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# CEREBRAL VENOUS FLOW MONITORING DURING SOLID ORGAN TRANSPLANTATION AND MAJOR SURGERY

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## Background

Transcranial Color-Coded Doppler (TCCD) is widely used for perioperative neuromonitoring in liver transplantation (LT). While arterial cerebral flow is well characterized, the role of cerebral venous flow (CVF) monitoring remains unexplored. Impaired venous return due to mechanical ventilation, multiple central venous catheters, or increased right atrial pressure after graft reperfusion may contribute to cerebral congestion and intracranial hypertension. Comparable pathophysiological conditions may occur during major surgical procedures associated with elevated thoraco-abdominal pressure or steep Trendelenburg positioning. Laparoscopic or robotic urologic and gynecologic surgeries represent typical examples, wherein intracranial pressure may further rise as a consequence of both reduced cerebral venous drainage and increased arterial inflow secondary to patient positioning and hypercapnia.

## Material and Methods

Two patients undergoing orthotopic liver transplantation (OLT) were monitored using venous transcranial color-coded duplex sonography (TCCD) to assess the flow in the vein of Rosenthal (VR), which serves as an indicator of global cerebral venous outflow. Insonation was performed through a transtemporal acoustic window, employing a low-flow setting at the diencephalic imaging plane (see Figure 1).
Patient 1 underwent transplantation using a veno-venous bypass technique, whereas Patient 2 was managed with the piggy-back technique.
In the postoperative period, clinical and instrumental signs of increased intracranial pressure (ICP), including optic nerve sheath diameter (ONSD) monitoring, were assessed.

## Results, Discussion and Conclusion

**In Case 1** (see Figure 2), cerebral venous flow (CVF) markedly decreased (mean velocity 22 → 11 cm/s) during mechanical ventilation after the placement of multiple central venous catheters, including the veno-venous bypass reinfusion cannula. This reduction preceded alterations in Mean Cerebral Artery flow and Optic Nerve Sheath Diameter (ONSD), suggesting early venous involvement in cerebral hemodynamic compromise. Venous flow assessment guided intraoperative management, including catheter removal, PEEP reduction, and $PaCO_2$ control to prevent ICP elevation. Despite these interventions, a transient postoperative ICP increase occurred (ONSD enlargement to 6.4 mm accompanied by nausea, without neurological deficits).
**In Case 2** (see Figure 3 - fewer central venous catheters), CVF remained stable and no subsequent postoperative ICP elevation was detected (Intraoperative and postoperative ONSD remained unchanged). The patient exhibited a lower absolute cerebral venous flow velocity than patient 1, likely due to the absence of a hyperdynamic circulation (OLT performed for neoplastic disease, MELD 10)

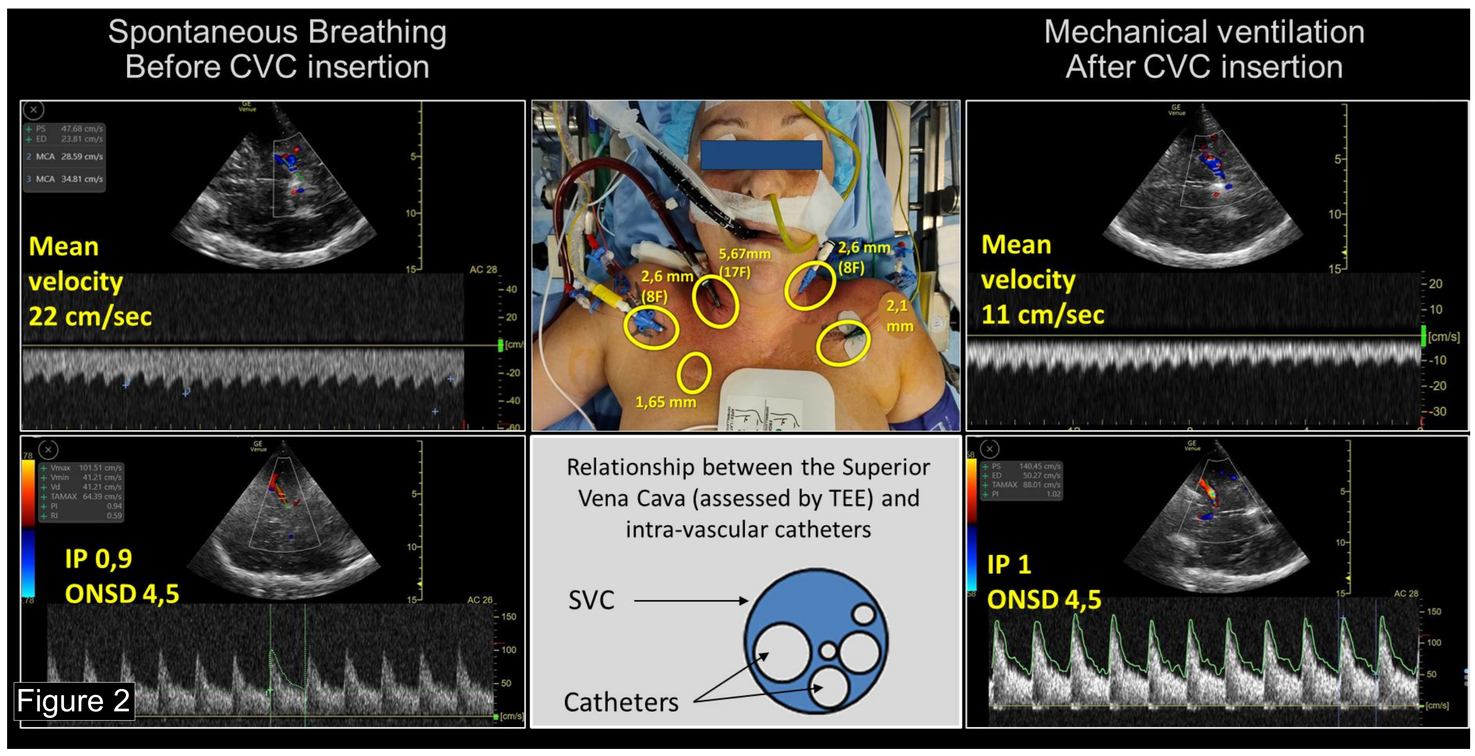

Figure 2

## INTRAOPERATIVE VENOUS FLOW MONITORING

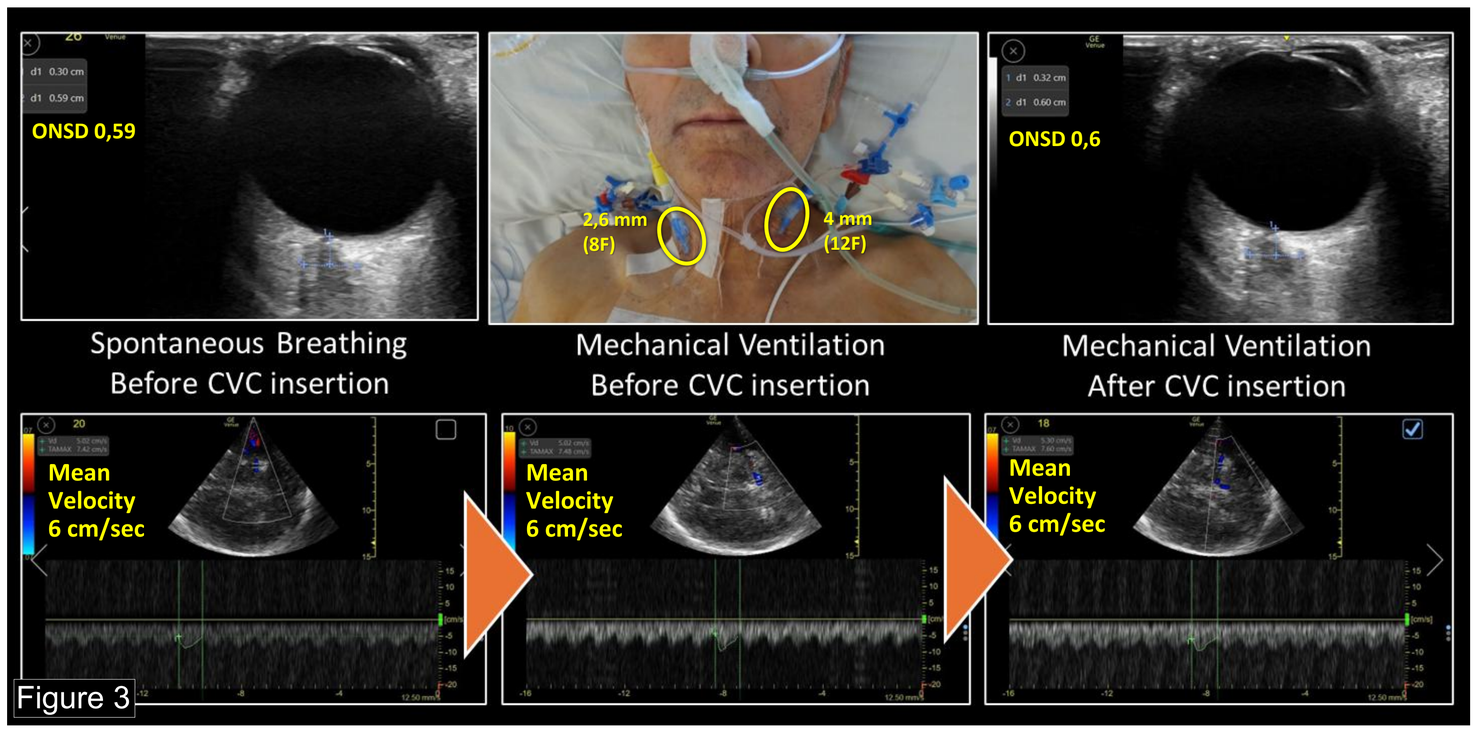

Figure 3

In transplant and major surgery, multiple factors may contribute to increased intracranial pressure by creating a mismatch between cerebral inflow and outflow. Figure 4 illustrates the factors contributing to impaired cerebral venous drainage, which can lead to intracranial hypertension. Intraoperative venous TCCD assessment can detect early venous congestion, potentially predicting ICP elevation before clinical or ultrasonographic signs, such as ONSD dilation or increased Middle Cerebral Artery Pulsatility Index, become apparent. In this context, intraoperative venous Doppler monitoring may provide an additional tool to guide tailored strategies aimed at preventing neurological complications. Further studies are recommended to evaluate changes in cerebral venous flow and intracranial pressure in specific clinical scenarios.

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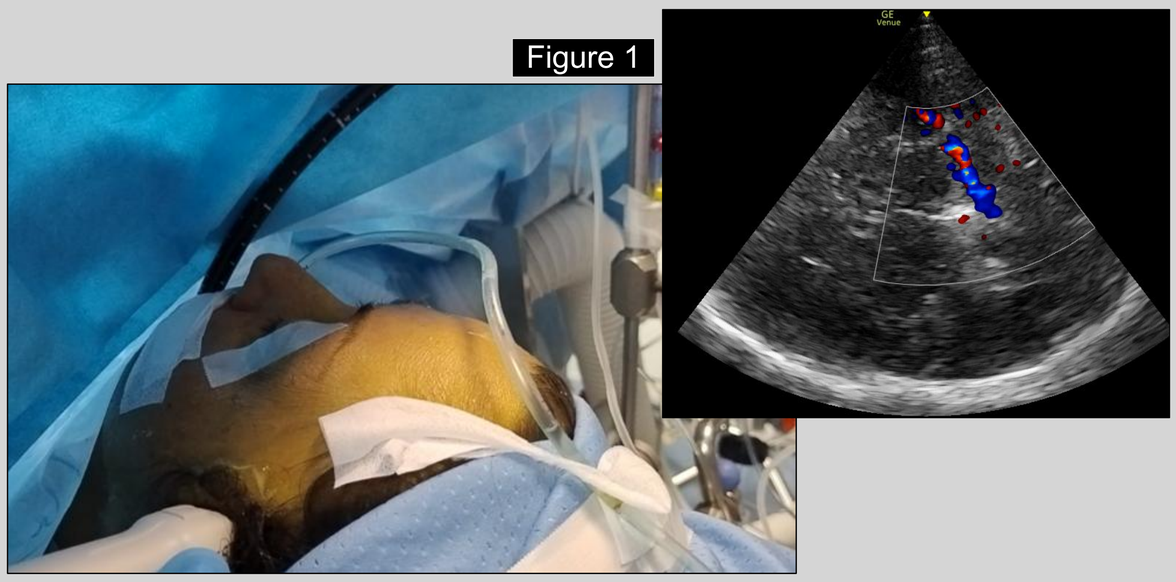
Figure 1



Wilson MH. Monro-Kellie 2.0: The dynamic vascular and venous pathophysiological components of intracranial pressure. J Cereb Blood Flow Metab. 2016 Aug;36(8):1338-50. doi: 10.1177/0271678X16648711

**Table I.** Suggested classification of venous causes of intracranial hypertension.

| | | Classification | Location of resistance/pressure | Clinical examples |
|---|---|---|---|---|
| I | a | Focal extramural venous sinus compression | External compression of a significant venous sinus at a focal point | Depressed skull fracture, periosteal hematoma, tumor. |
| | b | Focal intermural venous sinus stenosis | A focal narrowing within the sinus wall | Idiopathic intracranial hypertension |
| | c | Focal intramural venous sinus obstruction | Obstruction within a significant venous sinus | Sagittal or transverse sinus thrombosis. |
| | d | Diffuse venous compression | Throughout the venous tree | Any cause of cerebral swelling e.g. hypoxia, cerebral edema, contusions |
| II | | Extracranial venous hypertension – cervical | Within the neck | Cervical collars, hanging |
| III | | Extracranial venous hypertension – thoracic | Within the thorax | Any cause of increased intra-thoracic pressure – Chest infection, adult respiratory distress syndrome, mechanical ventilation |
| IV | | Extracranial venous hypertension – abdominal | Within the abdomen | Any cause of increased abdominal pressure - obesity, obstruction |
| V | | Orthostatic/gravity | | Visual impairment and raised ICP/space obstruction syndrome |

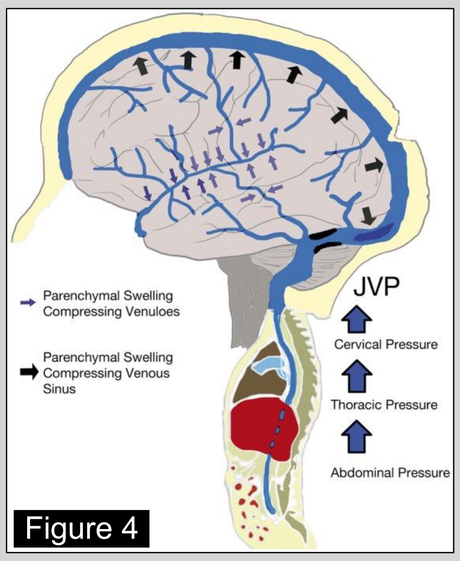


Figure 4