Brain Herniation

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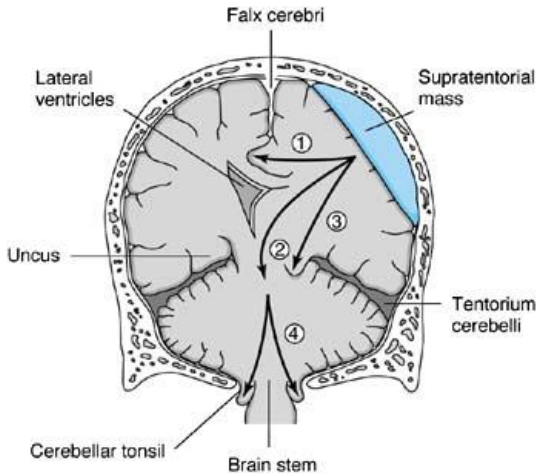
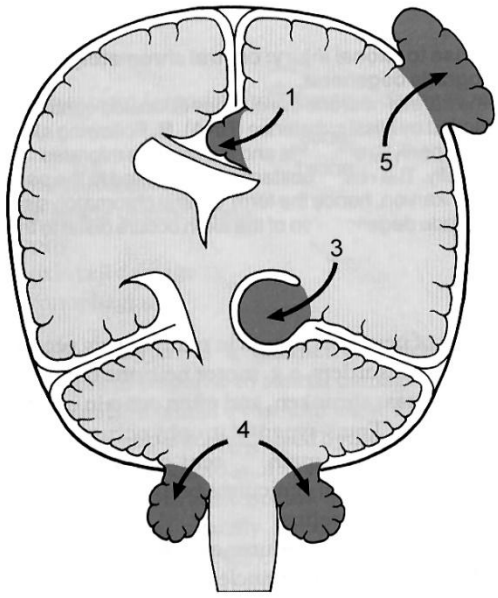
PATHOPHYSIOLOGY

- cranial cavity is *divided into compartments* by **semirigid, densely fibrous folds of dura mater**:
  1) falx (cerebri)
  2) tentorium (cerebelli).
- herniation occurs when brain is subjected to PRESSURE GRADIENTS that cause portions of it to flow from one compartment to another.
  - pressure gradients are created by **mass lesions** (often surrounded by extensive **edema**).
  - injudicious use of hyposmolar 5% dextrose IV often is sufficient to produce abrupt increase in brain edema and herniation.
- arteries and veins are relatively fixed in space - moving brain portions ***lose blood supply***.
  - in lateral herniation, hernia itself may compress or distort vessels.
- herniation produces ***dysfunction in white matter pathways*** (via geometrical distortion of pathways – distension or compression); extracerebral structures (esp. CN3) are also susceptible to distortion.
- brain tissue is ***injured along free edges of dural reflections*** across which it is squeezed.





(1) CINGULATE (SUBFALCINE) HERNIATION under falx cerebri.
(2) CENTRAL (DOWNWARD TRANSTENTORIAL) HERNIATION through tentorial notch.
(3) UNCAL (LATERAL MASS) HERNIATION over edge of tentorium.

(4) CEREBELLAR TONSILLAR HERNIATION through foramen magnum.
(5) TRANSCALVARIAL HERNIATION - swollen brain herniates through any defect in dura & skull.

- (2) – (4) types may cause death through ***brainstem compression***.
- **Cushing's triad** is response to diminished brain stem perfusion, regardless of etiology.

TYPES OF HERNIATION

All types may have **nonspecific symptoms of increased ICP** (headache, vomiting, hypertension, bradycardia, papilledema, CN6 palsy, transient visual obscurations, alterations in consciousness).

N.B. syndromes can develop over hours or minutes!

SUPRATENTORIAL MASSES

CENTRAL (S. DOWNWARD TRANSTENTORIAL) HERNIATION

- DIENCEPHALON symmetrically moves caudally through ***tentorial notch*** → whole brainstem is compressed toward foramen magnum → stretch on paramedian branches of basilar artery.

ETIOLOGY - diffuse brain swelling, hydrocephalus (esp. obstructive), centrally located supratentorial mass (or multiple bilateral masses).

CLINICAL FEATURES - ROSTROCAUDAL neurologic deterioration:

1. **RF dysfunction** - **progressive alteration of consciousness** - *earliest warning*!
2. **Diencephalic stage** (still reversible!):
    1) **pinpoint reactive pupils** (loss of sympathetic output from hypothalamus), roving eye movements.
    2) many individuals have hemiparesis before herniation; as diencephalic stage evolves, contralateral hemiplegia worsens with homolateral limbs developing paratonic resistance to movement → bilateral hyperreflexia and Babinski responses → **decorticate posturing**.
    3) frequent yawns and sighs → ***Cheyne-Stokes respiration***.
3. **Midbrain stage** (reflects infarction rather than reversible ischemia and compression – prognosis poor):
    1) **pupils enlarge to midposition and become fixed**, disconjugate gaze with failure to adduct (internuclear ophthalmoplegia).
    2) **decerebrate posturing**.
    3) central neurogenic ***hyperventilation***.
4. **Pontine stage** – impairment of oculocephalic and oculovestibular reflexes, rapid shallow respirations, **flaccid extremities** with bilateral Babinski responses.
5. **Medullary stage** – flaccidity, Cushing's triad → pupils dilate, hypotension, slow irregular respirations → apnea, death.

N.B. coma may develop before eye signs appear!

UNCAL (S. LATERAL MASS) HERNIATION

- impaction of ANTERIOR MEDIAL TEMPORAL GYRUS into ***anterior portion of tentorial opening*** (into ambient cistern surrounding midbrain).

ETIOLOGY - expanding unilateral, supratentorial mass.

CLINICAL FEATURES

1) **CN3 palsy**:
   a) 80-85% - ***ipsilateral*** (CN3 is compressed against tentorial notch by inferomedial temporal lobe).
      N.B. pupillary fibers are located most peripherally in nerve, and are primarily damaged - *prominent early sign* is FIXED LARGE PUPIL! (level of consciousness may still not be significantly altered!); later complete CN3 paralysis develops (ptosis and lateral eye deviation).
   b) 15% - ***contralateral*** or ***bilateral*** (may relate to midbrain ischemia as brainstem moves away from basilar artery).

Mydriasis is most reliable sign for determining side of hematoma (correct in 85% cases)

   – CN3 palsy often *takes many months to resolve* once pressure is relieved.

2) PCA may be compressed against tentorium, producing **homonymous hemianopia** →→→ **PCA stroke**.

3) as diencephalon begins to shift laterally away from mass, **consciousness begins to diminish**; midbrain compression causes **coma**.
   N.B. early depression of consciousness correlates more with lateral brain displacement than with transtentorial herniation

   DIASCHISIS - function inhibition produced by acute focal disturbance in brain portion at distance from original injury, but anatomically connected with it through fiber tracts (e.g. sudden large unilateral lesion functionally disrupts contralateral hemisphere → coma).

4) **ipsilateral hemiplegia** (contralateral corticospinal tract in cerebral peduncle is compressed against tentorium edge - **KERNOHAN notch*** **phenomenon**) – false localization of primary lesion!
   *__notch__ - deep indentation of crus cerebri by free edge of tentorium – described in Kernohan and Woltman's 1929 article
   N.B. hemiparesis in 50% cases is **contralateral**!

Hemiplegia is not as accurate lateralizing sign as is mydriasis (50% vs. 80-85%)

5) eventually (if uncal syndrome is allowed to progress), **brainstem dysfunction**:
   – bilaterally fixed pupils, ophthalmoplegia, loss of brainstem reflexes.
   – breathing (normal → hyperventilation → ataxic).
   – bilateral decerebrate posturing (decorticate posturing is not usually seen), progressive flaccidity.
   – circulating collapse and death.

NEUROIMAGING

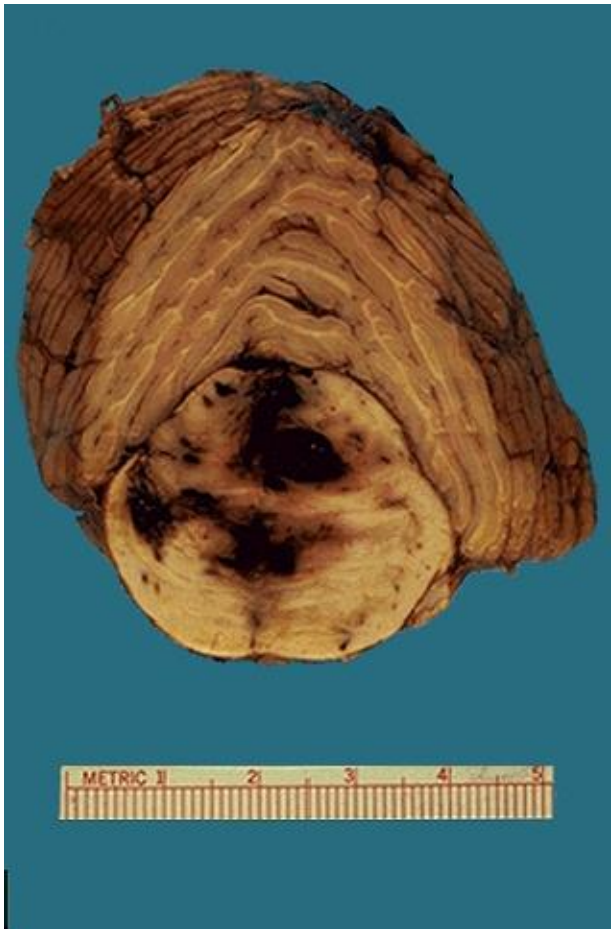
Best diagnosed by evaluating CSF spaces around midbrain (**MESENCEPHALIC CISTERNS**) - absence or occlusion of mesencephalic cisterns usually indicates dislocation of supratentorial structures below tentorial notch!

early – only *enlargement of ipsilateral perimesencephalic cistern* (*opposite perimesencephalic cistern is compressed* from lateral movement of brainstem);
later – *transtentorial movement* of temporal lobe (both perimesencephalic cisterns are compressed);
   – *uncus* and *hippocampus* are most medial portions of temporal lobe, they are first structures to cross tentorial edge.
   – movement of more posterior aspects of medial temporal lobe may *compress posterior cerebral artery* → ischemia in medial temporal lobe and sometimes in occipital lobe (hemianopsia).

N.B. **consciousness↓** occurs with moderate *lateral shifts at level of diencephalon* (when there is only minimal vertical displacement of structures near tentorial opening, i.e. well before downward herniation is evident on neuroimaging).

3-5 mm horizontal displacement of pineal body from midline corresponds to ***drowsiness***;
5-8 mm corresponds to ***stupor***;
> 8 mm corresponds to ***coma***.

- *aqueductal obstruction* may raise supratentorial pressure further.
- progression of transtentorial herniation is often accompanied by **DURET hemorrhages** (linear or flame-shaped hemorrhages in **midline** and paramedian regions of **midbrain and pons**):
  herniation displaces brainstem downward, stretching medial perforating branches of basilar artery (as artery is tethered to circle of Willis)



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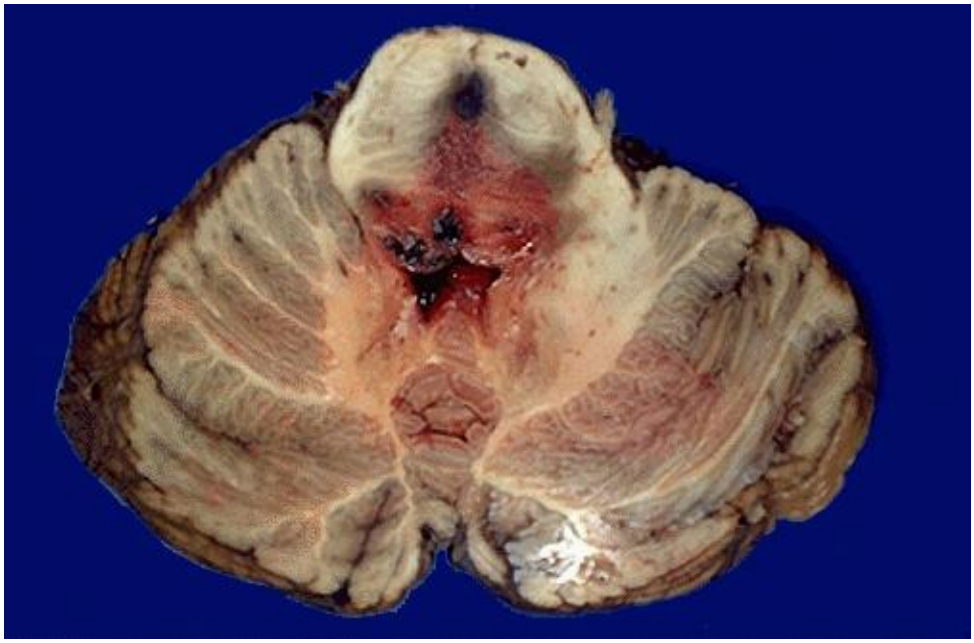

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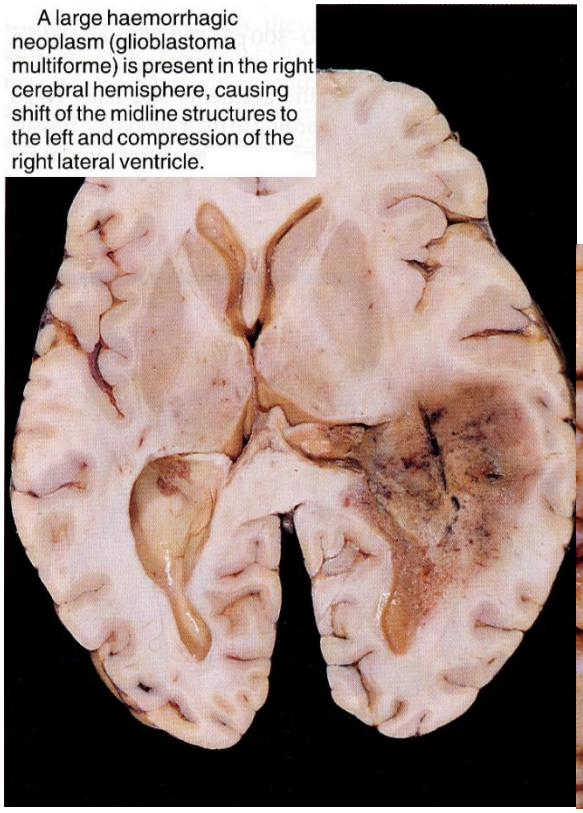
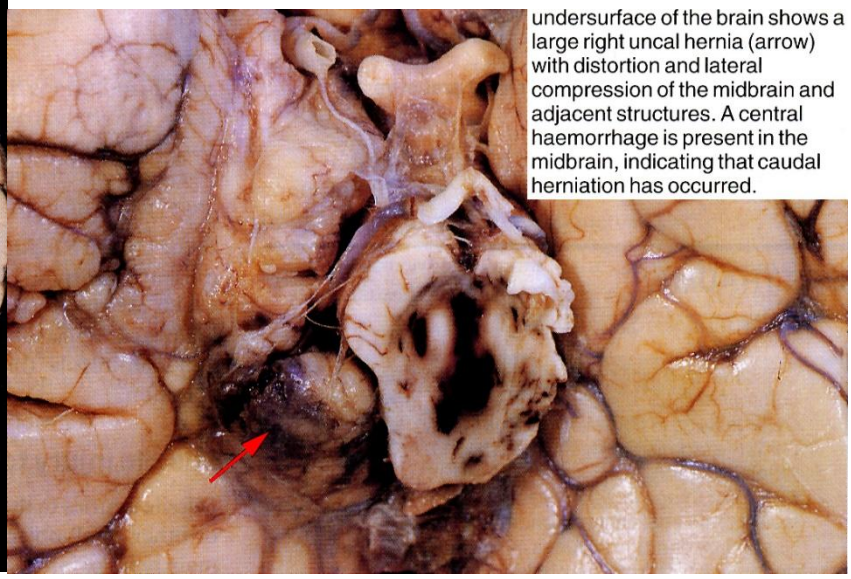


A large haemorrhagic neoplasm (glioblastoma multiforme) is present in the right cerebral hemisphere, causing shift of the midline structures to the left and compression of the right lateral ventricle.



undersurface of the brain shows a large right uncal hernia (arrow) with distortion and lateral compression of the midbrain and adjacent structures. A central haemorrhage is present in the midbrain, indicating that caudal herniation has occurred.

Arrow shows strangulation groove:

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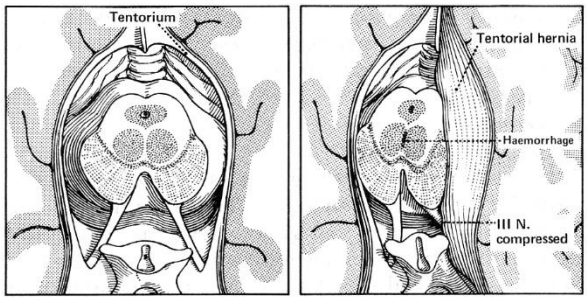
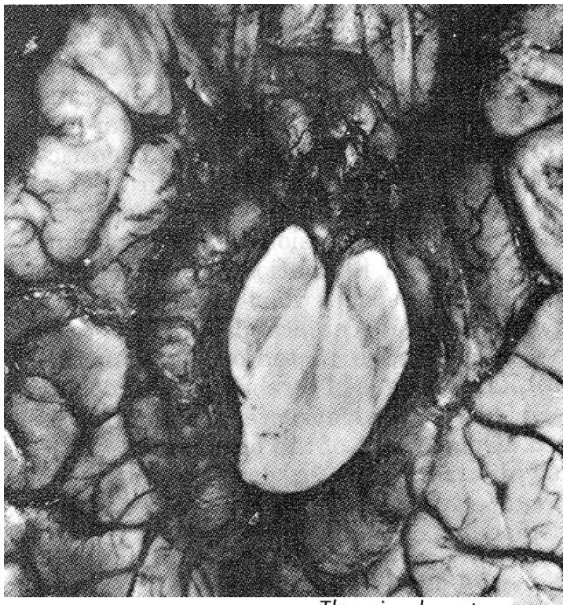
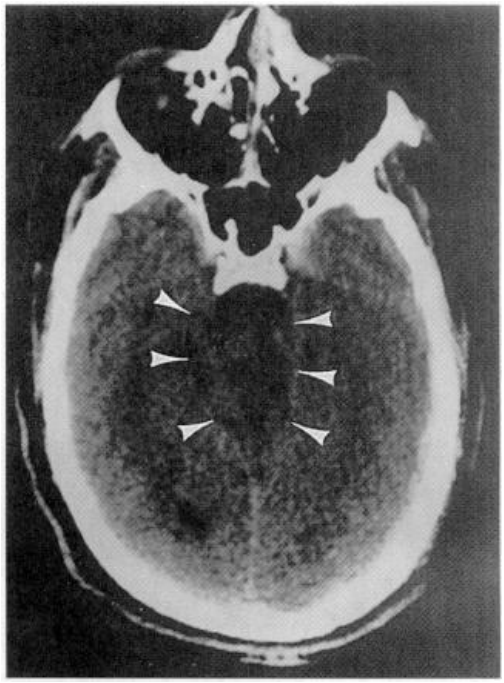


Fig. 20.13. Transverse section through mid-brain (from above), showing compression by a tentorial hernia. Left: normal. Right: abnormal



There is a large tentorial hernia on the left of the illustration and a smaller one on the right. The mid-brain is narrowed.



CINGULATE (s. SUBFALCINE) herniation

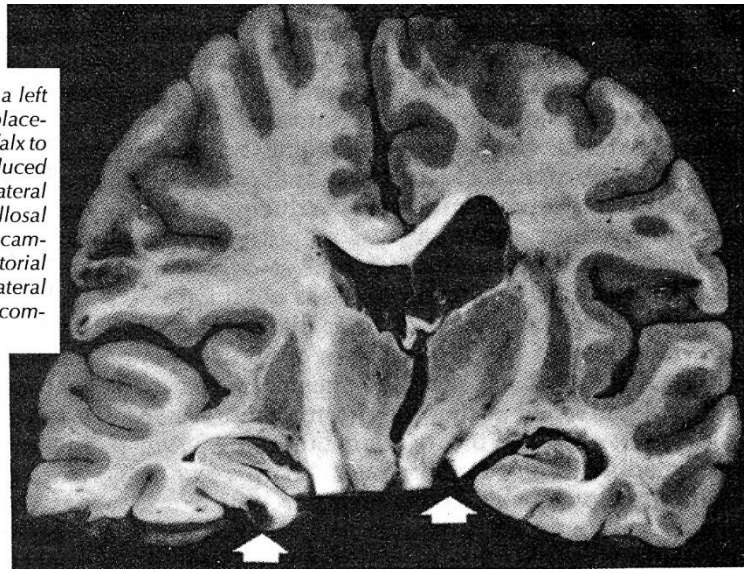
- MEDIAL FRONTAL STRUCTURES (e.g. cingulate gyrus) herniate ***beneath falx*** → compression on internal cerebral veins, ipsilateral anterior cerebral artery.

ETIOLOGY - frontal lobe masses.

CLINICAL FEATURES

1) signs related to mass itself
2) signs related to ICP↑
3) rarely, ischemia in *ipsilateral anterior cerebral artery* (loss of function in opposite leg, loss of bladder control).

- does not affect consciousness!



Internal herniae produced by a left subdural hematoma. In addition to lateral displacement, the cingulate gyrus has herniated under the falx to produce a supracallosal hernia. This has produced downward displacement of the roof of the left lateral ventricle and selectively severe shift of the pericallosal arteries. There is also hemorrhage in the left hippocampal gyrus (arrow) in the groove produced by a tentorial hernia and a hemorrhagic infarct in the contralateral cerebral peduncle (arrow) where it has been compressed against the tentorium.

INFRATENTORIAL MASSES

- infratentorial compartment is much smaller than 2 supratentorial compartments - ***compensatory mechanisms are exhausted much more quickly*** (tolerated volume increases are much smaller).

CEREBELLAR TONSILLAR herniation

- CEREBELLAR TONSILS pushed ***through foramen magnum***.

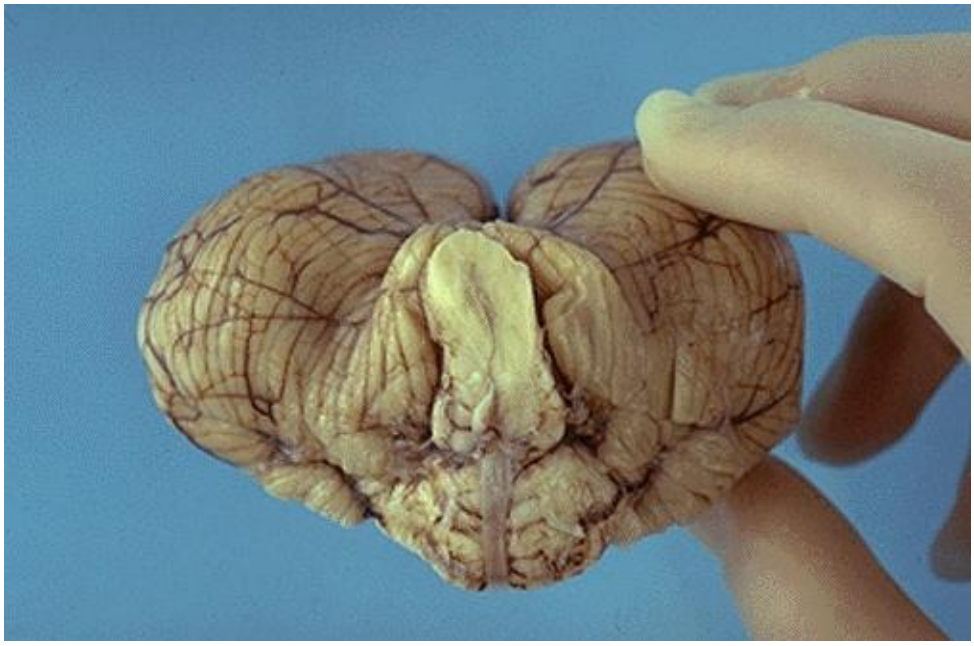
ETIOLOGY - posterior fossa (infratentorial) masses.

CLINICAL FEATURES - medulla compression:

1) stiff neck (due to tension of dura mater around cerebellar pressure cone).
2) flaccid quadriplegia (bilateral compression of corticospinal tracts)
3) ***apnea and circulatory collapse*** (as medulla is impinged) → secondary coma (medullary RF has little direct role in arousal!).

- mass itself may compress-destroy brainstem (→ pinpoint pupils, disconjugate eye movements, etc).
- mortality ≈ 70%

Note cone shape of tonsils around medulla:



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UPWARD TRANSTENTORIAL herniation

- CONTENTS OF POSTERIOR FOSSA herniate ***through tentorial opening*** into diencephalic region.

ETIOLOGY - **posterior fossa (infratentorial) masses** + **(overdraining) ventriculostomy**.

- posterior fossa masses usually herniate caudally because obstructive hydrocephalus they produce prevents pressure gradient across tentorial opening.
  N.B. obstructive hydrocephalus is particularly common complication of posterior fossa mass!
- ventriculostomy allows pressure gradient to quickly build up (never drain EVD at < 15 cmH2O in posterior fossa pathology).
- prepare for *emergent posterior fossa decompression* just in case this problem occurs.

CLINICAL FEATURES - midbrain compression:

1) ***rapid decline in level of consciousness***
2) ***upward gaze paresis***; other oculomotor signs.

- superior cerebellar artery ischemia → ataxia.

HERNIATION after LUMBAR PUNCTURE

- CSF removal lowers lumbar spinal canal pressure → increased gradient between cranial and lumbar compartments → **ROSTROCAUDAL HERNIATION**:
  a) **TRANSTENTORIAL**
  b) **CEREBELLAR TONSILLAR**.
- 50% patients lose consciousness immediately after LP.
- in 50% cases opening lumbar pressure is normal; 2/3 patients have no papilledema.
- general risk of herniation after lumbar puncture is 1.2-3%.
- preventive measures: see Op3 p.!
  1) **careful neurologic examination & CT** before LP.
  2) **small-caliber spinal needles**.
  3) **minimal CSF volume loss**.
  4) aggressive concurrent use of **ICP-lowering agents**.

INVESTIGATIONS

CT findings that suggest ***unequal pressure*** between intracranial compartments:

1) lateral shift of midline structures
2) loss of suprachiasmatic, circum-mesencephalic, superior cerebellar and quadrigeminal plate cisterns with sparing of ambient cisterns.
3) shift or obliteration of 4th ventricle
4) posterior fossa mass (strong contraindication to lumbar puncture!).

- CT may miss cerebellar tonsillar herniation due to beam hardening artifact at level of foramen magnum.

Angiographic signs:

subfalcine herniation → displacement of ACA or internal cerebral veins across midline;
transtentorial herniation → medial displacement of anterior choroidal artery;
tonsillar herniation → displacement of PICA branches below foramen magnum.

TREATMENT

Herniation must be recognized and treated ***immediately*** (to prevent irreversible brain damage)!

1. Temporary **measures to lower ICP**: see p. S50 >>
   1) **hyperventilation**.
   2) MANNITOL.
   3) DEXAMETHASONE.

2. **Neurosurgical intervention** (evacuation of mass).

BIBLIOGRAPHY see p. S50 >>