A.3. Intracranial hypertension

a. Clinical Manifestation of Acute and Chronic Intracranial Hypertension

i. Cerebrospinal Fluid (CSF) Physiology

CSF is produced by choroid plexus in the lateral, third, and fourth ventricle through an ultrafiltration process. Normally CSF is produced at the rate of 0.3cc/minute (or 400-500 cc a day). CSF circulates through the ventricular system, then down to the spinal subarachnoid space, before it returns to the intracranial cavity. CSF is generally absorbed by the arachnoid granulation over the cerebral convexity and returned to the vascular system. Some minor absorption occurs near the cranial nerve root sheaths. Under extremely high pressure, CSF may be resorbed over the ventricular ependyma in proportion to the pressure gradient. The total volume of CSF in an adult is about 150cc, meaning that the CSF volume is turned over more than 3 times a day. Only about 40cc of the total body CSF volume is found in the ventricular system.

Definition: Normal intracranial pressure (ICP) is generally considered to be less than 20mm Hg. The Monro-Kellie doctrine states that the total intracranial volume, consisting of brain, blood, cerebrospinal fluid (CSF), and other pathological entity, remains fixed. The cerebral perfusion pressure (CPP) is defined as Mean Arterial Pressure (MAP) minus the Intracranial pressure (ICP). [CPP=MAP-ICP] In management of increased intracranial pressure, an effort is made to maintain the ICP below 20 mmHg and the CPP above 70 mmHg.

ii. Etiology: Elevated intracranial pressure is the final common pathway of a variety of intracranial pathology.

- The most common cause of elevated ICP is traumatic brain injury. Trauma can lead to hematoma (epidural, subdural, and intraparenchymal) formation with resultant mass effect on the involved hemisphere or lobe. Other parenchymal lesions such as cerebral contusion can also exert significant pressure on the surrounding brain tissue. Edema and hyperemia (probably due to loss of cerebral autoregulation) can cause further increase ICP, leading to secondary injury after the initial insult.
- Both primary and metastatic neoplasm of the brain can cause breakdown of blood brain barrier, edema, mass effect, and increased ICP. Generally, benign neoplasms do not cause ICP elevation unless their size becomes substantial, or if they block CSF flow. Malignant neoplasms, because of the breakdown of blood brain barrier, readily cause edema, mass effect, and ICP elevation. Obstruction of the ventricular system by a mass lesion can lead to obstructive hydrocephalus and intracranial hypertension.
- Hydrocephalus can occur in two forms, communicating or obstructive. Communicating hydrocephalus generally occurs as a result of prior infection, hemorrhage, or arachnoid irritation. Normal pressure hydrocephalus (NPH) in the elderly population may occur without the above predisposing factors. Rarely, a cerebrospinal fluid (CSF) producing neoplasm such a choroid plexus papilloma can cause CSF overproduction and result in communicating hydrocephalus. Obstructive hydrocephalus is the result of CSF obstruction at strategic points of the CSF pathway. Common causes include intraventricular or periventricular tumors,

brain herniation causing trapping of the lateral ventricle, intraventricular hemorrhage, or aqueductal stenosis. A combination of these two types of hydrocephalus can occur.

- Venous sinus thrombosis can occur as the result of systemic hypercoagulable states or trauma. Risk factors include the use of oral contraceptive and certain connective tissue disorders, including lupus erythematosis. CSF absorption depends on flow across the arachnoid granulations. Because of the reduced venous drainage, increased intracranial blood volume leads to decreased CSF resorption, resulting in intracranial hypertension.
- Idiopathic intracranial hypertension is a disease most commonly found in adult female patients. Obesity is a predisposing factor. Headache and progressive loss of visual acuity are the primary symptoms. Chronic elevation of ICP causes papilloedema, which could be detected on fundoscopic examination.
- Systemic hypertension can cause breakdown of the blood brain barrier and causes hypertensive encephalopathy. This can increase ICP because of higher MAP and cerebral blood volume.

b. Clinical Presentation of Intracranial Hypertension

Acute intracranial hypertension most frequently presents with a constellation of symptoms including headache, altered mental status (level of consciousness), nausea/vomiting, and occasionally sudden death. In addition to the above symptoms, chronic intracranial hypertension can present with cranial nerve palsies (third or sixth cranial nerve), ataxia, memory disturbance, personality changes, or urinary incontinence. Both acute and chronic intracranial hypertension can cause seizures.

c. Emergency Management of Intracranial Hypertension

- Head position: Simple head elevation can promote venous return from the head and reduce ICP. It should be noted that head elevated does lower the mean arterial pressure supplying the brain, possibly negating the beneficial effects of increased venous drainage.
- Hyperventilation: Though very effective in reducing ICP through its cerebral vasoconstrictive effect, hyperventilation causes reduced cerebral blood flow (CBF). This can lead to secondary hypoxic injury. Only mild to moderate degree of hyperventilation is recommended. (pCO_2 > 30mmHg) The patient need to be mechanically ventilated to have desired controlled ventilatory effects. The effect of hyperventilation is generally transient. (48-72 hours)
- Diuretics/Hyperosmolar agents: Mannitol can be used to draw water out of the brain tissue by osmotic forces. This reduces brain tissue volume and ICP. Other agents that can be used include furosemide and urea. These agents should generally be "weaned off" to prevent rebound cerebral edema.
- Sedation and paralytic agents. Agitation and muscle tremors/spasms can artificially elevate ICP. Benzodiazepines, narcotics, and if necessary, chemical paralytic agents should be employed.
- CSF drainage with an external ventricular drain, or much more rarely with a CSF shunt, can lower the ICP.

- Barbiturate coma can be induced with pentobarbital to reduce cerebral blood flow (CBF) and cerebral metabolic requirement of O2 (CMRO2). Close arterial pressure monitoring is mandatory because of its cardio-depressive effect. Most clinicians advocate continuous electroencephalographic (EEG) monitoring to assess the end point of burst suppression of EEG.
- Avoid hypotension in order to maintain CPP.
- Evacuate the causative intracranial mass lesion, including hematoma or neoplasm. A depressed skull fracture can also cause elevated ICP. The underlying injury may not be reversible.