

Intraoperative Intracranial Pressure Instability in a Polytrauma Patient Undergoing Emergent Fasciotomy: Anesthetic Challenges

Juan P. Garcia-Mendez, M.D.¹, Alexander Kiel, M.D., Ph.D.¹, Benish Fatima M.B.B.S.¹

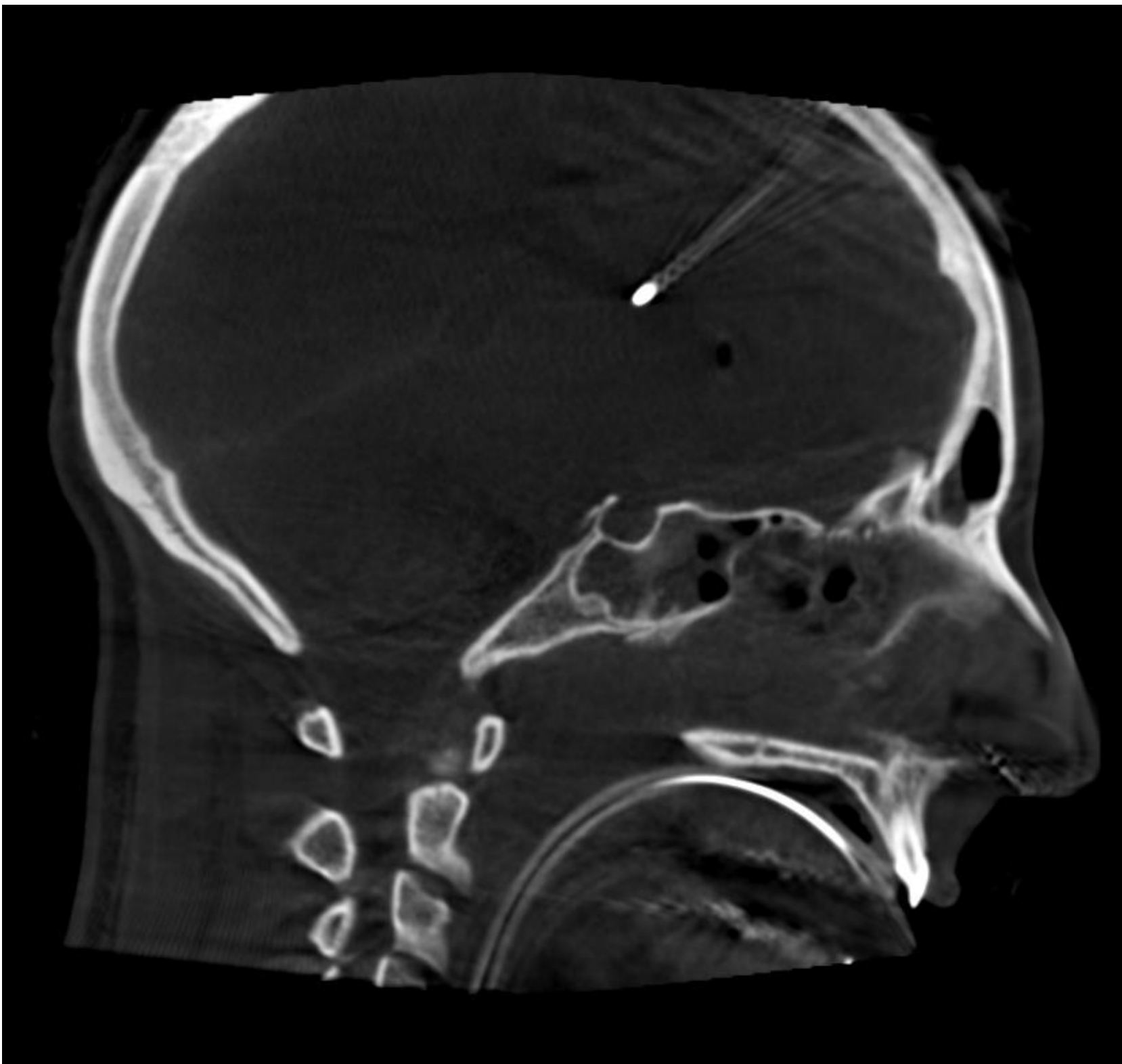
¹Department of Anesthesiology and Perioperative Medicine, Mayo Clinic, Rochester MN

INTRODUCTION

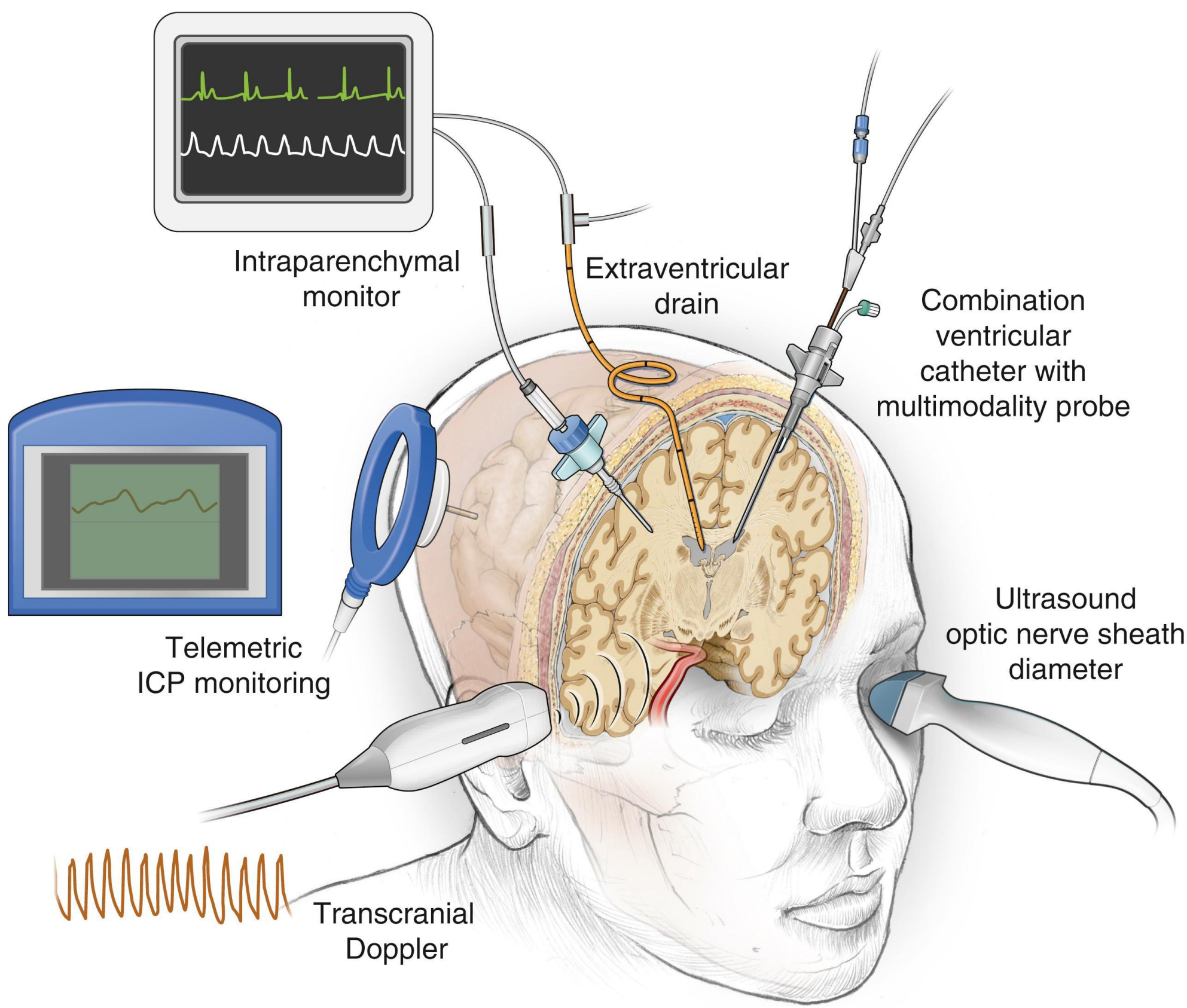
- Polytrauma patients with traumatic brain injury (TBI) who require emergent lower extremity fasciotomies are at risk of intraoperative intracranial pressure (ICP) instability.
- The choice of medications, ventilation strategy, or anesthetic technique greatly impact the ICP and hence neurologic and mortality outcomes.
- Learning objectives: to discuss the modalities and indications of ICP monitoring, as well as the treatment of intraoperative intracranial hypertension.

CASE REPORT

- 21-year-old female involved in a high-speed motor vehicle collision as an unhelmeted motorcycle passenger, initially combative and then unresponsive, requiring on field intubation.
- On arrival to the ED, she was hemodynamically unstable with a FAST exam positive for retroperitoneal bleeding, requiring massive transfusion protocol of 15 pRBCs.
- Initial evaluation identified skull, spine, lower extremity, and open-book pelvic fractures, as well as bilateral pneumothorax.
- Glasgow Coma Scale score was 6 and head CT showed left frontal SDH, severe cerebral edema with minimal ventricular space and right sphenoid fracture with pneumocephalus.
- Due to small ventricle size, placement of external ventricular drain (EVD) failed; hence a right frontal intraparenchymal ICP monitor was placed at bedside, with readings of around 10 mmHg.
- The patient was emergently taken to the ICU, where she was found to have signs of bilateral lower extremity compartment syndrome, and emergency surgery was planned for fasciotomies.
- **On arrival to the operating room, ICP precipitously rose to 40 mmHg.**
- Neuroprotective measures were immediately initiated, including reverse trendelenburg positioning, administration of 1g/kg of 20% mannitol, and switching volatile anesthetics to propofol and increasing fentanyl infusions, which allowed surgery to proceed safely. Any external stimulation was stopped to avoid any pain.
- **OUTCOMES:** Patient was transferred to the floor on POD 16 and discharged to a rehab facility on POD 44. Developed paroxysmal sympathetic hyperactivity (bouts of fever/tachycardia) requiring propranolol and clonidine but no other neuro deficits.



Intracranial pressure monitor entering the cranium; pneumocephalus evident anteriorly to monitor tip.



MODALITIES OF ICP MONITORING. Taken from <https://clinicalpub.com/intracranial-pressure-monitoring/>

TAKE HOME POINTS

CPP = MAP – ICP

- Normal CPP is **70-80 mmhg**
- Ischemia occurs at CPP of **30-40 mmhg**
- Avoid hypotension (SBP $\geq$ 110 mm Hg or MAP $>$ 80), hypoxia SpO₂ $\geq$ 94%, and normocarbida (35–45 mmHg)

ANESTHETIC CHOICE

- TIVA with RSI plus NMB
- Neuraxial techniques must be avoided in unstable ICP

INDICATIONS FOR ICP MONITORING

- (GCS $\leq$ 8) + CT head w/ structural damage

PREFERRED ICP MONITORING METHOD

- External ventricular drain (diagnostic & therapeutic) OR Intraparenchymal monitor when distorted anatomy or emergent

ICP INSTABILITY RESCUE MEASURES

- Hyperosmolar agents (mannitol, hypertonic saline)
- Deepening sedation/opioids + NMB
- Reverse Trendelenburg + stop stimuli

DISCUSSION

- Intracranial hypertension during non-neurosurgical interventions is known to occur **within the first two weeks after TBI.** (1)
- **“ICP monitoring is recommended in comatose patients with (GCS $\leq$ 8) when evidence of structural brain damage is seen on initial CT imaging.”** ACS Best Practices In The Management Of Traumatic Brain Injury 2024 Guidelines.
- In patients with radiologic signs of severe TBI, invasive ICP and arterial pressure monitoring should be established prior to surgery. (2)
- The **gold standard** for ICP measurement is an **EVD** as this has both diagnostic and therapeutic value. **An intraparenchymal transducer is an alternative** when rapid placement is needed, especially in the setting of severe cerebral edema or distorted ventricular anatomy.
- Releasing compartment pressures may lead to systemic vasodilation by releasing the accumulated metabolites like potassium or lactic acid, triggering a compensatory increase in ICP.
- Maintenance of anesthesia should be intravenous as inhalational agents increase ICP. Propofol is widely used for neuro-sedation and ICP reduction but may cause hypotension, so close monitoring is required.
- Normocapnia (PaCO₂ 35–45 mmHg) is therefore the target in TBI management, as both extremes—hypocarbida and hypercarbida—are associated with poor outcomes due to their harmful effects on cerebral physiology.
- **Rescue measures** for acutely increasing ICP include reverse Trendelenburg positioning, increasing opioids, avoiding pain stimulation, and hyperosmolar agents like mannitol. (3)
- Furthermore, interventions like neuromuscular blocking, deepening sedation, and starting propofol and fentanyl infusions help reduce cerebral metabolic demand and avoid straining.

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