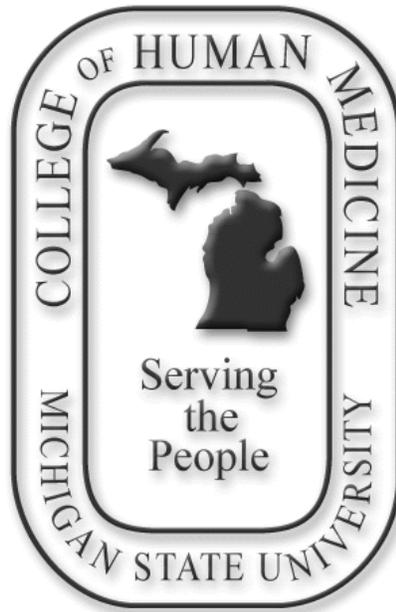# INCREASED INTRACRANIAL PRESSURE MODULE

Objectives:

1. Discuss the features of intracranial space-occupying expanding mass lesions within the rigid cranium and list compensatory mechanisms for space-occupying lesions.

2. List effects of increased intracranial pressure produced by supratentorial space-occupying lesions.

3. List the consequences of uncal herniation.

4. Describe the effects of downward displacement of the brainstem (central herniation).

5. Describe the pathogenesis and consequences of cerebellar tonsil herniation.

6. List the types of cerebral edema and discuss their pathogenesis and consequences.

7. For non-communicating and communicating hydrocephalus, define and list examples of causes and sites of obstruction to cerebrospinal fluid flow.

IA. Mass lesions - introductory concepts

    A. Intracranial Space-Occupying Expanding Mass Lesions

1. General Considerations

Since the brain is enclosed in a rigid cranium, the free space to expand is minimal. When the mass of brain intracranial contents increases in the presence of disease, intracranial pressure (ICP) increases. When ICP exceeds a critical point, displacements and herniations occur. The nature of herniation is determined by the location of the lesion.

Intracranial pressure is usually estimated by measuring CSF pressure through lumbar puncture. The normal value is < 200 mm water. CSF pressure depends on cerebral blood volume (varies with systole/diastole and respiration), volume of brain tissue and volume of CSF.

2. Compensatory Mechanisms

- Reduction in cerebrospinal fluid (CSF) volume
- Reduction in intracerebral blood volume
- Loss of brain tissue, e.g. necrosis and atrophy

3. Factors Governing Increased ICP

- Size of lesion and size of remaining space, e.g. more space may be available due to atrophy in elderly brain, or in brain with previous infarct.
- Severity of edema
- Vasoregulatory mechanisms: Autoregulation involves the maintenance of constant cerebral blood flow (CBF) over wide range of perfusion pressures. (i) When systemic blood pressure decreases, brain vessels normally dilate; with more decrease, CBF decreases and Cushing's phenomemon occurs (systemic hypertension and bradycardia.) (ii) When systemic blood pressure increases, brain vessels normally constrict. (iii) With loss of autoregulation, vasoparalysis and edema occur.
- Patency of CSF pathway; e.g. block of CSF leads to hydrocephalus
- Speed of expansion: slowly expanding lesions are more readily accommodated
- Age of patient; e.g. in infants, skull can increase in size and accommodate expansion; in elderly, more space may be available due to atrophy

4. Clinical Features of Increased ICP: Headache, nausea, vomiting, decreased level of consciousness, papilledema

5. Types of Disorders Commonly Associated with Mass Lesions: hemorrhage, neoplasms, infections

IB.Effects of space-occupying lesions

      B. Effects of Supratentorial Space-Occupying Lesions (SOL)

Displacement effects of rapidly expanding SOL include:

- Cingulate gyrus herniation under the falx cerebri (subfalcine herniation)
- Midline shift (lateral displacement)
- Uncal (transtentorial, uncinate, mesial temporal) herniation - herniation of the medial temporal lobe (uncus) across the tentorium cerebelli
- Cerebellar tonsil (tonsillar) herniation through the foramen magnum
- Downward displacement of the diencephalon and brainstem
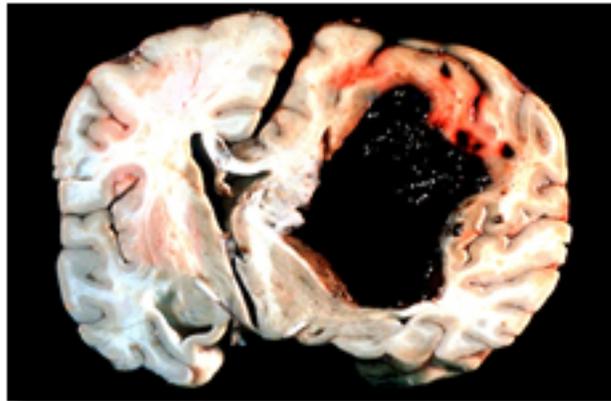
ICP 01

This coronal section shows cingulate gyrus herniation and midline shift. Uncal herniation and downward displacement of the brainstem also occurred in this patient.

1. Consequences of cerebellar tonsil herniation
Displacement of the cerebellar tonsils through the foramen magnum compresses the medullary respiratory centers, leading to death.

2. Consequences of lateral displacement of brain
Changing levels of consciousness have been correlated with lateral displacement of diencephalon (containing parts of the ascending reticular activating system.)

3. Consequences of Uncal (Temporal Lobe) Herniation
The features below are based mainly on ipsilateral compression; in some cases, however, one uncus may push the midbrain against the contralateral tentorium, causing compression of the contralateral cerebral peduncle, with hemiparesis ipsilateral to the side of the herniation. The changes in the peduncle in this setting are referred to as Kernohan's notch.

ICP 02
Uncal herniation occurred in this case. The brainstem has been removed to show the midbrain. Compression of the cerebral peduncles can be seenin the lower half of the image, along with petechial hemorrhages in the midbrain. (The dark areas adjacent to the cerebral peduncles represent the substantia nigra.)

a. Compression of **cranial nerve III**. The ipsilateral third nerve, as it passes between the posterior cerebral and superior cerebellar arteries, is initially flattened and may show hemorrhage.   The first clinical sign is ipsilateral pupil dilation, since the parasympathetic fibers are located on the outside of the nerve and are inactivated first by compression. Complete third nerve paralysis may also occur. As the herniation progresses, the contralateral oculomotor nerve may be compressed, producing bilateral pupil dilation.

   b. Compression of **midbrain cerebral peduncles**. Most often the ipsilateral cerebral peduncle is compressed, resulting in contralateral hemiparesis or hemiplegia. In addition the cerebral peduncle on the side opposite the space-occupying lesion may be compressed against, or indented by, the free edge of the tentorium cerebelli. This results in ipsilateral hemiparesis or hemiplegia (if it occurs alone) or quadriplegia (if both peduncles are compressed)

   c. Compression of the **posterior cerebral artery**. Obstruction of the posterior cerebral artery or its branches, due to compression of the artery against the free edge of the tentorium, produces hemorrhagic infarction on the medial and inferior aspects of the ipsilateral occipital lobe. Hemorrhagic infarction occurs because the compression may be reduced after damage to arterial walls and blood then enters the infarcted area. The lesion is often confined to the distribution of the calcarine branches of one posterior cerebral artery, leading to homonymous hemianopsia. If the occipital lobe lesions are bilateral, cortical blindness is a clinical result (the patient does not comprehend visual images, but pupillary reflexes are intact).

 d. Brainstem compression. The patient becomes comatose and may develop cardiac and respiratory changes secondary to increasing brainstem compression. Brainstem

compression and dysfunction will begin in the midbrain and will gradually progress caudally. Two types of events should be understood.

- Secondary brainstem hemorrhages (Duret hemorrhages) may occur, probably because of compression and stretching of blood vessels, especially veins. Death may ensue directly from the midbrain and pons destruction.
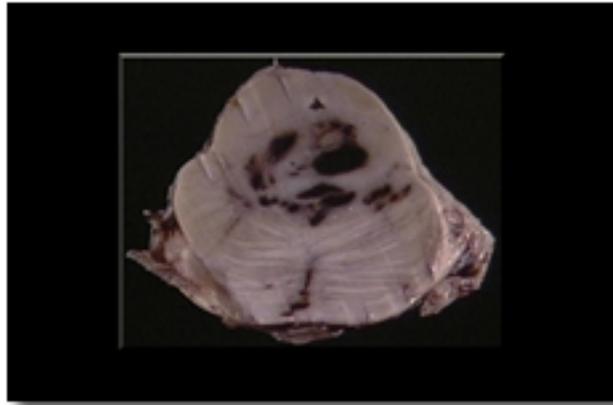
ICP 03

In this section showing components of both the midbrain (note aqueduct at top) and pons (note crossing fibers at bottom), Duret hemorrhages are present as a result of increased intracranial pressure.

- Compression will be transmitted downward from the midbrain, causing dysfunction with respiratory, postural, and oculomotor changes (see section below). Finally, as damage in the medulla causes slow irregular respirations, an irregular pulse and falling blood pressure, death may occur due to respiratory arre

4. Consequences of Central Herniation (Rostrocaudal deterioration)

a. General comments. Rostrocaudal deterioration (or rostrocaudal decompensation) is the progressive decline in neurological status due to lesions progressively more caudal as a result of a supratentorial space-occupying mass and downward displacement of the brainstem. Lesions lying medially or in the frontal pole may not compress the diencephalon and midbrain laterally, but rather result in rostrocaudal dysfunction of the brainstem with bilateral progression of impairment. As in uncal herniation, secondary or Duret hemorrhages may occur in the midbrain and pons as the brainstem is displaced.

b. Clinical Signs.

Changes in consciousness begin with decreasing alertness, progressing to drowsiness, stupor and coma.

Respiratory changes which occur with lesions in various sites:

Site of lesion	Respiratory pattern
Diencephalon	Cheyne-Stokes respiration
Midbrain	Central neurogenic hyperventilation
Pons	Apneustic respiration
Medulla	Ataxic respiration

Changes in postural reflexes in response to a noxious stimulus: Decorticate rigidity, characterized by leg extension and arm flexion, results from widespread lesions in the cerebral cortex. Decerebrate rigidity, characterized by extension of both arms and legs, follows lesions disconnecting the cerebral hemispheres from the brainstem, e.g. lesions in the upper midbrain commonly produce decerebrate rigidity.

Pupillary changes in a comatose patient can be used to evaluate the general location of lesions.

Small reactive: Compression of the diencephalon impairs sympathetic fibers which originate there; impairment of sympathetic mediation of pupil dilation leads to small pupils.
Dilated, fixed: Compression of one oculomotor nerve (III nerve) by the uncus impairs the parasympathetic fibers travelling along the periphery of the III nerve; inactivation of these parasympathetic fibers leads to dilation of the ipsilateral pupil and loss of the light reflex in that pupil.
Midposition, fixed : Compression of both III nerves or compression of the midbrain results in bilateral impairment of both parasympathetic and sympathetic fibers travelling to the pupil; thus the pupils are midposition (medium size) and fixed (no light reflex in either pupil).

Ocular movements

Pathways for ocular reflexes are localized in the brainstem, making them useful for testing pathways in comatose patients. The pathway involves stimulation of the abducens (CN6) and the contralateral oculomotor (CN3) nuclei, connected by the medial longitudinal fasciculus (MLF) to produce conjugate deviation of the eyes. Usually the oculomotor responses to caloric stimulation (water put into the ear; oculovestibular reflex) and to passive head turning (oculocephalic reflex or "doll's eye movements") are similar. The oculocephalic reflex ("doll's eye movements") can only be tested in a comatose patient (a conscious patient's pupils will stay looking straight ahead in front of the face when the head is turned). In a comatose patient, when the oculocephalic reflex is PRESENT, the patient's brainstem is INTACT. To examine the oculocephalic reflex, hold the eyelids open and rotate the patient's head from side to side. If the MLF is intact, the result will be contraversive (opposite to the direction the head is rotated), conjugate (both eyes move

the same direction) eye deviation. E.g. if the head is rotated to the right, the eyes deviate to the left in a comatose patient with an intact MLF (this is what happens in a doll with movable eyes.)   If there is a lesion affecting the MLF in the brainstem, the eyes will not deviate conjugately when the head is turned.

Remember that if a comatose patient has uncal herniation compressing CN3, ocular movements on one side may be impaired, so that the affected eye cannot deviate medially, even though the MLF and the CN3 nucleus are intact. E.g. if there is a tumor in the left hemisphere, causing left uncal herniation and compression of the left CN3, there will be a fixed and dilated left pupil. In that case, if the head is turned to the left, the right eye will deviate laterally but the left eye will not move. If the head is turned to the right, both eyes will look to the left, showing contraversive, conjugate eye movements, and indicating that the MLF is intact.

II. CEREBRAL EDEMA

Edema may occur in a number of diseases, and may lead to increased intracranial pressure. Following an infarct, edema peaks between 2 and 4 days after the ischemic event and may exert a mass effect; intravenous mannitol may be helpful in therapy.

A. Vasogenic Cerebral Edema (most common form of edema)

Increased permeability of small vessels (breakdown of blood-brain barrier)

Escape of proteins, fluids into extracellular space, especially of white matter. Because the fluid can flow along fiber tracts, the swelling may be greater in white matter than in gray matter.

B. Cytotoxic Cerebral Edema (cellular brain edema)

Increased permeability of cell membranes secondary to cellular injury

Intracellular accumulation of excess fluid; may occur with ischemia or with other conditions such as metabolic poisons. Because neurons are most vulnerable to cell injury, cytotoxic edema may be more severe in gray matter than white matter.

C. Hydrocephalic (Interstitial) Edema

Fluid flows from CSF into brain through ventricular lining in cases of hydrocephalus.

III. CEREBROSPINAL FLUID (CSF) FLOW AND HYDROCEPHALUS

Hydrocephalus is defined as the accumulation of excessive CSF within the ventricular system of the brain. The ventricular system dilates when CSF flow is obstructed.
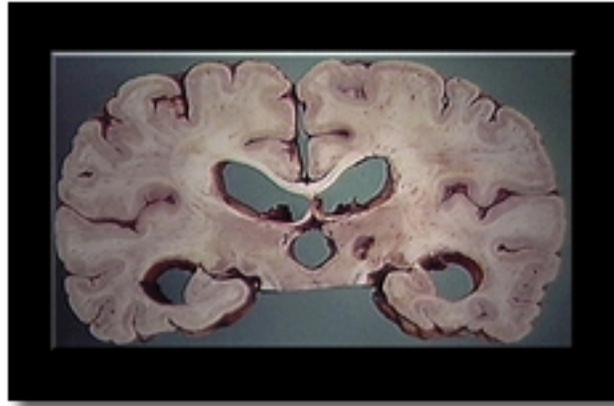
ICP 04
Hydrocephalus of lateral ventricles and third ventricle can be seen in this coronal section.

Therapy, regardless of etiology and pathogenesis, involves shunting the CSF and re-establishing its flow. If hydrocephalus occurs before closure of the cranial sutures, there is enlargement of the head, with an increase in head circumference. After fusion of the sutures, hydrocephalus is associated with increased intracranial pressure.

CSF Circulation: CSF is produced by the choroid plexus within the lateral and fourth ventricles. Normally the pathway of flow is: lateral ventricles, foramen of Monro, third ventricle, aqueduct of Sylvius, fourth ventricle, foramina of Magendie and Luschka; subarachnoid space over brain and spinal cord; reabsorption into venous sinus blood via arachnoid granulations.

A. Non-Communicating Hydrocephalus
Obstruction to CSF flow within ventricular system or at outlet foramina. Sites of narrowing are commonly obstructed, e.g. aqueductal stenosis.

B. Communicating Hydrocephalus
Obstruction to CSF flow in the subarachnoid space after exit from fourth ventricle. (The lateral ventricles still 'communicate' with the spinal cord subarachnoid space'.) Causes include leptomeningitis (fibrosis seals subarachnoid space and obstructs CSF flow) and subarachnoid hemorrhage.