Optimizing the Care of the Patient with Raised ICP

When patients have elevated intracranial pressure, the priority is to optimize care and prevent increases in ICP or decreases in CPP. Patient with non-compliant brains will demonstrate characteristic changes in their ICP waveform that warns us that even tiny changes in intracranial volume will usually cause ICP to rise. Careful monitoring of the ICP during all procedures is essential.

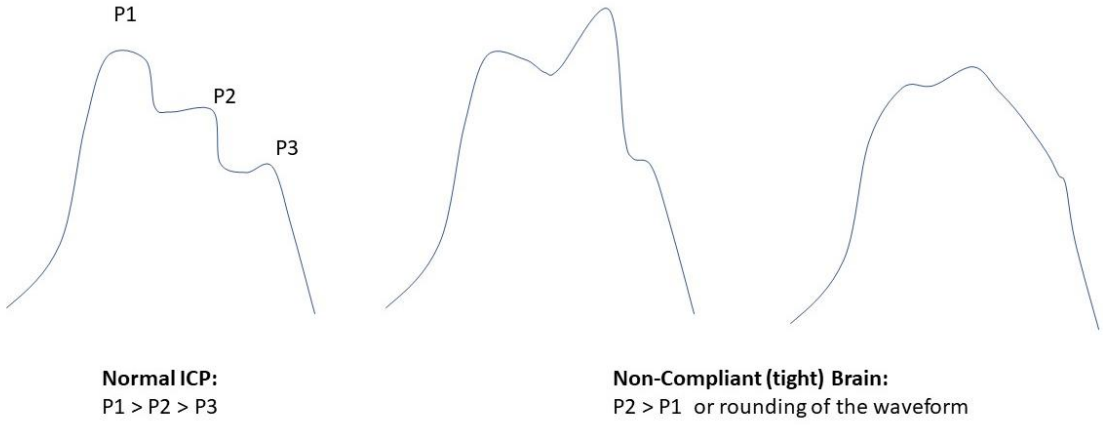

Figure 1 ICP Waveform showing normal and non-compliant brain

Normal ICP is < 10 mmHg. An ICP > 20 mmHg is the threshold for aggressive intervention. It is also usually associated with a non-compliant waveform that means small changes in volume will have a much more rapid and significant rise in ICP. The total time and the intensity of the ICP determines the ICP "dose". Episode above the ICP threshold as well as prolonged periods below the classical threshold may be important determinant for negative outcomes, particularly if CPP is not maintained.

Treatment of raised ICP is aimed at lowering the volume components in the cranial compartment:

1. **Reduce Tissue Volume**

    a. Prevent secondary injury and edema by the prevention of:
        i. Hypoxemia
        ii. Hypercarbia
        iii. Hypotension
        iv. Hypoglycemia
        v. Hyperglycemia
        vi. Fever
        vii. Seizures

    b. Administer administration of osmotic diuretics if indicated

    c. Surgery to evacuate blood and/or decompress the brain (the potential benefit of survival needs to be balanced against the increased risk for survival with poor neurological outcome)

2. **Reduce Blood Volume**

   a. Prevent secondary injury from reflex increase in cerebral blood flow by preventing:
      i. Hypoxemia
      ii. Hypercarbia
      iii. Hypotension
      iv. Hypoglycemia
      v. Fever
      vi. Increased sympathetic output (pain, agitation or seizure)

   b. Promote jugular venous drainage:
      i. Increase head of bed 30-45 degrees/reverse Trendelenburg (ICP can be used to guide optimal position)
      ii. Ensure C-Spine collars and trach ties are not impeding jugular veins

   c. Prevent elevation in intrathoracic pressure
      i. Prevent gag and cough reflex
      ii. Maintain ventilator asynchrony
      iii. Careful titration of PEEP to maintain adequate SpO2 without increasing ICP (use ICP to guide optimal PEEP setting)

   d. Prevent elevation in abdominal pressures
      i. Decompress stomach (NG)
      ii. Implement bowel routine (prevent abdominal distention)
      iii. Monitoring for signs of abdominal compartment syndrome
      iv. Use reverse Trendelenburg for obese/pregnant patient

   e. Ventilate to PCO2 target (not pH) to keep PCO2 35-40 and prevent hypercarbia/hypoxemia during suctioning.

      In severe intracranial hypertension, orders may be given to lower PCO2 targets to 30 or 32 to 35. While hyperventilation does dramatically lower ICP, prolonged hyperventilation < 25 mmHg is not recommended as it may cause more cerebral ischemia. Hyperventilation < 32 should be avoided in the first 24 hours post injury. On occasion if a patient is on route to the CT scanner or OR with an acute decompensation, neurosurgery may want the patient hyperventilated to buy time.

3. **Reduce CSF Volume**

   We make approximately 20 ml of CSF more every hour than we have space within our ventricles. Consequently, to prevent hydrocephalus, CSF must be able to flow unimpeded from the ventricles into the subarachnoid spaces. From the subarachnoid space, CSF is reabsorbed into the large draining veins (sagittal sinus). Drainage of the jugular veins promotes both the drainage of blood and CSF from the cranial vault.

Hydrocephalus can develop if pressure in the brain obstructs the outflow of CSF, if blood within the ventricles or subarachnoid space interferes with the reabsorption of CSF into the venous blood, or if there is an obstruction of jugular venous drainage. To reduce CSF volume:

a. Continue all of the interventions in 2a-e; any reduction in jugular venous drainage will decrease CSF drainage

b. Drain CSF if an external ventricular drain is in place. Ensure that the level of the drain is maintained at the appropriate level at all times.

The drain may be ordered off with intermittent drainage orders. This may be done if the ICP is normal upon insertion to allow the team to observe whether intracranial hypertension is a concern. When the patient has elevated ICP, the drain will usually be left in the open position with the level of the drainage chamber positioned to ensure an "automatic pressure relief". Drainage will occur if the pressure exceeds the level of the drain and will stop as soon as the pressure falls again (as long as there is CSF available to drain and the drainage catheter is patent). This prevents swings in the pressure that can cause ischemia or bleeding.

During transport, keep drainage unit at the correct level with drain open. Patient transport should be anticipated to increase the ICP further, at a time when ICP monitoring may not be available (disconnected from Codman™). Keeping the drain open and as close to the optimal level provides pressure relief.

The drain should only be clamped momentarily during times when the drain must be lowered for patient positioning (to prevent over drainage). A drain that is too high will be similar to clamping. As soon as patient is repositioned, reopen the drain to provide pressure relief. Do not lay the drainage collection unit horizontally.

4. **Reduce the Metabolic Rate**

a. For patients with unstable intracranial hypertension, deep sedation is usually initiated to provide "brain rest". Like sedation for severe ARDS, the sedation should not be stopped for neurological assessment.

b. If ICP remains problematic despite deep sedation with narcotics and sedatives, neuromuscular blockade.

c. Maintain normothermia/prevent fever. Initiate acetaminophen around the clock. Consider NSAID for persistent fever if normal renal function. Rule out and treat infection.

Although evidence for using hypothermia is not associated with improved outcomes, mild hypothermia (35-36C) may be considered on an individual patient basis.

d. Space out nursing care. Minimize nursing care and break it up in to brief episodes of care versus "getting it all done followed by a rest".

e. Turn off lighting and minimize stimulation.

f. Prevent and treat sepsis and ARDS and complications of critical illness.

g. Barbiturates can be considered to reduce cerebral metabolic rate.

5. **Suctioning**

Suctioning is one of the worse interventions for patients with raised ICP, however, ***it is a necessary evil***. Pneumonia and secretion retention also increases the risk for pneumosepsis, hypoxemia and hypercarbia and/or need for higher levels of PEEP.

Suctioning is associated with elevations in ICP among patients with raised intracranial pressure. Patients with non-compliant waveforms are at the greatest risk for significant and/or persistent elevation after suctioning. There are at least 4 potential mechanisms that may cause ICP elevation with suctioning including: hypoxemia, hypercarbia, gagging/coughing or Valsalva response and/or sympathetic surge (with increased cerebral oxygen demand). To prevent or limit the duration of suctioning induced ICP elevation, the following interventions are implemented:

a. Administer preemptive sedative or narcotic to blunt the gag and cough reflex and reduce sympathetic surges

Observe for gagging and coughing with suctioning and modify sedation management as required. If the ICP elevation persists despite deep level of sedation, neuromuscular blockade may be added (either PRN before suctioning as a continuous infusion). Neuromuscular blockade provides the highest level of gag/cough suppression and may provide additional benefit through reduction in metabolic rate.

b. Prevent suctioning induced hypoxemia or hypercarbia

Preoxygenate and give a few manual breaths before (and after if ICP spikes with suctioning). The patient's baseline frequency will influence the number of additional breaths that can be given. Use changes in the ICP to determine whether additional recovery breaths or rest periods between attempts are needed. Even minor increases in PCO2 above a level of 40 mmHg can cause elevations in ICP.

ETCO2 trends may be used to identify correlations between ICP and CO2, however, ETCO2 rarely equals PaCO2. Record the ETCO2 at the time of arterial blood gas sampling to help identify individual patient correlation.

c. Limit Stimulation

Limit suctioning passes and use the ICP to guide interventions. One or two passes followed by a period to return to baseline is preferable over multiple passes with sustained ICP.

d. Use ICP to Guide Intracranial Impact

If any intervention causes the ICP to rise, return to the previously tolerated strategy and consult the CCTC resident (senior if junior is uncertain). Changes in minute volume or in the mode of ventilation may impact the ICP.

6. **Maintain Cerebral Perfusion Pressure**

When ICP increases and autoregulation is normal, cerebral perfusion pressure is maintained by when BP is increased (which reduces ICP).

When monitoring blood pressure and CPP, check the level of the arterial line transducer frequently. There is uncertainty in the literature on the optimal position of the arterial line transducer. While a minimum MAP of 60 mmHg is advocated, some centres base this on a transducer that is leveled to the cerebral arteries (to the tragus), whereas, others base this on a transducer leveled to the phlebostatic axis. The position used in most research studies is unknown. This difference in transducer position translates to a difference in CPP of ~15 mmHg based on transducer position. Currently, we use the phlebostatic axis to level our arterial transducers and calculate CPP; it is essential that the transducer is not positioned below the phlebostatic axis (which would produce a falsely elevated arterial pressure and overestimation of the CPP).

Maintaining a CPP (MAP - ICP) 50-70 mmHg may reduce the risk of cerebral ischemia. While this is an important initial step in the management of raised ICP, the optimal cerebral perfusion pressure is unknown. Initial target is usually 65 mmHg.

If autoregulation is preserved, ICP should decrease when MAP is increased. Increasing the CPP above 65-70 is not always associated with improved cerebral perfusion, particular if a patient has lost the ability to autoregulate. A blood pressure that is too high can increase the cerebral blood volume, cause cerebral edema and elevate the ICP.

Hypotension should always be avoided, however, too high a MAP may also cause harm. One strategy to see if a patient might benefit from a higher MAP is to monitor the patient's ICP closely after increasing the MAP by 10 mmHg. Avoid any other intervention that might impact the ICP during this "test period". If the ICP falls with an increase in MAP, the higher MAP may be beneficial.

Determination of optimal CPP may be best done using multimodal monitoring strategies not currently available in CCTC (e.g., measurement of brain tissue oxygen, lactate or glucose or continuous transcranial doppler/cerebral blood flow).

Bibliography

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