Normal Pressure Hydrocephalus
“A Workshop”
Why did you come and what would you like to get out of it?!
Why is Normal Pressure Hyrdocephalus Relevant to General Practice?

1. NPH is a well recognised problem
2. Treatment can be extremely beneficial for many patients
3. However, there are controversies regarding NPH and its treatment
4. There can be complications of treatment
5. There are some misconceptions about its treatment (amongst patients, neurosurgeons, and neurologists)
GOALS

Overall Aim

To increase your knowledge base about Normal Pressure Hydrocephalus to better patient care.

Specific Goals

1. To review CSF physiology and hydrocephalus
2. To review how hydrocephalus can be treated
3. To understand how NPH differs from other forms of hydrocephalus
4. To understand the principles of Endoscopic Third Ventricle (ETV)
5. To understand the principles and complications of Ventriculoperitoneal Shunts (VPS)

To think like a neurosurgeon for a brief period of time...
The skull is a rigid box and the contents (CSF, brain, blood) are in a state of volume equilibrium. An increase in the volume of one must be compensated by a reduction in volume of the other in order to maintain a constant pressure.

Alexander Monro
Primus (1720-1758)
Professor of Anatomy, Edinburgh
Definition of hydrocephalus:

- an excessive build up of cerebral fluid (CSF) within the head

- due to a problem with flow, absorption or formation of CSF

- (Usually the fluid builds up in the ventricles)
The Ventricular System
Figure 53. Dorsal view of a cast of the brain ventricles of man. (Retzius.)
CSF Production & Circulation

- CSF is made in the ventricles
- Mostly by the choroid plexus at a rate of 0.35 mls/min (500-750 mls/day)
- Total CSF volume in body is normally 120-150 mls (20 mls in the ventricles)
- Absorption primarily by arachnoid granulations
- Normal ICP 120 - 180 mmH2O in recumbent adults (less in children)
Types of Hydrocephalus

• OBSTRUCTIVE:
  – block to CSF flow within the ventricular system (eg Aqueduct stenosis, spina bifida, tumours)

• COMMUNICATING:
  – Block in subarachnoid space/ arachnoid granulations (eg meningitis, bleeding)
Communicating Hydrocephalus

2. Overproduction of CSF (very rare)

Choroid Plexus Papilloma
Aetiology  
Congenital & acquired

Congenital malformations:
- aqueduct obstruction  
  (CSF flow is impaired when cross-sectional area of aqueduct is <0.25mm²)
- Arnold Chiari malformation
- Dandy-Walker syndrome
- benign intracranial cysts
- vein of Galen aneurysms
- congenital CNS infections
- craniofacial anomalies

Acquired causes:
1. Tumours and cysts
   Post.fossa, pineal, 3rd ventricle
   eg astrocytoma, choroid plexus papilloma
2. Inflammation
   Meningitis
3. Absorption blockages
   SAH, normal pressure hydroceph (NPH)
Investigations for hydrocephalus:

- Ultrasound
- CT SCAN
- MRI SCAN - in older children
- CSF analysis - cells, protein
- Lumbar infusion studies
- Tympanic membrane displacement
- Transcranial Doppler
Clinical Presentation: Neonates

- Increased head circumference & dilated scalp veins
- Tense fontanelle
- Developmental delay
- Failure to thrive
- "Setting sun" sign
Clinical Presentation:
children & adults

- Symptoms of raised intracranial pressure
  - headaches
  - vomiting
  - drowsiness or coma
  - visual deterioration
  - double vision

- Gradual decline in mental function

- Hormone disturbance

- CSF rhinorrhoea
Clinical Presentation: children & adults

- Papilloedema

Normal optic disc

Papilloedema
Hydrocephalus: Treatment Options
DRIFT treatment - for premature babies

DRIFT: Set-up Of Drainage-Irrigation System
Basic Principles

- Correct **indication** & big enough ventricles
- Correct **site** of burrhole
- Correct **trajectory** of ventric cannulation
- Cannulate on 1st attempt. Avoid ‘plunging’
- Avoid distortion of the brain - use peel-apart cannula, flexible scopes
- RIGID for ETVs, flexible for other uses
Current Indications for Third Ventriculostomy (ETV)

• **Strong indications**
  - Delayed-onset aqueduct stenosis
  - Obstructive hydrocephalus due to pineal-region and some posterior fossa tumors.
  - CSF shunt obstruction in any patient with a history of obstructive hydrocephalus and large enough ventricles for endoscopy to be safe.
  - Older patients with spina bifida and a blocked CSF shunt.
ETV

• **Weaker indications**
  - Neonates with aqueduct stenosis, or spina bifida
  - CSF shunt obstruction in children or adults with history of communicating hydrocephalus

• **Contra-indications** - previous infection / ?DXT
ETV - technique

Head up !!
Site the burrhole carefully
Curved Incision to Accommodate Ventricular Reservoir
Carefully planned trajectory
Have a good assistant if possible
Don’t let the Peel-Apart plunge!
Mark the desired depth on the Peel-Apart Cannula
Try not to ‘distort’ the brain
Double Balloon Catheter - NMT
Endoscopy
Third ventriculostomy
Shunts

From here

To here

V-P Shunt

V-A Shunt

MANAGEMENT
Shunt valve mechanisms

(A) Slit valve

(B) Diaphragm valve

(C) Ball in cone valve
When CSF pressure builds up to a certain pressure, the opening pressure of the valve, ...
Programming
Valve programming
SIPHONING

\[ IVP = OPV - HP - IAP \]

**Intraventricular Pressure (IVP)**

**Intraabdominal Pressure (IAP)**

**Hydrostatic Pressure (HP)**

**Opening Pressure Valve (OPV)**

**Vertical Position**

\[ OPV = 5; \ IAP = 0; \ HP = 30 \]

\[ IVP = -25 \]

**Horizontal Position**

\[ OPV = 5; \ IAP & HP = 0 \]

\[ IVP = 5 \]
What is normal pressure hydrocephalus?

NPH is a syndrome that was first described by Hakim in his thesis in 1964.

Hakim S. Some observations on CSF pressure. Bogata, Javerianna University School of Medicine, 1964 (Thesis No. 957)
What is normal pressure hydrocephalus?

**NPH is a syndrome that was first described by Hakim in his thesis in 1964**

Hakim S. Some observations on CSF pressure. Bogata, Javerianna University School of Medicine, 1964 (Thesis No. 957)

**NPH entered the literature in 1965 - 3 cases of hydrocephalus described**


What is normal pressure hydrocephalus?

NPH is a syndrome that was first described by Hakim in his thesis in 1964

Hakim S. Some observations on CSF pressure. Bogata, Javerianna University School of Medicine, 1964 (Thesis No. 957)

NPH entered the literature in 1965 - 3 cases of hydrocephaalus described


Hakim’s triad of symptoms in all patients

1. Gait disturbance
2. Cognitive deterioration
3. Urinary incontinence

Associated with ventricular enlargement but without any elevated CSF pressure

Symptoms improve with CSF drainage (15ml)
Classification of NPH

Secondary NPH

- Other disease process precedes the syndrome of NPH
- SAH, traumatic brain injury, cerebral infarction, meningitis

Primary, or idiopathic NPH (INPH)

- NPH without any known precipitants

Pathophysiology of NPH is unknown - but it may represent a unique form of reversible neuronal injury
Classification of NPH

Is this classification important?

• It may affect the diagnostic approach if the potential for recovery if NPH is less influenced by comorbid factors.

• However, it appears that comorbidity, particularly in >70’s, is equally important in INPH as secondary NPH.
Epidemiology of NPH

- Very few studies available
- Incidence reported as 1.8 - 2.2 cases per 100,000 individuals in US
- In Germany, prevalence in >65’s estimated at 0.41% (door-to-door survey of villages)
- 1.6 - 5.4% of patients with dementia estimated to have NPH
Why is NPH important?

1) Numbers of NPH patients appear to be increasing

   - Increased neurological/neurosurgical services?
   - Increased longevity?
   - Increased demand for surgical options in >80’s?

2) Although several thousand papers on NPH - there are no accepted evidence-based guidelines
How is NPH diagnosed?
How is NPH diagnosed?

http://www.lifenph.com

SELF DIAGNOSIS ON THE INTERNET.....!
How is NPH diagnosed?

5 STEPS to Diagnosis & Treatment

If you believe you or a friend are experiencing symptoms of NPH, please follow these 5 steps:

1. **Complete** online screening test and print for use with primary care physician.

2. **Make an appointment** with your primary care physician. Describe your symptoms and provide a printout of your screening test.

3. **Ask your primary care physician** for an MRI test referral. This step is critical to receiving an accurate diagnosis.

4. **Make an appointment** and complete your MRI test.

5. Using the **physician locator**, **find a neurosurgeon** near you and make an appointment. Be sure to bring your MRI results to the appointment.

http://www.lifenph.com
1. My feet feel stuck to the floor when I walk.
   YES  NO

2. I have trouble keeping my balance when walking or turning.
   YES  NO

3. I have experienced sudden falls without loss of consciousness.
   YES  NO

4. I have difficulty maintaining attention.
   YES  NO

5. I have trouble remembering things.
   YES  NO

6. I have experienced sudden urgency in urinating.
   YES  NO

7. I have had urinary accidents
   YES  NO
“Screening test”

1. My feet feel stuck to the floor when I walk.
   YES      NO

2. I have trouble keeping my balance when walking or turning.
   YES      NO

3. I have experienced sudden falls without loss of consciousness.
   YES      NO

4. I have difficulty maintaining attention.
   YES      NO

5. I have trouble remembering things.
   YES      NO

6. I have experienced sudden urgency in urinating.
   YES      NO

7. I have had urinary accidents
   YES      NO

You scored 7 out of a possible 7.

Your answers suggest that you may have Normal Pressure Hydrocephalus.

We encourage you to discuss your results with your doctor -

Because only a doctor can make a diagnosis!

http://www.lifenph.com
Diagnosis of NPH is a Dark Art

1. Incidence and prevalence of NPH not known
2. There is no agreement on the precise diagnostic criteria
3. CSF pressure is not truly normal
4. The natural history of untreated NPH has not been studied systematically
Diagnosis of INPH is a Dark Art

However...

The clinical features and prognosis of NPH are considered sufficiently unique as a syndrome to warrant treatment.

Early treatment of NPH may prevent severe and irreversible impairments some patients.
Guidelines for Diagnosis of INPH

- NPH independent study group assembled in 2000 and evidentiary tables assembled over next few years
- Consensus presented at various workshops for discussion
- Final consensus published in Neurosurgery Supplement in 2005

Further classified INPH into 3 groups based on clinical findings, neuroradiology, and physiological parameters:

- Probable INPH
- Possible NPH
- Unlikely NPH
Clinical Presentation of NPH

- Primary triad of gait disturbance, dementia, and urinary incontinence

  **Gait** - broad-based, slow, shuffling steps, difficulty turning, often the first symptom

  GAIT DISTURBANCE MUST BE PRESENT FOR DIAGNOSIS OF PROBABLE INPH

  **Dementia** - inattention, psychomotor retardation, diminished executive function

  THERE MUST BE ‘DOCUMENTED’ IMPAIRMENT OF COGNITION FOR DIAGNOSIS OF PROBABLE INPH

  **Urinary incontinence** - frequency and urgency early on, develop to full incontinence later, urodynamic testing may reveal hyperactive bladder

- Symptoms insidious in onset, may be a history of falls

- Peak incidence over age of 60

- No evidence of HI, ICH, meningitis etc. (for idiopathic NPH, otherwise NPH)
Differential Diagnosis of NPH - must be considered in each case

**Gait disturbance**
- Peripheral neuropathy
- Cervical/lumbar stenosis
- Vestibular disease
- Parkinson’s disease

**Dementia**
- Other dementias (LBD, PD, vascular dementia)
- Alzheimer’s disease

**Urinary incontinence**
- Prostate problems
- Stress incontinence
- Chronic UTIs
Radiological Evaluation of NPH

CT or MRI Head

Features necessary for diagnosis of INPH
- Ventriculomegaly - not attributable to cerebral atrophy
  - no macroscopic obstruction of CSF flow
- Evan’s index >0.3 (frontal horn ratio = maximal frontal horn width/internal diameter skull)

Associated features
- Periventricular hyperintensities (small vessel disease)
- Thinning and elevation of corpus callosum on sagittal images
- Aqueductal flow void on MRI
Physiological Evaluation of NPH

CSF opening pressure on lumbar puncture

• Normal range = 8.8 +/- 0.9 mmHg

• NPH = 11 +/- 3.3 mmHg

• Consensus guidelines indicate that a CSF-OP of 5-18 mmHg must be present for diagnosis of probable NPH
“The only reliable means of validating a diagnosis of NPH is to document a positive response to shunt placement”
The only reliable means of validating a diagnosis of NPH is to document a positive response to shunt placement


But there are several problems with this philosophy -

• False negatives would occur - patients with true NPH would have diminished response with co-existing disorders, e.g., Alzheimer’s, Parkinson’s

• False positives - patients with similar conditions, e.g., aqueduct stenosis, would benefit from shunt (perhaps this is OK...?)

• Placebo effect
Supplemental Prognostic Tests in NPH

Are they needed?

- Without additional testing 46-61% of probable and possible NPH patients will improve with surgical treatment (Gallia et al. Nature 2006)

- Therefore, any supplementary tests that will predict which patients will respond to shunt placement will be beneficial
Supplemental Prognostic Tests in NPH

Are they needed?

• Without additional testing 46-61% of probable and possible NPH patients will improve with surgical treatment (Gallia et al. Nature 2006)

• Therefore, any supplementary tests that will predict which patients will respond to shunt placement will be beneficial

Tests available

• ICP monitoring
• CSF tap test
• CSF drainage via spinal drainage
• CSF outflow resistance determination
How can supplemental tests be assessed?

Move the goal posts!

Redefine the diagnosis to -

• Shunt responsive NPH (SRNPH)
• Shunt non-responsive NPH (SNRNPH)

Therefore, SRINPH can be used to calculate specificity, sensitivity, positive predictive value (PPV), and negative predictive value (NPV)
ICP Monitoring

Aim = to look for B-waves

• "B-waves" - sinusoidal or ramp-form waves in the frequency range of 0.5/min to 3/min, and whose occurrence may indicate impending brain pressure attacks.
• Hypothesis = increased frequency of B waves during sleep is indicative of lowered compliance

Criticisms

1) Lack of age-matched control data
2) No correlation between overnight B waves and CSF-OP on LP

Evidence?
One small retrospective study only (Class III evidence)
20/24 patients showed ‘improvement’ with shunts
(Raftopoulos et al. Neurol Res 1992)
Tap-Test

Aim = to observe improvement with removal of 40-50 ml of CSF

Criticisms

- What is meant by ‘improvement’ - very subjective
- Can be overcome by using objective measures

Evidence?

Two class II studies only

<table>
<thead>
<tr>
<th>Series (ref. no.)</th>
<th>INPH/SNPH (n)</th>
<th>Sensitivity</th>
<th>Specificity</th>
<th>PPV</th>
<th>NPV</th>
<th>Accuracy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Malm et al., 1995 (15)</td>
<td>35/0</td>
<td>62% (16/26)</td>
<td>33% (3/9)</td>
<td>73% (16/22)</td>
<td>23% (3/13)</td>
<td>54% (19/35)</td>
</tr>
<tr>
<td>Walchenbach et al., 2002 (23)</td>
<td>43/6</td>
<td>26% (9/35)</td>
<td>100% (12/12)</td>
<td>100% (9/9)</td>
<td>32% (12/38)</td>
<td>45% (21/47)</td>
</tr>
</tbody>
</table>
CSF drainage via spinal drainage

Aim = to observe improvement with prolonged, controlled CSF drainage, e.g., 10ml/hr via lumbar drain over 72 hours

Criticisms as per tap-test

Evidence?

Class III studies only

<table>
<thead>
<tr>
<th>Series (ref. no.)</th>
<th>INPH/ SNPH (n)</th>
<th>Sensitivity</th>
<th>Specificity</th>
<th>PPV</th>
<th>NPV</th>
<th>Accuracy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Haan and Thomeer, 1988 (10)</td>
<td>17/0</td>
<td>100% (12/12)</td>
<td>100% (5/5)</td>
<td>100% (12/12)</td>
<td>100% (5/5)</td>
<td>100% (17/17)</td>
</tr>
<tr>
<td>Williams et al., 1998 (25)</td>
<td>86 (mixed)</td>
<td>97% (31/32)</td>
<td>60% (9/15)</td>
<td>84% (31/37)</td>
<td>90% (9/10)</td>
<td>75% (40/47)</td>
</tr>
<tr>
<td>Walchenbach et al., 2002 (23)</td>
<td>43/6</td>
<td>50% (14/28)</td>
<td>80% (8/10)</td>
<td>80% (14/16)</td>
<td>36% (14/22)</td>
<td>58% (22/38)</td>
</tr>
</tbody>
</table>

* INPH, idiopathic normal-pressure hydrocephalus; SNPH, secondary NPH; PPV, positive predictive value; NPV, negative predictive value.
CSF Ro Studies

Aim = to measure the impedance to flow of CSF absorption pathways, i.e., outflow resistance (Ro)

Various methods

1) Katzman test
   - Infusion of saline at a known rate into lumbar subarachnoid space
   - $Ro = \frac{\text{final steady state pressure} + \text{opening pressure}}{\text{infusion flow rate}}$

2) Bolus method
   Infusion of known volume of saline (4ml) in lumbar subarachnoid space at a rate of 1 mls/s

Considerations
- Ro values depend on method used
- Ro values known to increase in age
CSF Ro Studies

Evidence?

One class II study for INPH

<table>
<thead>
<tr>
<th>Series (ref. no.)</th>
<th>INPH/SNPH (n)</th>
<th>Ro threshold (mm Hg/ml/min)</th>
<th>Sensitivity</th>
<th>Specificity</th>
<th>PPV</th>
<th>NPV</th>
<th>Accuracy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Malm et al., 1995 (15)</td>
<td>35/0</td>
<td>58% (15/26)</td>
<td>44% (4/9)</td>
<td>73% (15/20)</td>
<td>27% (4/15)</td>
<td>54% (19/35)</td>
<td></td>
</tr>
</tbody>
</table>

Three class II studies for NPH

<table>
<thead>
<tr>
<th>Series (ref. no.)</th>
<th>INPH/SNPH (n)</th>
<th>Ro threshold (mm Hg/ml/min)</th>
<th>Sensitivity</th>
<th>Specificity</th>
<th>PPV</th>
<th>NPV</th>
<th>Accuracy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Boon et al., 1997 (2)</td>
<td>101 (mixed)</td>
<td>&gt;18</td>
<td>46%</td>
<td>87%</td>
<td>92%</td>
<td>34%</td>
<td>67%</td>
</tr>
<tr>
<td>Børgesen et al., 1979 (3)</td>
<td>20/40</td>
<td>&gt;8</td>
<td></td>
<td></td>
<td>96%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Kahlon et al., 2002 (12)</td>
<td>51/68</td>
<td>&gt;14</td>
<td></td>
<td></td>
<td>80%</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Getting it Wrong....
Case Presentation - SS

46 F

R handed nursery teacher

First seen by Neurologists in January 2006 with -

1 year history of headaches

‘intense’

duration 1-2 hours

associated nausea and vomiting

frequency of headaches not specified
SS

46 F

R handed nursery teacher

First seen by Neurologists in January 2006 with -

1 year history of headaches
  ‘intense’
  duration 1-2 hours
  associated nausea and vomiting
  frequency of headaches not specified

Background

Large head noted as a child

Also noted to shake head quite often

Tetralogy of Fallot

Diagnosed with Grave’s disease in 2006 and
started on thyroxine
January 2006

Clinical examination

- Normal fundi
- Horizontal diplopia on R lateral gaze
- Partial ptosis on right
- R pupil size > L pupil size
- Up-going plantars
- No other neurological deficits
SS

Seen February 2006 by Mr Stacey

Referral query - Arrested hydrocephalus with recent decompensation?

- History of headaches for > 18 years
- Headaches worse over the last year with morning headache and double vision
- Subjective decrease in short-term memory

MRI – possible aqueduct stenosis, no obstructive mass lesion
SS

Admitted March 2006

Consented for ICP monitor (R frontal)

ICP monitoring – totally normal

Re-referred in 2007 by neurologists - ? NPH

Given date for repeat ICP monitoring but unable to attend due to needing electrical cardioversion for abnormal rhythm
SS

Re-admitted for ICP monitor

- Detailed consent taken for re-insertion ICP monitor
- ICP monitor reading in theatre = 45 mmHg
- ICP monitor changed after 5 min as reading so high
- New reading 38, dropped to 30 after another 5 min
SS

- ICP settled to a baseline of aprox. 10 mmHg
- The following runs sustained high ICPs recorded

31/7/07 23:08 > 30 mmHg for 1 min
01/08/07 01:56 > 31 mmHg for 1 min
01/08/07 07:30 > 34 mmHg for 30 secs
01/08/07 03:58 > 40 mmHg for 4 min
01/08/07 04:03 > 40 mmHg (spikes) for 4 min
02/08/07 04:02 > 25 mmHg for 2 min
Baseline

Example pressure waves
What would you do next?

Decision to place insert programmable VP shunt set @ 90 mmHg
SS CT Head Post-Insertion VP-Shunt
SS

Discharged home 3 days post-shunting
SS

Discharged home 3 days post-shunting
Readmitted 5 days later with confusion and ataxia
Repeat CT Head showed small subdural hygromas (NOT bleeding)
SS CT Head 9/7 post shunt
SS

Shunt setting changed to 180

SS improved over next 8 days and discharged home with plan to repeat CT in 2 weeks time
SS CT Head after increase in valve setting to 180

Readmitted 10 days later with recurrent confusion and ataxia

Repeat CT Head showed bilateral CSDH’s

What would you do next?
What would you do next?

Burr-holes performed

Patient went into fast AF post-op and had DC cardioversion

Shunt setting checked - still at 180

CSDH still present on CTH
What would you do next?
Decision made to remove shunt

SS CT Head 4/7 post shunt removal
Patient’s symptoms resolved

Including diplopia, headaches, ataxia, and UMN signs!
Summary

Patient hopes and expectations are critical

There are no promises and no guarantees of success

But there is certainty of risk

This needs to be understood

Best results if GP and neurosurgeon understand the prospects!
Conclusions 2

A multi-centre trial needed with the following criteria

• Patients are classified into probable, possible and unlikely NPH categories
• Multiple supplemental tests administered +/- randomisation of tests
• Shunts every patient with acceptable surgical risk irrespective of supplemental testing results
• Uses well-defined end-points with inter-observer reliability
ETV v VPS for NPH?

<table>
<thead>
<tr>
<th>Decade of publication</th>
<th>Number of studies</th>
<th>Number of patients</th>
<th>Age (mean ± SD)</th>
<th>Percentage of improved patients at 3 months postoperatively</th>
<th>Percentage of improved patients at 1 year postoperatively</th>
<th>Percentage of improved patients at more than 3 years postoperatively</th>
</tr>
</thead>
<tbody>
<tr>
<td>1970s</td>
<td>2</td>
<td>92</td>
<td>67.5±2.1</td>
<td>45</td>
<td>45</td>
<td>--</td>
</tr>
<tr>
<td>1980s</td>
<td>8</td>
<td>262</td>
<td>67.4±4.6</td>
<td>68</td>
<td>53</td>
<td>--</td>
</tr>
<tr>
<td>1990s</td>
<td>12</td>
<td>459</td>
<td>68.0±3.2</td>
<td>64</td>
<td>81</td>
<td>40</td>
</tr>
<tr>
<td>2000s</td>
<td>42</td>
<td>2,250</td>
<td>70.6±3.6</td>
<td>74</td>
<td>79</td>
<td>72</td>
</tr>
<tr>
<td>Since 2006</td>
<td>30</td>
<td>1,573</td>
<td>71.4±3.6</td>
<td>81</td>
<td>82</td>
<td>73</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Table 2: Mortality and morbidity of shunt insertion in INPH stratified by the decade of study publication and expressed as percentages of the total patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>Decade of publication</td>
</tr>
<tr>
<td>-----------------------</td>
</tr>
<tr>
<td>1970s</td>
</tr>
<tr>
<td>1980s</td>
</tr>
<tr>
<td>1990s</td>
</tr>
<tr>
<td>2000s</td>
</tr>
<tr>
<td>Since 2006</td>
</tr>
</tbody>
</table>

SDH subdural haemorrhage or effusion, ICH intracerebral haemorrhage

ETV v VPS for NPH?

One RCT of ETV v VPS

- San Paolo Brazil
- 42 patients with 12 months follow-up
- 50% improvement with ETV
- 77% improvement with VPS

However, mult-centre study in Italy showed 69% success with ETV (Gangemo et al., 2008)

Pinto et al. Neurosurgery 2013
ETV v VPS for NPH?
Alexander Monro *Primus* (1720-1758)  
*Professor of Anatomy, Edinburgh*

Alexander Monro *Secundus* (1754-1798)  
*Professor of Anatomy Edinburgh*

Alexander Monro *Tertius* (1798-1846)  
*Professor of Anatomy Edinburgh*

David Monro (1813-1877)  
*Speaker of New Zealand House of Representatives*

David Monro (1851-1933)  
*Introduced Rugby to New Zealand in 1870*
<table>
<thead>
<tr>
<th></th>
<th>Test Positive</th>
<th>Test Negative</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patient with disease</td>
<td>True Positive (TP)</td>
<td>False Negative (FN)</td>
</tr>
<tr>
<td>Normal patient</td>
<td>False Positive (FP)</td>
<td>True Negative (TN)</td>
</tr>
</tbody>
</table>

**Calculation**

- **Sensitivity**  
  \( \frac{TP}{TP+FN} \)
- **Specificity**  
  \( \frac{TN}{TN+FP} \)
- **Positive predictive value (PPV)**  
  \( \frac{TP}{TP+FP} \)
- **Negative predictive value (NPV)**  
  \( \frac{TN}{TN+FN} \)