2006;12:768–771.
10. Hu JT, Yang SS, Lai YC, et al. Percentage of peak-to-peak pulsatility of portal blood flow can predict right-sided congestive heart failure. *World J Gastroenterol*. 2003;9:1828–1831.
11. Rengo C, Brevetti G, Sorrentino G, et al. Portal vein pulsatility ratio provides a measure of right heart function in chronic heart failure. *Ultrasound Med Biol*. 1998;24:327–332.
12. Styczynski G, Milewska A, Marczewska M, et al. Echocardiographic correlates of abnormal liver tests in patients with exacerbation of chronic heart failure. *J Am Soc Echocardiogr*. 2016;29:132–139.
13. Tang WH, Kitai T. Intrarenal venous flow: a window into the congestive kidney failure phenotype of heart failure? *JACC Heart Fail*. 2016;4:683–686.
14. McNaughton DA, Abu-Yousef MM. Doppler US of the liver made simple. *Radiographics*. 2011;31:161–188.
15. Denault AY, Couture P, Lamarche Y, et al. *Basic Transesophageal and Critical Care Ultrasound*. Boca Raton, FL: CRC Press; 2017.
16. Gallix BP, Taourel P, Dauzat M, et al. Flow pulsatility in the portal venous system: a study of Doppler sonography in healthy adults. *AJR Am J Roentgenol*. 1997;169:141–144.
17. Gorka W, Gorka TS, Lewall DB. Doppler ultrasound evaluation of advanced portal vein pulsatility in patients with normal echocardiograms. *Eur J Ultrasound*. 1998;8:119–123.
18. Goncalvesova E, Lesny P, Luknar M, et al. Changes of portal flow in heart failure patients with liver congestion. *Bratisl Lek Listy*. 2010;111:635–639.
19. Maruyama H, Ishihara T, Ishii H, et al. Blood flow parameters in the short gastric vein and splenic vein on Doppler ultrasound reflect gastric variceal bleeding. *Eur J Radiol*. 2010;75:e41–e45.
20. Nashef SA, Roques F, Sharples LD, et al. EuroSCORE II. *Eur J Cardiothorac Surg*. 2012;41:734–744.
21. Haddad F, Elmi-Sarabi M, Fadel E, et al. Pearls and pitfalls in managing right heart failure in cardiac surgery. *Curr Opin Anaesthesiol*. 2016;29:68–79.
22. Sundaram V, Fang JC. Gastrointestinal and liver issues in heart failure. *Circulation*. 2016;133:1696–1703.
23. Abu-Yousef MM. Normal and respiratory variations of the hepatic and portal venous duplex Doppler waveforms with simultaneous electrocardiographic correlation. *J Ultrasound Med*. 1992;11:263–268.
24. Scheinfeld MH, Bilali A, Koenigsberg M. Understanding the spectral Doppler waveform of the hepatic veins in health and disease. *Radiographics*. 2009;29:2081–2098.
25. Amsallem M, Kuznetsova T, Hanneman K, et al. Right heart imaging in patients with heart failure: a tale of two ventricles. *Curr Opin Cardiol*. 2016;31:469–482.
26. Agarwal R, Andersen MJ, Pratt JH. On the importance of pedal edema in hemodialysis patients. *Clin J Am Soc Nephrol*. 2008;3:153–158.
27. Marik PE, Baram M, Vahid B. Does central venous pressure predict fluid responsiveness? A systematic review of the literature and the tale of seven mares. *Chest*. 2008;134:172–178.
28. Iida N, Seo Y, Sai S, et al. Clinical implications of intrarenal hemodynamic evaluation by Doppler ultrasonography in heart failure. *JACC Heart Fail*. 2016;4:674–682.
29. Murphy GJ, Pike K, Rogers CA, et al; TITRe2 Investigators. Liberal or restrictive transfusion after cardiac surgery. *N Engl J Med*. 2015;372:997–1008.