# SPINAL CORD CYSTS AND CAVITATIONS Forrest Hsu and Dr. Hurlbert FMC NSX HALFDAY 07JUN07

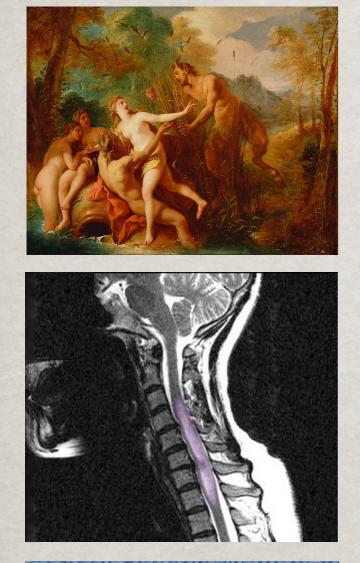
# OBJECTIVES

## **Definitions**

Pathophysiology/ClassificationClinical Presentation

\* Management

# "SYRINX"





Greek mythological character

\* Fluid filled cyst within spinal cord

### Syringohydromyelia:

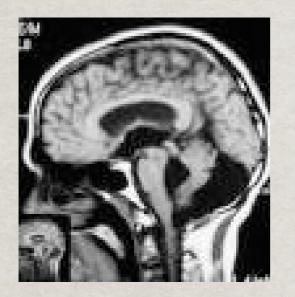
- Syringomyelia: fluid collections outside of central canal
- \* Hydromyelia: fluid collections enlarging central canal, congenital, almost always benign. Usually lined by ependymal cells.

# PATHOPHYSIOLOGY

Rare, poorly understood natural history

Associated w/: \* Trauma Chiari malformations Tumour Vascular malformations Arachnoiditis Occult Spinal Dysraphisms

## TRADITIONAL CLASSIFICATION



<u>Communicating</u> Chiari Malformations Spinal Dysraphisms



### Non-Communicating

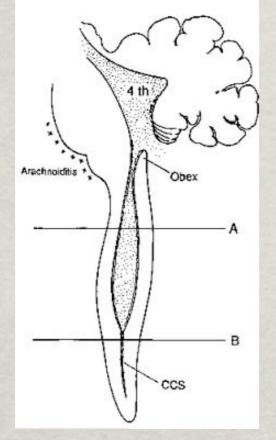
Trauma Tumour Vascular Malformations Arachnoiditis

## **ANATOMICAL CLASSIFICATION**

Classification of syringomyelia

- I communicating syringomyelia central canal dilations
  - communicating hydrocephalus (posthemorrhagic, postmeningitic)
  - complex hindbrain malformations (Chiari II, encephalocele)
  - 3) Dandy-Walker cyst
- II noncommunicating syringomyelia
  - central canal/paracentral syringes
    - 1) Chiari malformations
    - 2) basilar invagination
    - 3) spinal arachnoiditis (posttraumatic, postmeningitic)
    - extramedullary compressions (spondylosis, tumors, cysts)
    - tethered cord
    - 6) acquired tonsillar herniation (hydrocephalus, intracranial mass lesions, craniosynostosis)
  - primary parenchymal cavitations
    - 1) spinal cord trauma
    - 2) ischemia/infarction
    - 3) intramedullary hemorrhage
- III atrophic cavitations (syringomyelia ex vacuo)
- IV neoplastic cavitations

### COMMUNICATING SYRINX'S CAUSED BY OBSTRUCTION BELOW 4TH VENTRICLE



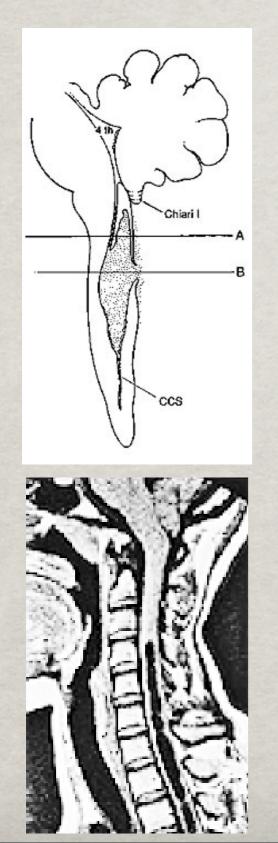


Communicating central canal dilatations are caused by obstructions of the CSF pathways **distal to 4th Ventricle** <u>Etiology</u>:

- \* Postmeningitic/Post hemorrhagic hydrocephalus
- Basilar/Medullary Archnoiditis
- Chiari II Malformations
- # Encephalocele
- Dandy-Walker Cysts

- Length defined by canal stenosis
- Congenital lesions may be contiguous (myelomenigocele)
- Rare to rupture/dissect paracentrally
- Most cases assymptomatic w/minor neuro findings

### NON-COMMUNICATING SYRINX'S CAUSED BY OBSTRUCTION @ FORAMEN MAGNUM



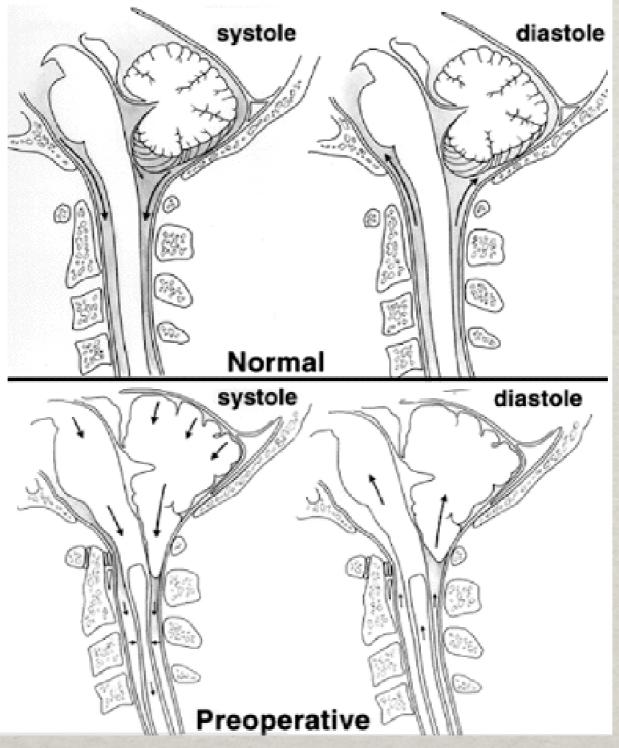
Non-Communicating central canal dilatations are caused by obstructions of the CSF pathways at or **below foramen magnum**.

Etiology:

- Chiari I malformations / Basilar Invagination
- Spinal Arachnoiditis
- Extramedullary compression(spondylosis,tumor,cysts)
- Tethered cord
- Acquired Tonsiller herniation

- Length defined by force of arterial pulse wave in subarachnoid space "cord diverticuli" via virchow-robin spaces
- Isolated cavities flanked by spinal canal stenosis
- Commonly rupture/dissect paracentrally longitudinally along white-matter tracts
- Symptomatic w/ progressive neuro findings

#### PROPOSED MECHANISM OF NON-COMMUNICATING SYRINX 2° TO FORAMEN MAGNUM OBSTRUCTION HEISS-OLDFIELD THEORY



Heiss et al. J Neurosurg 1999. 91(4):553-62.

# **ALTERNATE THEORIES**

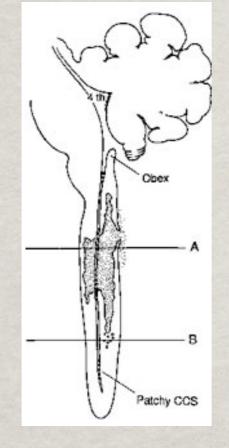
Hydrodynamic: Water-Hammer theory (aka Theory of Gardner).

- systolic pulsations transmitted from intracranial cavity down thru central canal
- Now disproven with MRI studies

Williams Theory:

Valsalva manuevers raise CSF pressure causinghydrodissection thru spinal cord tissue

### Non-Communicating 1° Parenchymal Syrinx's CAUSED BY CORD INJURY





Tubular cavitations of the spinal cord that originate within the spinal cord parenchyma. No communication w/central canal or 4th Ventricle.

Etiology:

- # Trauma
- Ischemia/Infarction
- Spontaneous intramedullary hemorrhage

- Length defined by focal arachnoiditis @ time of injury drives CSF into interstitial spaces (thru virchow-robin spaces -similar to Heiss-Oldfield theory)
- Typically arise in watershed areas of spinal cord, dorsal and lateral to the central canal.
- Commonly rupture/dissect paracentrally w/ longitudinal extension along white matter tracts.
- Symptomatic w/ progressive neuro findings

### ATROPHIC CAVITATIONS ARE CAUSED BY SPINAL CORD DEGENERATION



Degenerative changes with cord atrophy leads to formation of microcysts, intramedullary clefts and localized dilatations of the spinal canal aka Syringomyelia ex vacuo.

Etiology:

- Spondylolysis
- Old Trauma

- Length defined by area of myelomalacia
- May or may not communicate w/central canal
- Can be symptomatic w/ progressive neuro findings

### **NEOPLASTIC CAVITATIONS**



Syrinx-like cavities may form from cystic degeneration of intramedullary tumors. Cysts contain proteinaceous fluid NOT CSF.

Etiology:

- Astrocytomas
- Ependymomas
- Oligodendroglioma
- Spinal metastases
- Natural Hx:
  - Length defined by tumor volume and necrosis that starts centrally and extends rostral and caudal.
  - Typically arise in watershed areas of spinal cord, dorsal and lateral to the central canal.
  - Commonly rupture/dissect paracentrally thru to subarachnoid space.
  - Symptomatic w/ progressive neuro findings

# **CLINICAL PRESENTATION**

#### Symptoms

- # Pain/Numbness
- Weakness/Fatigue
- Stiffness
- ₩ H/A
- Exacerbation w/valsalva
- Loss of temperature sensation

#### Signs

- loss of pain and temperature sensation in chest and arms
- Dissociated sensory loss
- motor limb weakness U/E>L/E
- Spasticity
- Ataxia
- Cord Syndromes

| Syndrome              | Motor                            | Sensory   |
|-----------------------|----------------------------------|---|
| Brown-Sequard         | Ipsi paresis                     | Contra loss of pain and temp<br>Ipsi loss of vibration and proprio<br>Preserved tactile sensation |
| Anterior Spinal Cord  | Bilateral paresis                | Loss of pain and temp<br>Preserved vibration and proprio  |
| Posterior Spinal Cord | Preserved                        | Loss of 2pt, vibration and proprio<br>Preserved pain, temp, and tactile                           |
| Central Cord          | Variable segmental<br>amyotrophy | Bilateral loss of pain and temp<br>Preserved touch, vibration, and proprio                        |

## MANAGEMENT PRINCIPLES

Severe chronic pain

Progressive neurological deficit

Medical vs. Surgical

# SURGICAL GOALS

Prevent worsening neurological deficit! <u>Extramedullary decompression</u> (Chiari's, Arachnoiditis) © Occipital-C1 posterior decompression w/ duroplasty © Laminectomy +/- duroplasty <u>Intramedullary decompression</u> © cyst drainage (Post-traumatic cysts) © tumor debulking <u>Shunting</u>

VP Shunt (hydrocephalic cases)

Syringo-pleural/peritoneal (post-traumatic cysts)

## HURLBERT'S CLASSIFICATION

Determine whether Syringo- vs. Hydro- myelia (central vs. paracentral) Syringomyelia 1° vs 2°

- 1°= Idiopathic dx of exclusion 2°= Etiology:
  - % Chiari I or II
  - \* Post Traumatic
  - # Tumour
  - \* Vascular Malformation
  - # Arachnoiditis

## MANAGEMENT PRINCIPLES

Hx/PHx:

Be sure to ask for features of Chiari I/II and Myelomenigocele, Tethered cord, trauma, tumour, infection

Imaging:

MR Head MR C-T-L spine Management: Mechanism Location Expansion/Progression

# REFERENCES

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- Screenberg MS. Syringomyelia. <u>Handbook of Neurosurgery</u>. Thieme 2001 p348-352.
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