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Editorial

Portal Vein Pulsatility After Cardiac Surgery—Who Cares?

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See article by Benkreira et al., pages 1134–1141 of this issue.

Congestive Encephalopathy: New Horizons in Postcardiotomy Delirium

Delirium is a clinical syndrome defined by transient neurological disturbances in attention, cognition, and consciousness.[1] Postoperative delirium affects more than 25% to 50% of patients undergoing cardiac surgery, is often multifactorial, and has been associated with increased morbidity and mortality.[1-3] Studies report prolonged lengths of hospital stay, as well as an increased risk of cognitive and functional decline requiring rehabilitation or long-term facility care due to delirium.[1-3] Common causes of delirium in the postsurgical setting include need for circulatory arrest, duration of surgery or cardiopulmonary bypass time, chronic illness, sleep deprivation, fever, infection, metabolic imbalance, organ dysfunction, drugs, and toxins.[3] In the present issue of the *Journal*, Benkreira et al.[2] report a novel association between markers of venous congestion and increased risk of delirium and encephalopathy in their retrospective and prospective cohorts of 237 and 145 patients postcardiac surgery, respectively. For instance, portal vein flow pulsatility was associated with an increased risk of delirium (odds ratio [OR], 2.63), cognitive dysfunction (OR, 2.10), and asterixis (OR, 5.19).[2] In turn, cognitive dysfunction was associated with higher N-terminal pro B-type natriuretic peptide levels (OR, 4.03) and cerebral oximetry desaturations (OR, 2.54).[2] In another recent study, Mailhot et al.[4] reported an association of cumulative fluid balance with increased occurrence of delirium (OR, 1.20; confidence interval [CI], 1.066-1.355; $P = 0.003$). Cumulative fluid balance and central venous pressure have been shown to be independent predictors of portal pulsatility.[2] Taken together, these novel findings underscore the need to understand the etiological contributions of fluid status leading to delirium in the critically ill patient cohort.

Portal Vein Flow Pulsatility: A Potential Marker of Cerebral Dysfunction?

Perioperative fluid management in the intensive care unit requires a delicate balance between optimizing adequate organ perfusion while preventing organ dysfunction due to vascular congestion. In critically ill patients, venous congestion leading to increased interstitial edema has been linked to poor tissue perfusion and multisystem organ dysfunction, including of lung, heart, gut, liver, and kidney parenchyma.[2,5-7] Rather than draining into the heart, the portal vein drains venous blood into another capillary system, namely, the hepatic sinusoids.[8] Decreased compliance of the hepatic vascular bed, increased blood volume in the liver in the setting of right ventricular dysfunction, and severe tricuspid regurgitation can result in portal venous hypertension.[5] Portal venous hypertension in turn can contribute to hepatic encephalopathy resulting from increased hepatic release of neurotoxic products into the circulation. Portal venous hypertension can be detected on the basis of enhanced pulsatility in portal vein flow, defined in this study by a pulsatility fraction of $\geq 30\%$, taken as the difference between peak and minimal flow velocities divided by the peak flow velocity (Fig. 1).[2] Portal vein flow is normally directed toward the liver and monophasic (<30% amplitude phasicity) with a constant velocity throughout the cardiac cycle.[8] Portal vein flow pulsatility can be detected and quantified by noninvasive Doppler ultrasound.[8]

Building on prior observations demonstrating pulsatile portal flow as a marker of altered intrarenal perfusion and right ventricular dysfunction after cardiac surgery,[5-7] Benkreira et al.[2] explore for the first time the significance of portal venous congestion on cerebral tissue function. They not only report significant associations between the pulsatility fraction and the various measures of delirium but also demonstrate a significant correlation between pulsatility fraction and cognitive dysfunction (OR, 1.2; CI, 1.08-1.32; $P = 0.001$), as well as the severity of cognitive dysfunction based on the delirium index score (β-estimate 0.13; CI, 0.03-0.23; $P = 0.01$) for each 10% increase in portal pulsatility.[2] Although there is limited specificity (59%) for delirium in patients postcardiotomy using the 30% pulsatility fraction cutoff, Benkreira et al. provide prospective validation of their retrospective

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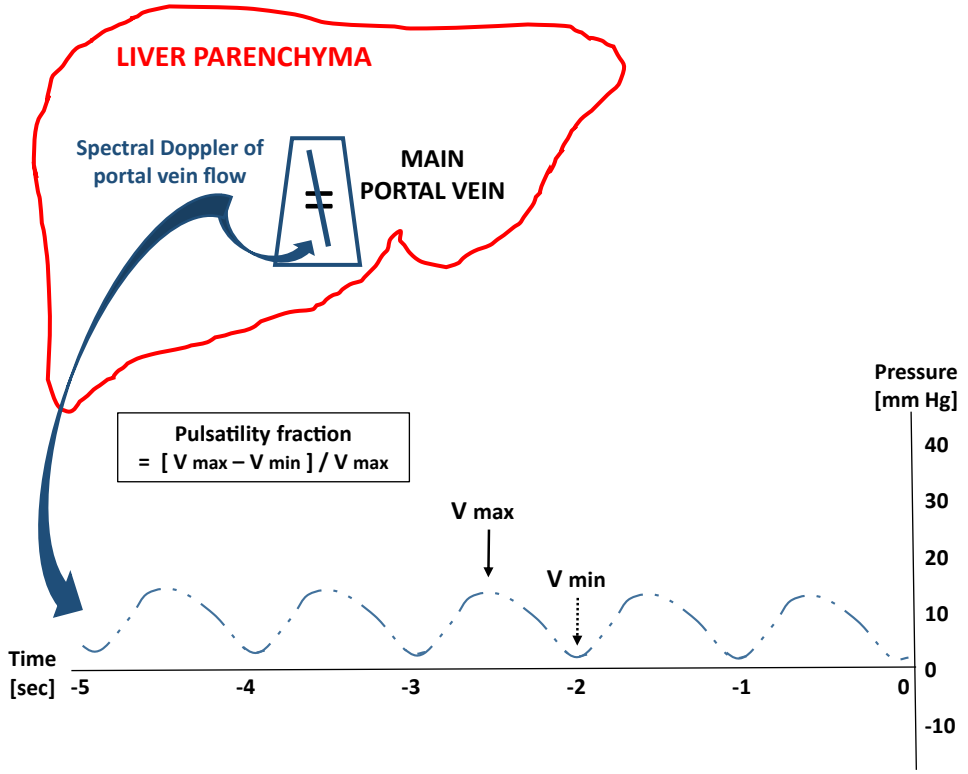

Figure 1. Ultrasonography of portal vein using spectral Doppler tracing. Pulsatility fraction is determined by the difference between the peak (V max) and minimal (V min) flow velocities in the portal vein divided by the peak velocity. Portal vein pulsatility is defined by a pulsatility fraction of $\geq$ 30%.

study finding of an association between portal pulsatility and delirium, with ORs of 2.63 and 2.69 in their prospective and retrospective study cohorts, respectively. Moreover, they correlate portal pulsatility with relative cerebral desaturation, hypothesizing that "venous congestion may reduce brain oximetry by increasing the relative contribution of venous blood to the oximetry reading and by directly impairing brain perfusion via an elevation of intra-cranial pressure."[2] These findings highlight the utility of enhanced portal pulsatility as a specific measure of cerebral dysfunction in critically ill patients experiencing delirium in their subset of patients postcardiotomy. It is conceivable that increased interstitial edema within the brain can increase intracranial pressures such that intracranial hypertension can contribute to cerebral encephalopathy.

Why Should Portal Pulsatility Even Matter?

There are several limitations to this study. First, the findings are generated by retrospective and prospective analyses of small study cohorts of patients undergoing cardiac surgery at a single center. Whether this study can be generalized to all cardiac surgery patients or even critically ill patients in any intensive care unit requires added experimental testing. Second, the authors[2] report that only "155 (65%) patients had at least one assessment showing portal pulsatility during their ICU stay." How reliable is a single finding of enhanced portal pulsatility? Why should a single measure of portal pulsatility be enough to diagnose cerebral end-organ dysfunction and guide the management of delirium in a patient? Quite likely, portal pulsatility would have to be considered in the clinical context of the patient who manifests other signs of delirium and in whom this serves as a tool to rule in or out the contribution of venous congestion to congestive encephalopathy. Third, although the study findings are intriguing, they are all still only hypothesis-generating correlations. An assessment of intracranial hypertension and cerebral vascular compliance with the use of transcranial Doppler, optic nerve sheath diameter ultrasonography, computed tomography, or magnetic resonance imaging in these patients postcardiotomy might have provided further mechanistic insight into how enhanced portal vein flow pulsatility could serve as a marker of cerebral end-organ dysfunction or even as a cause of delirium.[1,2,9,10] An important question that remains to be answered is whether diuresis and normalization of portal vein pulstatility result in an improvement of delirium. If not, portal vein pulsatility might simply be a marker for advanced volume overload or long-standing hepatic congestion that slows anesthetic drug clearance postoperatively. Despite these limitations, Denault's group[11] has further clarified our understanding of portal vein pulsatility as a potential surrogate of congestive encephalopathy and a marker of other end-organ dysfunction. Although their observations may not be broadly applicable as yet, they open the door to future evaluation and introduce a potentially valuable noninvasive tool to assist in the management of a subset of patients with delirium postcardiotomy.

Conclusions

In an era when there is a burgeoning number of patients undergoing cardiac surgery, the need to improve patient outcomes requires cost-effective interventions to reduce postsurgical complications. Certainly, identification of the

underlying cause of delirium in the patients postcardiotomy may help alleviate the multitude of associated adverse outcomes. To date, the empiric use of psychoactive medications has "failed to reduce the rate of delirium in clinical trials."[2] Delirium is clearly multifactorial. Portal vein pulsatility, measured by point-of-care ultrasonography, can serve as another tool to guide the intensive care management of delirium due to venous congestion in patients postcardiotomy. The potential to use this bedside analysis in the management of venous congestion and its negative impact on cerebral or other end-organ function are highlighted by the extensive work of Denault's group.[11] Future studies still need to analyze the potential broad-scoping application of portal pulsatility in guiding delirium management in all critically ill patient cohorts. This intriguing work opens the door for additional exploration.

Disclosures

Dr Tong declares speaking honorarium and consultant agreement with Abiomed and Abbott. The other authors have no relevant conflicts of interest to disclose.

References

1. Rasulo FA, Bertuetti R, Robba C, et al. The accuracy of transcranial Doppler in excluding intracranial hypertension following acute brain injury: a multicenter prospective pilot study. Crit Care 2017;21:44.

2. Benkreira A, Beaubien-Souligny W, Mailhot T, et al. Portal hypertension is associated with congestive encephalopathy and delirium after cardiac surgery. Can J Cardiol 2019;35:1134-41.

3. Mangusan RF, Hooper V, Denslow SA, et al. Outcomes associated with postoperative delirium after cardiac surgery. Am J Crit Care 2015;24:156-63.

4. Mailhot T, Cossette S, Lambert J, et al. Delirium after cardiac surgery and cumulative fluid balance: a case-control cohort study. J Cardiothorac Vasc Anesth 2019;33:93-101.

5. Eljaiek R, Cavayas YA, Rodrigue E, et al. High postoperative portal venous flow pulsatility indicates right ventricular dysfunction and predicts complications in cardiac surgery patients. Br J Anaesth 2019;122:206-14.

6. Beaubien-Souligny W, Eljaiek R, Fortier A, et al. The association between pulsatile portal flow and acute kidney injury after cardiac surgery: a retrospective cohort study. J Cardiothorac Vasc Anesth 2018;32:1780-7.

7. Beaubien-Souligny W, Benkreira A, Robillard P, et al. Alterations in portal vein flow and intrarenal venous flow are associated with acute kidney injury after cardiac surgery: a prospective observational cohort study. J Am Heart Assoc 2018;7:e009961.

8. McNaughton DA, Abu-Youself MM. Doppler US of the liver made simple. Radiographics 2011;31:161-88.

9. Dubourg J, Javouhey E, Geeraerts T, et al. Ultrasonography of optic nerve sheath diameter for detection of raised intracranial pressure: a systematic review and meta-analysis. Intensive Care Med 2011;37:1059-68.

10. Cardim D, Griesdale DE, Ainslie PN, et al. A comparison of non-invasive versus invasive measures of intracranial pressure in hypoxic ischaemic brain injury after cardiac arrest. Resuscitation 2019;137:221-8.

11. Denault AY, Beaubien-Souligny W, Elmi-Sarabi M, et al. Clinical significance of portal hypertension diagnosed with bedside ultrasound after cardiac surgery. Anesth Analg 2017;124:1109-15.