Update on Brain Tumours

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University of Otago
Aims & Objectives

1. Take the opportunity to review the epidemiology, classification and clinical presentation of brain tumours
2. To understand the principles of surgical management
3. To be aware of some of the advances in the management of malignant gliomas
4. Review of advances in pituitary surgery

*To think like a neurosurgeon, for a short while....*
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.

The Monro-Kellie Doctrine

Alexander Monro
Primus (1720-1758)
Professor of Anatomy,
Edinburgh
Epidemiology

• Primary = 6:100,000 / yr
  – 1:12 in <15 yr olds
• Secondary = probably > 6:100,000 / yr
Site

Pathology

‘Benign’ vs ‘Malignant’

Definitions have different meanings when applied to the CNS
INTRACRANIAL TUMOURS

Classification

Site

Extrinsic
Meningioma
Acoustic neuroma
Pituitary adenoma
Other

Intrinsic

Type

Histology
Genetic

Grade 1           Grade 2
Grade 3
Grade 4

Benign
Malignant

1° - “Glioma”
2° - Metastasis
Clinical Features

1. Generalised

Headache  Commonest symptom of brain tumour

‘Typical’ = on waking, new (or change chronic HA), increased by coughing/movement, associated vomiting and/or other neurological symptoms

Confusion, memory loss, personality change
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2. Localizing

Seizure  Commonest localizing sign - occur in 1/3 of patients

‘Typical’ = focal

20% of new onset adult seizures have an underlying tumour

Commoner with low grade tumours

Usually tumour is adjacent to cortex - esp. motor strip

Focal neurology - weakness, aphasia, visual field loss etc. i.e. DISTURBED LOBE FUNCTION
Clinical Features

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3. False localizing

Associated with raised ICP: I, III, IV, VI, VIII abnormality, ataxia
Principles of Surgical Management

- Need to consider 3 factors in the risk:benefit analysis -
  1. Tumour - natural history (rate of growth, effects - anatomical location)
  2. Procedure - morbidity/mortality of procedure
  3. Patient - life expectancy

- Full resection may not be feasible

- Management > cure is the aim

- Palliative procedures called for in 2 circumstances -
  1. Symptomatic \( \uparrow \) ICP
  2. Reversible local brain dysfunction
     - Vision - chiasmal decompression
     - Hemiplegia - may improve after cystic decompression, but can be aggravated if vascular infiltrative tumour
     - Seizures - may undergo temporary or permanent remission
INTRACRANIAL TUMOURS

Period of silent growth

• Epilepsy
• Focal neurological symptoms & signs

Symptoms & signs of ↑ ICP

Brain displacement, coning & death

Pressure/Volume curve

Neurological status

ICP

Time

Lesion volume
Disturbed Function

• Frontal lobe
  – Contralateral face, arm, leg weakness
  – Expressive dysphasia
  – Personality change
    • Loss of inhibition
    • Dementia
    • Abulia
....disturbed function

- **Occipital**
  - Homonymous Hemianopia
- **Corpus Callosum**
  - Disconnection syn.
  - Apraxia
- **Temporal**
  - Receptive dysphasia
  - Upper homonymous hemianopia
- **Parietal**
  - Sensory disturbance
  - Acalculia, agraphia
  - Finger agnosia
  - Sensory neglect
...disturbed function

- Hypothalamus/Pituitary
  - Endocrine disturbance
  - Bitemporal hemianopia

- Infratentorial
  - Midbrain/Brainstem
    - CN 3-12
    - Long tract signs
    - Tremor
    - Dec. GCS
    - Eye movements
    - Pupillary abnormality
    - Vomiting, hiccough
Cerebellum

- Ataxia
- Intention tremor
- Dysmetria
- Dysarthria
- Nystagmus
LOCALISING SIGNS - Visual pathways
ANATOMY: Visual pathways – optic radiation

(L) side

Meyer’s loop

(R) homonymous hemianopia - incomplete in lower quadrants, incongruous & less marked in eye on side of lesion

Intrinsic lesions - (L) posterior temporal glioblastoma multiforme
False Localising Sign - VI (Abducens)

Longest intracranial course?

• Exits the pons at its lower border just above the pyramid of the medulla. Passes in SAS of pontine basal cistern and ascends over the front of the BS before angling sharply over the petrous bone. Passes under the petroclinoid ligament, or Gruber’s ligament (Dorello’s canal) to enter the back of the cavernous sinus.

• It passes freely in the cavernous sinus lateral to the ICA and enters the orbit via the SOF through the annulus of Zinn.

• Terminates in the medial aspect of LR.

*Fig 8. Posterior view of the petro-clival region. The yellow arrow indicates the Gruber’s ligament. The blue arrow shows the trigeminal nerve and the red arrow points at the abducens nerve leading itself through the Dorello’s channel to the cavernous sinus.*
CT Scan

• Mass Effect
  – Midline shift
  – Hydrocephalus
• Site
• Size
• Hyperostosis
• Single or Multiple
• Contrast enhancement
Principles of Surgical Management

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• Commonest 1° brain tumours = gliomas

• Commonest brain tumour = metastases
Gliomas

- 2/3 of all primary brain tumours

- Arise from 3 basic types of glial cell -
  1. Astrocytes: have endfeet on walls of capillaries, thought to be involved in chemical exchange between blood and neural tissue, probably constitute the BBB
  2. Oligodendrocytes: form myelin sheath
  3. Ependymal cells: involved in repair of nervous tissue, form a lining in ventricles

- Gliomas share common characteristics -
  - Indistinct margins - complete resection almost impossible
  - Incurability
  - Genetic instability
  - Increased malignancy over time
  - Tendency for local recurrence
Gliomas

WHO Classification

**Astrocytomas***
- Glioblastoma multiforme
- Anaplastic astrocytoma
- Astrocytoma
- Pilocytic astrocytoma

**Oligodendrogliomas**
- Anaplastic oligodendroglioma
- Oligodendroglioma

**Mixed gliomas**
- Anaplastic oligoastrocytomas
- Oligoastrocytoma

**Ependymomas**
- Anaplastic ependymomas
- Ependymomas
- Myxopapillary ependymoma

*Commonest form*
## Astrocytomas

- **2 grading systems:** Kernohan & WHO

<table>
<thead>
<tr>
<th>Kernohan</th>
<th>WHO</th>
<th>Type</th>
<th>Median Survival*</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>I</td>
<td>Special tumours (e.g. pilocytic astrocytomas)</td>
<td>10 years</td>
</tr>
<tr>
<td>I/II</td>
<td>II</td>
<td>Astrocytoma (low-grade)</td>
<td>8 years</td>
</tr>
<tr>
<td>III</td>
<td>III</td>
<td>Anaplastic astrocytoma (AA)</td>
<td>3 years</td>
</tr>
<tr>
<td>IV</td>
<td>IV</td>
<td>Glioblastoma multiforme (GBM)</td>
<td>&lt;1 year</td>
</tr>
</tbody>
</table>

- Relative incidence? IV:III:II (WHO system) = 5:3:2
- Peak age incidence increases with grade
  - II  34 years
  - III 41 years
  - IV  53 years

- III and IV also known as malignant astrocytomas
Malignant Astrocytomas

- WHO III & IV
- Anaplastic astrocytoma (AA) & Glioblastoma multiforme (GBM)

**GBM**
- Commonest primary brain tumour
- Most aggressive brain tumour
- 50% of all primary brain tumours
- 20% of all brain tumours
- M:F ratio 1.5:1
- Ages 45-65
- Median survival - 14 weeks (without Rx), 9-12 months (surgery & DXT)
- Ring-enhance on CT due to necrosis

**AA**
- 20% of all gliomas
- M:F ratio 1.2:1
- Median survival 24-36 months with Rx
- Ages 35-55
- Complex enhancement on CT
Malignant Astrocytomas

Prognostic indicators in GBMs and AAs

1) **Patient age** - the most significant prognostic indicator

   E.g. GBMs (Wen *et al*. 1995)

<table>
<thead>
<tr>
<th>Age</th>
<th>18 months survival</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;40</td>
<td>50%</td>
</tr>
<tr>
<td>40-60</td>
<td>20%</td>
</tr>
<tr>
<td>&gt;60</td>
<td>10%</td>
</tr>
</tbody>
</table>

2) **Histological features** - AA survive 3 x longer than GBM

3) **Performance status** - Karnofsky score (KPS)

   E.g. GBM

<table>
<thead>
<tr>
<th>KPS</th>
<th>18 m survival</th>
<th>5 yr survival</th>
</tr>
</thead>
<tbody>
<tr>
<td>≥ 70</td>
<td>34%</td>
<td>7.6%</td>
</tr>
<tr>
<td>&lt; 60</td>
<td>13%</td>
<td>3.2%</td>
</tr>
</tbody>
</table>
Low-grade Astrocytomomas

WHO II

- 10% of all gliomas
- 30-50 years
- Most present with seizures
- Median survival 8 years
- Typical MRI features
  - MRI: non-enhancing
    - hypodense on T1 + contrast
    - no/minimal oedema on T2
Treatment of Astrocytomas

Malignant astrocytomas

- Gold standard = cytoreductive surgery + DXT* (*DXT 40 Gy to whole brain + 20 Gy to tumour bed)

- Controversy over extent of tumour resection
  
  Reduce mass effect or get as much out as possible?

  Tumours **NOT** cured with surgery :: aim for **QUALITY** survival

- Role of chemotherapy? Not proven for all patients, but may be beneficial for some

Low-grade astrocytomas

- Various options, none shown to be superior to another -
  
  1) Observe - serial imaging
  2) Surgery
  3) Chemo +/- DXT

- Surgery may be indicated if -
  
  1) Large tumours, young patient, short clinical history
  2) Radiological progression
  3) Clinical grounds - seizures, CSF flow obstruction etc.

Pilocytic astrocytomas

- Surgery
1st Neurosurgical Operation for Intracranial Lesion

Cerebral localisation by “functional neurological mapping”

Pre-XRAY Localisation on basis of focal neurological signs

Sir William MacEwan
Glasgow 1876
Frontal lobe abscess
Patient survived
1st Operation for a Primary Brain Tumour

• Performed by Rickman Godlee (nephew of Sir Joseph Lister) in London, 1884
• Operation observed by Hughlings Jackson, David Ferrier and Victor Horsley....
• Was the surgery a success?
1st Operation for a Primary Brain Tumour

- Performed by Rickman Godlee (nephew of Sir Joseph Lister) in London, 1884
- Operation observed by Hughlings Jackson, David Ferrier and Victor Horsley....
- Was the surgery a success?
- The patient died of infection
- But for Godlee it was very successful
  - Baronet 1912
  - Hunterian Orator 1913
  - President RCS 1914
  - Surgeon to Victoria, Edward II, George V
Current Gold Standard for GBM

- Maximal safe resection
- Stupp protocol = Concomitant temozolomide during radiation treatment
Advances in Neurosurgery over Last 100 Years

- CT and MRI Scanning
- Microscope (Yasargil)
- Endoscope (Dandy, Cappabianca)
- Image Guidance (E.g. Stealth) – but brain shift
- 3D Imaging
So What is New for GBM?

Molecular characterisation of GBMs

Adjuncts for surgical resection
Molecular Classification of GBM

GBM one of the most molecularly characterised of all human cancers
Molecular profiling has identified molecular prognostic factors and potential therapeutic targets

Weathers & Giblert. 2014
Antiangiogenic Therapy: BEVACIZUMAB (Avastin)

- Angiogenesis a pathologic hallmark of GBM
- Vascular Endothelial Growth Factor (VEGF) is one of the most important regulators of angiogenesis
- VEGF antagonism by humanised mono-clonal antibody was promising
- Early results with BEVACIZUMAB (Avastin) suggested prolongation of progression free survival - but radiology may have been misleading
- Effect of QoL?

Weathers & Giblert. 2014
Extent of Resection (EOR) for GBM

“Total Resection” appears to be associated with better survival than “Sub-Total Resection”

However, likely selections bias towards younger, fitter patients with tumours in non-eloquent regions for more aggressive surgery

Table 1: Grading of intraparenchymal tumors according to functional location

<table>
<thead>
<tr>
<th>Grade</th>
<th>Functional Location</th>
</tr>
</thead>
<tbody>
<tr>
<td>I: noneloquent brain</td>
<td>frontal or temporal pole of cerebrum rt parietooccipital lobe cerebellar hemisphere</td>
</tr>
<tr>
<td>II: near eloquent brain</td>
<td>near motor or sensory cortex† near calcarine fissure near speech center corpus callosum near dentate nucleus near brainstem</td>
</tr>
<tr>
<td>III: eloquent brain</td>
<td>motor or sensory cortex visual center speech center internal capsule basal ganglia hypothalamus or thalamus brainstem dentate nucleus</td>
</tr>
</tbody>
</table>

* Adapted from Sawaya, et al.
† Includes tumors in the supplementary motor area.
## Extent of Resection (EOR) for GBM

<table>
<thead>
<tr>
<th></th>
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<th></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Design</strong></td>
<td>Retrospective series</td>
<td>Retrospective series</td>
<td>RCT</td>
<td>RCT</td>
</tr>
<tr>
<td><strong>Class of evidence</strong></td>
<td>II</td>
<td>II</td>
<td>I</td>
<td>II*</td>
</tr>
<tr>
<td><strong>Randomization</strong></td>
<td>None</td>
<td>None</td>
<td>Biopsy versus resection</td>
<td>Fluorescence-assisted surgery versus conventional surgery</td>
</tr>
<tr>
<td><strong>Number of patients</strong></td>
<td>92</td>
<td>416</td>
<td>30</td>
<td>322 (270 in analysis)</td>
</tr>
<tr>
<td><strong>Follow-up</strong></td>
<td>Lifespan of patients</td>
<td>Lifespan of patients</td>
<td>Lifespan of patients</td>
<td>6 months</td>
</tr>
<tr>
<td><strong>Primary endpoints</strong></td>
<td>Time to progression (TTP)</td>
<td>Survival</td>
<td>Survival</td>
<td>Progression free survival (PFS) Degree of resection</td>
</tr>
<tr>
<td><strong>Secondary endpoints</strong></td>
<td>None</td>
<td>None</td>
<td>Clinical status KPS</td>
<td>Volume of residual tumour Overall survival Neurological deficit Toxicity</td>
</tr>
<tr>
<td><strong>Number of centres</strong></td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>17</td>
</tr>
</tbody>
</table>

* Stummer et al.’s paper may be considered class I evidence for the role of 5-ALA in patients with high-grade gliomas. However, it should only be considered class II evidence for the question under consideration in this chapter, as maximal safe resection was attempted in both treatment arms.
Extent of Resection (EOR) for GBM

### Results

**Keles et al. (1999)**
- EOR also showed a significant correlations with post-operative KPS ($p < 0.05$).

<table>
<thead>
<tr>
<th>EOR</th>
<th>&lt;25%</th>
<th>25–49%</th>
<th>50–74%</th>
<th>75–99%</th>
<th>100%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Median TTP (weeks)</td>
<td>14.1</td>
<td>24</td>
<td>31.9</td>
<td>45.8</td>
<td>53.1</td>
</tr>
<tr>
<td>Median survival (weeks)</td>
<td>31.8</td>
<td>56.6</td>
<td>62.9</td>
<td>88.5</td>
<td>93</td>
</tr>
</tbody>
</table>

**Lacroix et al. (2001)**

<table>
<thead>
<tr>
<th></th>
<th>&gt;98% resection</th>
<th>&lt;98% resection</th>
<th>Statistical significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Median survival (months)</td>
<td>13</td>
<td>8.8</td>
<td>$p &lt; 0.001$</td>
</tr>
<tr>
<td>PFS (6 month)</td>
<td>41%</td>
<td>21%</td>
<td>$p = 0.0003$</td>
</tr>
</tbody>
</table>

**Vuorinen et al. (2003)**

<table>
<thead>
<tr>
<th></th>
<th>Craniotomy and resection</th>
<th>Biopsy</th>
<th>Statistical significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Median survival (days)</td>
<td>171</td>
<td>85</td>
<td>$p = 0.03$</td>
</tr>
</tbody>
</table>

**Stummer et al. (2006)**

<table>
<thead>
<tr>
<th></th>
<th>Fluorescence-assisted surgery</th>
<th>Conventional surgery</th>
<th>Statistical significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Percentage of complete resection</td>
<td>65%</td>
<td>36%</td>
<td>$p &lt; 0.0001$</td>
</tr>
<tr>
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<td>21%</td>
<td>$p = 0.0003$</td>
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Conclusions

Keles et al. (1999)
The extent of tumour resection in glioblastoma multiforme affects overall survival and time to progression.

Lacroix et al. (2001)
Greater than 98% tumour debulking is associated with improved survival.

Vuorinen et al. (2003)
Craniotomy and debulking offers a modest survival advantage over biopsy in elderly patients with GBM.

Stummer et al. (2006)
Fluorescence-assisted surgery with 5-ALA enables more complete resection of malignant glioma with improved PFS.
Strategies to Maximise Extent of Resection

Need to augment surgical resection whilst minimising risk to eloquent brain

- Cortical and subcortical mapping
- Functional neuronavigation
- Intrapoperative MRI
- Fluorescence-guidance
Cortical and Subcortical Mapping

Intraoperative functional mapping of eloquent brain includes:

- **Somatosensory-evoked potentials (SSEPs)** to identify the central sulcus
- **Awake stimulation for language function**
- **Subcortical and cortical stimulation mapping of motor pathways**

Meta-analysis suggests that such strategies are effective

![Meta-Analysis of Glioma Surgery Outcome](image)
Functional MRI (fMRI) and diffusion tensor imaging (DTI) provide a means of identifying eloquent cortical areas and the course of subcortical fibre tracts displaced by the lesion.

One prospective randomised trial revealed reduced post-op motor deficits (15.3% v 32.8%, p < 0.001).

Wu et al. 2007
Intraoperative MRI (ioMRI)

One RCT suggested benefit – most likely due to correcting brain shift errors of intraoperative neuronavigation (Senft et al. 2011)

Meta-analysis shows level II evidence for effect

Likely that ioMRI will become more widespread

Kubben et al. 2011
ACOUSTIC NEUROMA/VESTIBULAR SCHWANNOMA
Pituitary Surgery
The “combined eye” of surgeon and artist: Evaluation of the artists who illustrated for Cushing, Dandy and Cairns

Reuben D. Johnson a,*, Willow Sainsbury b

a Department of Neurosurgery, Royal Melbourne Hospital, Grattan Street, Parkville, Victoria 3050, Australia
b Institute of Social and Cultural Anthropology, Oxford, UK
que from Cushing abandoning the approach, talk re the cross-atlantic stories
Galen 150 AD
“The Phlegm Gland”
Disposal of waste from ventricles to nose

Plate from Seventh Book of the Anatomic Fabrica of Vesalius in 1543

The oldest image in Western literature of the hypothalamic pituitary unit

A = pituitary gland
B = infundibulum
C-F = ducts ‘draining the phlegm’
Professor Pierre Marie
Paris 19th Century

Described acromegaly and its association with pituitary tumours in 1886

Antonii De Haen
Vienna 18th Century

Described the amenorrhoea associated with a pituitary tumour
Rathke
Germany 19th Century

Rathke’s cleft cyst

Craniopharyngioma
“The Master Gland”
What Pituitary Pathology Can Be Treated Surgically?
Symptomatic SOL: Endocrine or Neurological Compromise

- Macroadenoma
- Meningioma
- Rathke’s
- Craniopharyngioma
Hormone Secreting Lesions & Bleeds

- Prolactinoma
- Acromegaly
- Cushing’s
- Apoplexy
How Do We Get to the Pituitary Surgically?
Trans-Cranial Route to Sella

1st attempt at pituitary tumour resections in 1889

Subtemporal decompression – not always attempting to remove tumour

Treated 10 patients (mortality 20%)

Not reported until 1906

Sir Victor Horsley
(1857-1916)
London, UK
Trans-Cranial Route to Sella

First frontal transcranial approach to sella turcica in 1904

But Mortality >50%

F Krause
(1857-1937)
Berlin, Germany
“They first take a crooked piece of iron, and with it draw out the brain through the nostrils getting rid of a portion, while the skull is cleared of the rest by rinsing with drugs” Herodotus
“Until now, no such operation has yet been carried out on a living patient, at least none has been reported, obviously, because firstly the decision to perform such a difficult intervention bears the mark of a foolhardy novice, and is difficult even for the expert; secondly, because the function of the hypophysis remains obscure and hence the consequences of extirpation of the pituitary cannot be foreseen.”

Herman Schoffler, Insbruck 1906

Widely travelled - including visiting William Macewan in Glasgow
Herman Schoffler - Innsbruck 1907

First transnasal trans-sphenoidal approach to sella turcica

Patient - 30 y/o male
- 6/12 headaches and progressive visual loss
- Hypogonadism
- Skull radiograph showed an enlarged sella
Attempted removal of tumour with a blunt spatula

Patient died at week 10 from hydrocephalus

“the selection of the right cases for surgery is of primary importance for further development of the surgical technique”

Schoffler, 1907

Schoffler responsible for bringing the trans-sphenoidal route into the mainstream
Hirsch & Cushing – The Beginning and the End of the TSA?

Oskar Hirsch (Vienna)

Introduced endonasal procedure

4th June 1910

Cushing (Baltimore)

Modified Schoffler’s approach

4th June 1910
Cushing did 231 procedures with 5.6% mortality and then abandoned it in favour of subfrontal approach (mortality 4.5% in his hands)
Hirsch “The Obscure Voice in the Wilderness”

By 1937 (pre-antibiotic era) 277 patients with 5.4% mortality

By 1956 (post-antibiotic era) 413 patients with 1.5% mortality

He emigrated to Boston in 1938 after expulsion from Austria by Nazis

Never recognised or allowed to operate independently
The Resurrection of the TSA

Trained under Cushing in 1920

Continued TSA with >100 patients

0% mortality (antibiotics and perioperative steroids)

Low recurrence (DXT)

But never published results out of deference to Cushing

Norman Dott (Edinburgh)
The Resurrection of the TSA

Trained under Dott

Published his results with 1000 patients

Introduced X-ray II and fluoroscopy to guide surgery

“In the evolution of surgical techniques the introduction of the transcranial approach was progress, however then abandoning the transnasal approach, as a matter of fact, has been a step backwards”
The Resurrection of the TSA

Trained under Guiot

Re-introduced the TSA to North America

Introduced the microscope to the TSA

Hardy (Toronto)
Endoscopic Trans-Sphenoidal Approach (ETSA)

Cappabianca (Naples, Italy)

Kassam (Pittsburgh, USA)

Hae-Dong Jho (Pittsburgh, USA)
Sphenoid Ostium

FIGURE 2. Endoscopic view of the sphenoid ostium, reached with the simple introduction of the endoscope into the nasal cavity (right nostril). It provides entrance to a very wide surgical target area. SO, sphenoid ostium; ST, superior turbinate.
Fig. 1. Anatomical photograph showing the landmarks on the posterior wall of the sphenoid sinus. The asterisk indicates the tuberculum sellae. C = clivus; ICA = internal carotid artery; OCR = opticocarotid recess; ON = optic nerve; S = sella; SP = sphenoid planum.
Extended Approaches

Fig. 1 Schematic depiction of the sagittal plane module on a computed-tomography (CT) scan of the skull base. The various endonasal surgical approaches are numbered as follows: (1) transfrontal (2) transcribriform, (3) transplanum, (4) transsphenoid, (5) transclival and (6) transodontoid.
Extended Approaches

Fig. 5 Sagittal T1 MRI scans comparing the extent of bone removal involved in a conventional transsphenoidal approach (4) and extended transsphenoidal approach to a craniopharyngioma. The thick white lines indicate the site of bone removal, and thinner white lines indicate the extent of the exposure gained.
Cribriform Approach

- Extend the above further forward by taking septum from skull base (olfaction usually gone due to pathology)
- Drill out cribirform plate and crista gallae and cauterise ethmoidals as you go – will help devascularise lesion

Fig. 4. Transcribiform approach. A and B: Preoperative axial (A) and coronal (B) T1-weighted, contrast-enhanced MR images of an olfactory groove meningioma. C and D: Postoperative axial (C) and coronal (D) T1-weighted, contrast-enhanced MR images demonstrating complete resection of the lesion. The arrow in D indicates the skull base reconstruction using a vascularized nasoseptal flap. E: Intraoperative photograph of a transcribiform view during sharp dissection of the tumor (meningioma). The interface between the meningioma and the left frontal lobe is visible. The frontopolar artery (art) is shown over the left gyrus rectus.
Fig. 5. Transclival approach.  

A and B: Preoperative axial (A) and sagittal (B) contrast-enhanced, T1-weighted MR images showing a neurenteric cyst. Note the arrows indicating the neurenteric cyst delineated by the yellow dotted lines.

C and D: Postoperative axial (C) and sagittal (D) contrast-enhanced, T1-weighted MR images demonstrating complete resection of the lesion.

E: Intraoperative oblique, 45° endoscopic view of the preopticine cistern showing the brainstem at the level of the pons, CN V (V), CN VI (VI), and the complex of CNs VII and VIII (VII and VIII).
Kassam – Endoscopic Odontoid Surgery

**FIGURE 1.** Schematic drawing of the expanded endonasal approach to the craniocervical junction. Note that the transnasal exposure provides direct route to the odontoid along a rostral to caudal trajectory and do not require manipulation of the oropharynx or palate.

**Fig. 6.** Transodontoid approach. A and B: Preoperative axial (A) and sagittal (B) CT angiograms showing a foramen magnum meningioma. C and D: Postoperative axial (C) and sagittal (D) CT angiograms demonstrating resection of the bone and lesion. E: Intraoperative view of the cervicomedullary junction during resection of a foramen magnum meningioma (Tu). The anterior spinal artery (ASa) is seen ventral to the spinal cord. The inferior rootlet of the hypoglossal nerve is seen on the left side (XII). Note the C-1 ventral root (C1) and the denticulate ligament (DL). Bilateral VAs are identified (RVa and LVa).
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