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| Neurosurgery Resource 2016 |
| Neurosurgery |
| *Resource for Staff working in the ED Department* |

**Associate Professor Michael A Murphy – Honorary Neurosurgeon**

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| ***Contact Details:***  Between 0700 – 2200 hours, Weekdays and 0830 – 2000 Weekends for all Neurosurgical Conditions contact A/Prof Michael Murphy, Director of Neurosurgery, St. Vincent’s Hospital on his mobile phone 0412 981727. This includes clinical advice and patients for transfer. If the phone is not answered during this time and the situation is not time critical then leave a message and he will ring back.  If outside these hours or time critical contact the St Vincent’s Neurosurgical registrar on call via SVH switchboard (9231 2211). If patient is to be transferred then inform the registrar the patient is to come under Michael Murphy’s care. If he is on leave the BBH ED Department will be notified and in this case contact the registrar directly.  Where indicated, patients will be transferred, monitored for a period of 24 – 48 hours and if operation is not required then the patient should be transferred back to a Medical In-patient Unit of the BbH. |
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BRAIN

## BRAIN TUMOURS

### Malignant

Introduction

Malignant brain tumours, primary or secondary, will not uncommonly present to the ED. Physicians should be aware of the importance of the history taking here

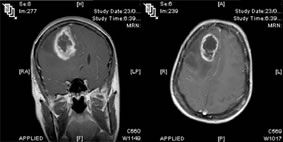
* Known history of cancer should raise possibility cerebral metastases, as wellas factors such as heavy smokers
* First presentation off a seizure in middle age -elderly person; less common in younger person

Clinical

* Signs of raised intracranial pressure
* Focal neurological symptoms or signs
* Epilepsy
* Confusion

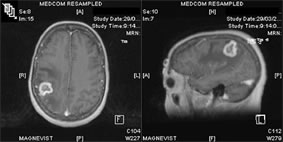
Investigations

* CT-Scan: Single or multiple lesion(s) with surrounding oedema
* MRI: potentially help with diagnosis of primary vs secondary
* Suggestive of unknown secondary: CT-scan: chest/abdomen/pelvis

**

*Left – Coronal MRI of Glioblastoma Multiforme*

*Right – Sagittal MRI of Glioblastoma Multiforme*



*Left – Axial MRI of breast metastasis*

*Right – Sagittal MRI of breast metastasis*

Management

* Contact Neurosurgical Unit
* Arrange transfer after patient stable (ie seizures -> anticonvulsants, raised ICP -> consider mannitol)
* Discuss with referring unit as to whether or to perform extra imaging locally or at tertiary institution.

Further Management

* Diagnosis Surgery
  + If suspected metatase then possible biopsy of extracranial lesion if appropriate. Cerebral metastases either surgery, radiosurgery or both.
  + Primary lesion: debulking if appropriate
  + Biopsy if deep lesion or elderly patient
* Obstructive hydrocephalus may require drain or shunt
* Adjuvant therapy
  + Radiotherapy
  + Chemotherapy
* **Primary tumours**
  + Glioblastoma
  + Anaplastic astrocytoma
  + Astrocytomas
  + Pineal tumors
  + Ependymomas
* **Secondary Tumours**
  + Breast
  + Lung
  + Colon
  + Renal
  + Melanoma

### Benign

Commonest types are:

* Meningiomas (2% malignant)
* Pituitary Tumours
* Acoustic Neuroma

Clinical

* Meningiomas
  + Signs of raised intracranial pressure
  + Focal neurological symptoms or signs
  + Epilepsy
  + Confusion
* Pituitary Tumours
  + Secreting
    - Depend on hormone been secreted. Commonest are prolactomas. Usually microadenomas (< 1 cm)
  + Non secreting
    - Presentation usually visual disturbance, typically homonymous hemianopia. Also symptoms due to reduced hormonal levels. Usually macroadenomas (> 1 cm)
* Acoustic Neuroma
  + Arise from 8th cranial nerves
  + Deafness
  + Tinnitus

Investigations

* CT-Scan: Identify tumour
* MRI: More definition of the tumour, where it is arising from, and whether there is more than one (multiple meningiomas). Also the presence of oedema and/or midline shift.



*Sagittal MRI of meningioma*

Management

* Contact Neurosurgical Unit
* Arrange transfer after patient stable (if indicated).
* Discuss with referring unit as to whether or to perform extra imaging (ie; MRI, if not already done) to be performed locally or at tertiary institution.

Further Management

* Conservative vs Surgery
  + Depends on many factors such as patient age, symptoms and whether tumour likely to enlarge or not.
  + Surgery: Biopsy or Excision
  + Other therapies such as stereotactic radiotherapy if indicated.
  + Also Monitoring

## HAEMORRHAGES

* Subarachnoid Haemorrhage
* Intracerebral Haemorrhage

### Subarachnoid Haemorrhage

Introduction

* Trauma is the commonest cause of subarachnoid haemorrhage
* Spontaneous subarachnoid haemorrhage
  + 75-85% of spontaneous subarachnoid haemorrhages are caused by intracranial aneurysms.
  + Other causes include vascular malformations, tumours and coagulation disorders.
  + One third of patients will die from a subarachnoid haemorrhage, one third will be disabled and another one third will return to their previous occupation.
  + The peak age for a subarachnoid haemorhage is approximately 60 years of age.
  + Increased incidence of cerebral aneuryms in patients that smoke, have a strong family history, polycystic kidneys and Ehlers-Danos syndrome, marfan syndrome and coarctation of the aorta, fibromuscular dysplasia and arteriovenous malformations (AVM).

Clinical

* Patients present with a sudden onset of a severe headache often in the occipital region. The headache is most severe initially often described as the worst headache of their life.
* Approximately 30% of patients will have a sentinel or warning headache in the weeks prior to the actual haemorrhage
* Approximately one third occur during sleeping, one third during activities, and one third when the person is doing normal activities.
* May be associated with nausea, vomiting, neck pain and loss of consciousness.
* Focal neurological signs may be present, the commonest would be a third nerve palsy but a hemiparesis may occur if there is an associated intracerebral component.

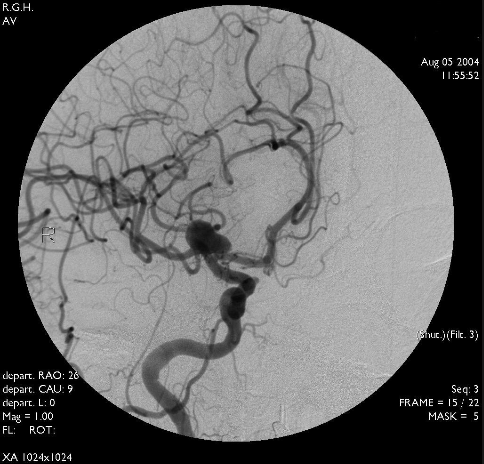
Investigations

* A CT scan and a CTA should be the initial form of investigation
  + Key features include;
    - The amount of blood seen
    - Distribution of blood
    - Presence of intracerebral haematoma
    - Presence of an aneurysm
    - Presence of multiple aneurysms (15% of patients will have more than one)
    - Location and Size



*CT Scan SAH*

* An MRA is usually performed if the CTA is negative for the presence of an aneurysm. It is often used for screening patients to exclude an unruptured aneurysm, as well as monitoring known aneurysms and aneurysms that have been treated endovascularly
* Cerebral Angiogram.
  + Four Vessel Cerebral Angiogram is the definitive study in the diagnosis of cerebral aneurysm. It can be used both as a diagnostic procedure and also therapeutic if the aneurysm is suitable for endovascular treatment.
  + May show other causes of subarachnoid haemorrhage including other vascular malformations such as AVMs and fistulas.



Giant right middle cerebral aneurysm

* Lumbar Punctures
  + Lumbar punctures are rarely now performed for the diagnosis of subarachnoid haemorrhage. Previously if there was no clear evidence of blood uniformally distributed over 3 tubes then the laboratory would look for xanthochromia. This is no longer the case in many laboratories.

Management

* Transfer to Neurosurgical unit after consultation
* Endovascular treatment (if the aneurysm is suitable for endovascular treatment then this is usually the first choice)
* Surgery. Surgery is reserved for aneurysms that cannot be treated endovasculary, aneurysms that have an intracerebral hematoma and also in patients who at the time of presentation have hydrocephalus and require an insertion of a ventricular drain.
* Initial medical imaging is negative. A repeat angiogram is often performed after a few days if nothing is seen on the initial one and this may be repeated a few weeks later. The commonest hemorrhages to have a negative angiogram are those where there is perimesencephalic blood.
* Complication
  + Re-bleeding
    - The greatest incidence of re-bleeding is in the first 24hours thereafter the rate of re-bleeding falls away, although there is a 50% chance in the first 6months of a re-bleed if the aneurysm is untreated.
  + Hydrocephalus
    - Frequently occurs due to obstruction of CSF pathways.
    - Patients may require temporary CSF diversion in the form of an external ventricular drain or lumbar drain until the blood has been reabsorbed.
    - In a small number of cases patients will require a permanent ventriculoperitoneal shunt.
  + Vasospasm
    - Vasospasm occurs secondary to vasoconstriction of the cerebral blood vessels due to factors associated with the hemorrhage.
    - The greater the amount of blood present on the CT scan the greater the likelihood of vasospasm.
    - Clinical vasospasm occurs in 20-30% of patients
    - Radiological vasospasm occurs in twice the number of patients
    - Maximum vasospasm occurs between days 7 -10
  + Presentation
  + New or increasing headaches
  + Altered conscious state
  + Focal neurological signs
  + Treatment
* All patients on presentation of subarachnoid haemorrhages are treated with Nimodipine which is a vasodilator and associated with a decreased incidence of vasospasm
  + - * Hyperdynamic therapy, including hypervolaemia and hypertension (may require inotrope support)
  + Cerebral angiogram
    - * Injection of intra-arterial drugs to dilate the vessels
      * Mechanical dilatation of vessels (angioplasty)
* Key Points;
  + Sudden onset of headache, worse at the time of initial onset in patients suffering from migraines or classical headaches. This headache is usual different to their normal one.
  + 30% have a warning bleed and if the patient presents with this although there may be no blood on the CT scan, a CTA at the very least should be performed.
  + Delayed presentation may occur in the form of hydrocephalus or vasospasm if CT scan does not show blood at the very least a CTA should be performed.

Asymptomatic Aneurysms

* Asymptomatic aneurysms are being more commonly diagnosed due to improved imaging and screening.
* Aneurysms < 7cm particularly in the elderly have a low incidence of bleeding.
* All aneurysms should be referred to a Neurosurgeon or Neurosurgical unit for assessment and advise and treatment options.
* Rarely asymptomatic aneurysms will present with epilepsy or mass effect due to size.

### Intracerebral Haemorrhages

Introduction

Parenchymal haemorrhages can be due to a number of causes. Younger patients are more likely to have an underlying anatomical abnormality. This also applies to haemorrhage location with lobar haemorrhages more likely to be associated with a structural abnormality than deep hemorrhages.

Causes of lobar hemorrhages

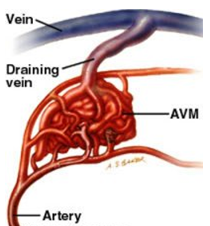
* Extension of a deep hemorrhage
  + Amyloid angiopathy
  + Arterio-venous malformations (AVM)
  + Haemorrhagic transformation of an ischemic infarct

Causes of deep haemorrhages

* + Hypertension
  + AVM
  + Tumours

Causes of Posterior fossa hemorrhages

* Hypertension
* AVMs
* Tumours

 *AVM*

Clinical

* Symptoms of raised intracranial pressure
* Focal neurological signs
* Rarely epilepsy

Investigations

* CT scan of the brain will identify the hemorrhage but a CTA should be performed to exclude an underlying vascular lesion



*CT Scan showing large ICH*

* MRI & MRA.
  + In lobar hemorrhages an MRI better identifies structural abnormalities
  + In amyloid angiopathy there is often evidence of an MRI of previous hemorrhages
  + In MRI where there is evidence of contrast enhancement then an underlying lesion should be suspected and if surgery is not performed on that admission the MRI should be repeated 4-6weeks later when there should be significant resolution of the blood.

Management

* Surgery
  + Depend on location, age of patient, neurological signs and symptoms
  + Deep hypertensive hemorrhages are not usually surgically drained unless extending to the surface. These patients usually have hydrocephalus due to intraventricular blood not uncommonly have an external ventricular drain inserted.
  + More superficial hemorrhages causing mass affect are often drained
  + With the exception of a cerebral aneurysm, most underlying structural abnormalities do not need to be dealt with in the acute setting.

## HAEMATOMAS

### Extradural Haematoma

Introduction

Extradural Hematomas arise in the layer between the inner table of the skull and dura. They are usually seen in younger people as the dura becomes more adherent to the inner table as a person gets older and more difficult therefore to be stripped away. They are traumatic in origin and classically have a ‘lucid interval’.

Causes

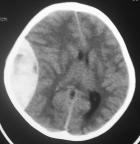
* Over 90% of patients will have a fracture in the temporal region through the middle meningeal vessels.

Clinical

* Brief loss of consciousness following an episode of trauma
* Classic lucid interval for several hours
* Patient then becomes obtundant contralateral hemiparesis and ipsilateral pupil dilatation
* May also present with headache, vomiting and seizures

Investigations

* CT scans
  + Over 85% or cases you have high density biconvex shape adjacent to the skull
  + Bone windows may reveal a fracture through the squamous temporal bone



*CT Head demonstrating large extradural haematoma*

Management

* Transfer to Neurosurgical unit after consultation
* If asymptomatic and or midline shift, then craniotomy and evacuation of hematoma
* If asymptomatic and no midline shift, may consider monitoring

### Subdural Hematomas

Introduction

Subdural hematomas can be divided into acute, sub-acute and chronic.

Classifications are based on the age of the haematoma in relation to its density to the brain parenchyma or CSF.

* Acute
  + 1-3 days
  + Hyperdense on CT scan
* Sub-Acute
  + 4 days to 3 weeks
  + Isodense on CT scan
* Chronic
  + Greater than 3 weeks
  + Hypodense on CT scan

There is also acute on chronic subdural hematomas where there has been ‘fresh bleeding’ into an existing chronic subdural hematoma.

### Acute Subdural Hematomas

* Acute subdural hematomas are usually a result of an acceleration and deceleration injury during a violent head motion where there is tearing of a bridging vein between the dura and brain. Underlying prior brain damage may be present and manifest itself as cerebral oedema with midline shift
* May also arise from an accumulation of a parachymal laceration of the front or temporal lobe. In these cases there is usual more severe underlying brain injury and more significant midline changes.
* May also occur in patients on anti-coagulation medication even with a history of minor trauma.

Investigations

* CT scans
  + Hematoma
  + Effacement of ipsilateral ventricle
  + Midline shift



*CT scan demonstrating acute subdural haematoma.*

Clinical

* Signs of raised intracranial pressure
* Focal neurological signs
* Seizures
* Loss of consciousness

Management

* Discussion with Neurosurgical unit and transfer if indicated (age, GCS, size of hematoma)
* In making a decision as to whether or not the patient should be transferred to a tertiary hospital the age of the patient, their independence, GCS and the size of the hematoma all need to be considered.
* Small subdural hematomas less than 1cm may be monitored with repeat imaging. If enlargement occurs then evacuation is indicated.
* Greater than 1 cm, focal neurological signs and/or midline shift should be evacuated.

Surgery

* With focal neurological signs, insertion of intracranial pressure monitoring

### Chronic Subdural Hematomas

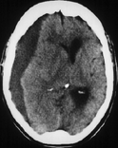
Chronic subdural hematomas are usually seen in the elderly and in over 50% of cases there is no history of trauma. However, systematic questioning may reveal even minor trauma in the weeks prior. These hematomas present either because of enlargement or because of acute bleeding into them.

Clinical

* Headaches
* Confusion
* Disorientation
* Focal neurological signs
* Seizures

Investigations

* CT Scan
  + Hypodense appearance on CT scan
  + Midline shift, with effacement of ipsilateral ventricle
  + Midline shift may have ipsilateral ventricular effacement, may have midline shift
  + May have acute component (hyperdense on CT Scan) with then chronic component.
  + If patient has been lying down for a period of time, then the acute component may be lying posterior showing a layered appearance



*Cranial CT scan shows the chronic right-sided subdural hematoma with mass effect*

Management

* Age, signs of hematoma and GCS as well as activities of daily living all need to be taken into account
* Discussion with and transfer to Neurosurgical Unit
* Minimal symptoms without midline shift may be monitored with repeat scans
* Surgical evacuation via burr-holes or craniotomy, the latter if there is an acute component and the drain is often left in for 24 -48hours
* 20% of patients will require reoperation
* As this is a condition of the elderly with cerebral atrophy, it is not unusual to have a small residual amount which needs to be monitored.

## HEAD INJURIES

Introduction

Head injuries can be divided into 3 categories. This is based on their Glasgow Coma Score (GCS).

* Mild head injury – GCS 13-15
* Moderate head injury – GCS 8-12
* Severe head injury – GCS 7 or less
  + If the patient falls into this category then intubation is indicated.

In head injuries in particularly moderate or severe it is fairly self-explanatory that these need to be managed in a Neurosurgical Unit. Conditions that fall into these categories are described above. Mild head injuries are the ones that often create a degree of difficulty in particular as to whether or not to scan the patient and/or admit them.

Clinical

* With mild head injuries it is very important to ascertain the history as to what was the mechanism of injury.
  + Is there any external evidence of trauma
  + Does the patient have any comorbidity that may put them at greater risk of an intracerebral hematoma (eg: anticoagulants)

Investigations

* CT scaning is indicated if;
  + GCS is less than 15
  + History of significant trauma
  + Patient unable to communicate (affected by substance abuse) or poor English
  + Documented loss of consciousness
  + Persistent headaches after 4 hours

Management

* Presence of intracranial abnormality is an indication for discussion with a Neurosurgical Unit and constitutes admission and rescanning in the first 24hours. This is due to a fact that a number of intracranial traumatic lesions enlarge in the first 24 hours.
* Discharged from ED, then they should be given a head injury information sheet and at the very least be followed up by their GP within 48 hours. They should also be a low threshold for plain films and a CT scan of the cervical spine, particularly in the elderly to exclude a fracture. If a fracture is seen then an MRI should be performed to exclude ligamentous injury as well.

## Skull Fractures

Skull Fractures can be divided into 2 groups

* Skull Vault
* Base of Skull.

### Skull Vault

These may be closed or open (compound depressed or depressed, and if depressed it may or may not be a dural laceration underlying contusion).

Clinical

* Soft tissue swelling overlying the involved area
* Laceration with palpable depression below

Investigations

* CT Scan with bone windows and fine slices through the suspected area is the gold standard.

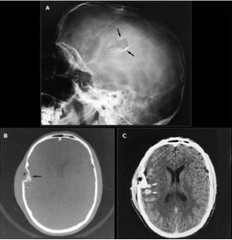
Plain skulle X-rays may or may not show evidence of a fracture.

* MRI may or may not show a fracture but is more important in showing whether or not there is damage to the underlying parenchyma, and potentially a dural laceration.



CT scan demonstrates open comminuted and depressed frontal bone fracture with clotted blood along the interhemispheric fissure.and middle fossa extradural

Management



Management

* Closed not displaced skull fractures are treated conservatively
* Open skull fractures: The wound should be debrided, thoroughly cleaned and sutured. If there is evidence of depression (the outer table has gone beyond the inner table) then elevation is indicated.
* Evidence of dural laceration and underlying contusion, increases the risk of intracranial infection and seizures. Surgery is more likely to be performed in these cases although elevation of the depressed fracture if there has been a dural laceration and contusion does not necessarily decrease the risk of epilepsy.
* “Cosmetic” elevation: in the case of a closed fracture, where the outer table has gone beyond the inner table, then elevation needs to be seriously considered. In an open depressed fracture, elevation is usually performed on lesser depressions.
* Antibiotics usually not given for a closed fracture. More likely in open fracture

### Fractured Base of Skull

In order to reduce a fractured base of skull a patient needs to suffer a significant injury to the skull. This may be a difficult diagnosis both clinically and radiologically.

Clinical

* CSF Rhinorrhoea
* CSF Otorrhoea
* Bilateral periorbital bruising (raccoon eyes, usually associated with periorbital nasal fractures)
* Mastoid bruising, also known as battle signs, whether it’s bruising behind the ear of the mastoid process
* Anosmia due to fractures through the ethmoid sinuses
* Hemotympanum
  + Blood in the external auditory canal where there does not appear to be any injury to the external structures of the ear.

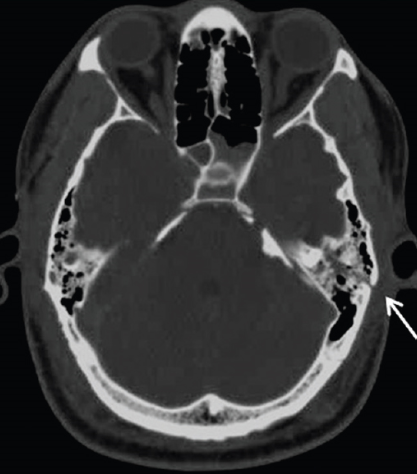
Investigations

* CT Scan with bone windows and fine slices through the suspected area is the gold standard.

Plain skull X-rays may or may not show evidence of a fracture.

* MRI may or may not show a fracture but is more important in showing whether or not there is damage to the underlying parenchyma (not as common as with depressed skull fractures)
* Biochemical, Beta2 transferrin can be used to see if CSF is present

*Depressed skull fractures*



CT demonstrating Base of Skull Fracture

Management

Most base of skull fractures do not require any surgical intervention unless there is evidence of CSF, rhinorrhoea or otorrhoea which does not settle.

* Antiobiotics are not indicated
* Discussion with a Neurosurgeon and possible transfer is always recommended.

## BRAIN ABSCESS

Introduction

Brain Abscess arise by contiguous or haematogenous spread. If not recognized early it is associated with high morbidity and mortality.

Contiguous (direct) spread is via infected sinuses or middle infections with the abscess usually adjacent to the infected extracranial area. Penetrating head injuries may also be a cause.

Haematogenous spread is usually associated a bacteremia or septicemia; risk factors associated with these include bacterial endocarditis, and pulmonary causes such as infection or A-V fistula, and. immunosuppressive patients are at the greatest risk.

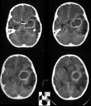
Although its incidence has decreased over recent decades with the aggressive treatment of many predisposing ENT infections in the more modern setting “atypical” infections are being seen in the setting of the immunosuppressed, including those with HIV/ AIDS.

Clinical

* Signs of raised intracranial pressure
* Focal neurological symptoms or signs
* Epilepsy
* Fevers
* Confusion

Investigations

* CT-Scan: Ring enhancing lesion with surrounding oedema
* Full septic screen
* DO NOT lumbar puncture



Management

* Contact Neurosurgical Unit
* Arrange urgent transfer after patient stable (ie seizures -> anticonvulsants, raised ICP -> consider mannitol)
* Discuss with referring unit as to whether or not to start AB

Further Management

* Surgery
  + Burrhole aspirations or craniotomy and drainage
  + Surgery may need to be repeated
* Antibiotics
  + Started empirically after septic workup +/- surgery

LUMBAR SPINE

## Lumbar Spine

#### Low Back Pain

(General outline)

* 40 – 60% LBP during their life
* Usually mechanical back pain (also non-mechanical & referred)
* Acute: < 6wks, subacute: 6 – 12 wks, chronic: > 12 wks.
  + 60-90% better in 6 weeks
* 50% of mechanical no cause identified
  + Pain from muscle or joint strain.
  + Settle with conservative measures.

Watch out for “red flags”

* + Known history of malignancy
  + Weight loss
  + Fevers
  + Severe pain with movement
  + Nocturnal pain
  + Neurological symptoms & signs.

Treatment

* + Nonspecific: pain relief, NSAID, steroids, CTGNRI, physio, modified activities
  + Suspicion: X-rays, CT-Scan, MRI, bone scan
  + Underlying pathology
    - Disc prolapse
    - Foraminal stenosis
    - Lumbar canal stenosis
    - Lumbar spondylolithesis
    - Malignancy

## Disc Prolapse

Introduction

* Nucleos pulposis herniates through annulus. Occur posterolateral.
* Often initially back pain then radiation down leg
* Approximately 50% history of precipitating event
* Other 50% “wear and tear” over time

Clinical

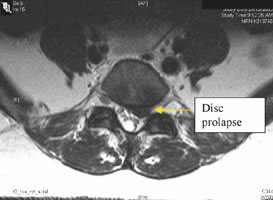
* Lower or buttock pain on relevant side
* Radiation of pain down leg; extent dependent on nerve root involved
* Paraesthesia/numbness usually on foot (distribution dermatomal dependent)
* Weakness (myotomal dependent)

Investigations

* CT-Scan: often show disc prolapse
* MRI: “gold standard” for imaging lumbar spine. Important is relationship of disc prolapse to the nerve root (eg contact or compression)



*Sagittal T2 MRI showing lumbar disc prolapse*

**

*Axial T2 MRI showing lumbar disc prolapse*

Management

* Initially conservative unless evidence of significant neurological deficit
* 80% will settle with conservative measures in 4-6 weeks
* NSAID, Tapering course of prednisolone, CT-guided nerve root injection
  + NSAID: Voltaren 50mg TDS for acute pain, mobic 15mg daily for pain of over 2 weeks duration.
  + Prednisolone 25mg TDS for 3 days

25mg BD for 3 days

12.5mg BD for 3 days

12.5mg mane for 3 days

* Physiotherapy
* Failure to respond to conservative treatment or neurological deficit, then referral for possible surgery

**\*Remember “Red Flags” and progressive neurological deficits: Early referral!**

## Foraminal Stenosis

Introduction

* Narrowing of the existing foramen
* Degenerative process
* Often multilevel
* More often seen in the elderly; very common
* Mainly asymptomatic or non-specific back pain

Clinical

* Lower or buttock pain on relevant side
* Radiation of pain down leg; extent dependent on nerve root involved
* Paraesthesia/numbness usually on foot (distribution dermatomal dependent)
* Weakness (myotomal dependent); not common

Investigations

* CT-Scan: often show foraminal narrowing
* MRI: shows the degree of nerve root compression

Management

* Initially conservative
* NSAID, Tapering course of prednisolone, CT-guided nerve root injection
* Structured rehabilitation program
* Failure to respond to conservative treatment or neurological deficit, then referral for possible surgery

## Lumbar Canal Stenosis

Introduction

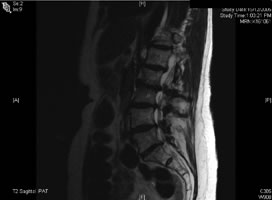
* Narrowing of spinal canal
* Combination of disc prolapse, hypertrophy of ligamentum flavum and overgrowth of facet joints (each to a varying degree).
* Degenerative changes that occurs over time. More often seen in the elderly

Clinical

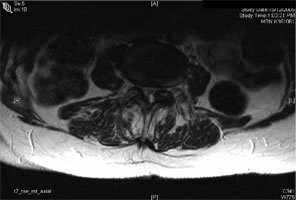
* Neurogenic claudication
* Pain in lower back or buttock radiating down one or both legs, worse with walking and standing, relieved with sitting
* May have sensory symptoms
* Often walk slightly flexed at hips weakness
* Progressive
* Usually no or very few neurological findings on examination

Investigations

* CT-Scan: often show lumbar canal stenosis
* MRI: better defines the extent of the stenosis



*Sagittal MRI demonstrating lumbar canal stenosis at multiple levels*



*Axial MRI demonstrating lumbar canal stenosis through L4/5*

Management

* Initially conservative unless progressive symptoms disrupting patient’s lifestyle or evidence of significant neurological deficit
* NSAID, or epidural steroid injection
* Physiotherapy may be of value
* Failure to respond to conservative treatment or neurological deficit, then referral for possible surgery

## Lumbar Spondylolithesis

Introduction

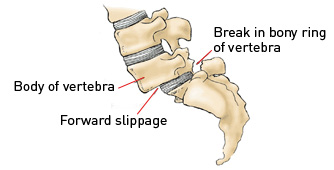
* Subluxation of one vertebral body on another
* Several different types
  + Isthmic: failure of neural arch producing a pars defect
  + Dysplastic or congenital
  + Degenerative
  + Traumatic
  + Pathological
* Pars defect and degenerative commonest
* Often asymptomatic
* May cause central, foraminal stenosis or both
* Grades 1-4 (<25%, 25-50%, 50-75%, >75%)

Clinical

* Back pain often a significant component
* Neurogenic claudication (see above)
* Leg pain with or without sensory symptoms (due to foraminal stenosis; see above)
* Symptoms may progress over time

Investigations

* CT-Scan: show degree of spondylolisthesis. May show central canal or foraminal stenosis or both. May show pars defect
* MRI: better defines the extent of the stenosis and nerve root compression. May show pars defect
* Plain films including flexion/extension views looking for instability. Also pars defect

*Sagittal MRI demonstrating lumbar spondylolisthesis at the L4/5 level*

Management

* Initially conservative unless progressive symptoms disrupting patient’s lifestyle or evidence of neurological deficit
* NSAID, or epidural steroid injection
* Structured rehabilitation
* Failure to respond to conservative treatment or neurological deficit, then referral for possible surgery. Often a fusion is required.

## CERVICAL SPINE

Neck Pain (General outline)

* Up to 60% of people will experience neck pain during their life
* May arise from several structures in the neck or be referred from other areas of the body; not always cervical spine

From Cervical Spine

* + Soft tissue – “whiplash” injuries
  + Cervical disc prolapse
  + Spondylitic changes – Facet joint degeneration and osteophytes, often produce foraminal stenosis
  + Central canal stenosis

Watch out for “red flags”

* + Known history of malignancy
  + Weight loss
  + Fevers
  + Severe pain with movement
  + Nocturnal pain
  + Neurological symptoms & signs.

Treatment

* + Soft tissue: pain relief, NSAID, physio, modified activities
  + Suspicion: X-rays, CT-Scan, MRI, bone scan
  + Underlying pathology

## Disc Prolapse

Introduction

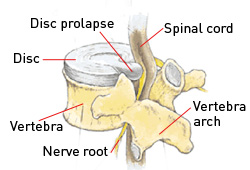
* Nucleos pulposis herniates through annulus. Occur posterolateral.
* Often initially neck pain then radiation down the arm
* Less than 40% history of precipitating event
* Others “wear and tear” over time

Clinical

* Lower or buttock pain on relevant side
* Radiation of pain down arm; extent dependent on nerve root involved
* Paraesthesia/numbness usually in hand and worse in particular finger (distribution dermatomal dependent). Commonest are thumb and/or index finger.
* Weakness (myotomal dependent). Commonest are elbow extension and flexion.
* C6 and C7 nerve roots commonest involved.

Investigations

* CT-Scan: may show disc prolapse. The absence of a prolapse of CT-Scan DOES NOT exclude a disc prolapse. CT-Scan best for bony structures
* MRI: “gold standard” for imaging cervical spine. Important is relationship of disc prolapse to the nerve root (eg contact or compression). MRI cervical spine are rebatable when ordered by GP



*Image © Martin Dunitz 2001, from Sports Injuries: Their Prevention and Treatment, 3rd Edition, by Lars Peterson and Per Renstrom, published by Martin Dunitz Ltd, www.dunitz.co.uk*

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*MRI of Cervical Disc Prolapse*

Management

* Initially conservative unless evidence of significant neurological deficit
* 80% will settle with conservative measures in 4-6 weeks
* NSAID, Tapering course of prednisolone, CT-guided nerve root injection
* Failure to respond to conservative treatment or neurological deficit, then referral for possible surgery

**\*Remember “Red Flags” and progressive neurological deficits: Early referral!**

## Foraminal Stenosis

Introduction

* Narrowing of the existing foramen
* Degenerative process
* Often multilevel
* More often seen in the elderly; very common
* Mainly asymptomatic or non-specific neck pain

Clinical

* Neck pain; not necessarily on the side
* Radiation of pain down arm; extent dependent on nerve root involved
* Paraesthesia/numbness usually on foot (distribution dermatomal dependent)
* Weakness (myotomal dependent); not common

Investigations

* CT-Scan: often show foraminal narrowing but not necessarily nerve root compression
* MRI: shows the degree of nerve root compression, although may be multilevel

Management

* Initially conservative
* NSAID, Tapering course of prednisolone, or CT-guided nerve root injection (CTGNRI). CTGNRI can be both diagnostic and therapeutic, particularly with the former in respect to multilevel stenosis.
* Failure to respond to conservative treatment or neurological deficit, then referral for possible surgery

## Cervical Canal Stenosis

Introduction

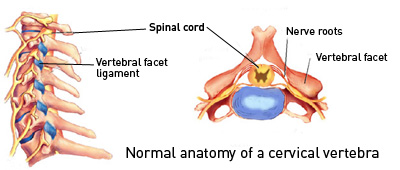
* Narrowing of spinal canal
* Combination of disc prolapse, hypertrophy of ligamentum flavum and overgrowth of facet joints (each to a varying degree).
* Central disc prolapses
* Degenerative changes that occurs over time. More often seen in the elderly

Clinical

* Myelopathic changes.
* Often pain radiating down one or both arms.
* Sensory symptoms and signs in one or both hands.
* Problems with fine motor movements
* Gait disturbance
* Progressive
* Neurological findings include weakness, sensory signs, hyperreflexia and/or clonus

Investigations

* CT-Scan: often show cervical canal stenosis.
* MRI: Shows the extent of the stenosis, whether there is compression from in front, behind or both. Also whether there is signal change within the spinal cord.





*Vertebra*

Management

* Careful assessment needs to be made both clinically and radiologically.
* The presence of either neurological symptoms/signs and or severe stenosis radiologically should initiate referral or at the very least a phone call.
* Surgery is frequently indicated where the above applies.

Spinal Infection

## Discitis

Introduction

Primary infection of the nucleus pulposus with secondary involvement of cartilaginous endplate and adjacent vertebral body.

Clinical

* Pain -> localised to the area of involvement
* Radiation -> abdomen, hip, groin and may radiate down leg
* Fever
* Significant limitation of back movements
* Localised tenderness and paraspinal muscle spasm.

Investigation

* Plain films (appearance depends on age of infection
  + Loss of disc height
  + Irregularity of vertebral endplates
  + Involvement of vertebral bodies (VB)
  + Fusion of vertebral bodies may occur at later date
* MRI
  + Improved imaging of disc space and VB
  + Show if paravertebral or epidural abscess
* Blood screen
  + Full septic workup
* Needle biopsy
  + If unable to obtain pathogen from septic workup



MRI showing Infectious spondylitis and discitis with paravertebral abcess

Management

* IV antibiotics (Staph Aureus commonest organism) and analgesia
* Immobilisation may help initially with pain relief.
* Surgery if unable to obtain organism (open biopsy) or if epidural collection

## Vertebral Osteomyelitis

Introduction

Often associated with discitis. Lumber spine commonest. Seen in:

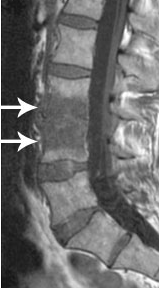
* IV drug users
* Diabetics, patient on haemodialysis, infective endocarditis
* Immunosuppressed patients
* Post spinal surgery

Clinical

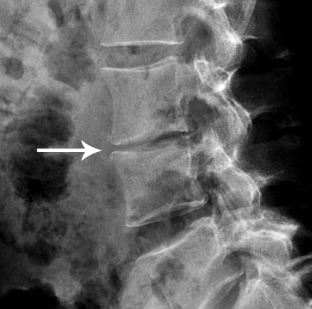
* Pain -> localised to the area of involvement
* Radiation -> abdomen, hip, groin and may radiate down leg
* Fever
* Limitation of neck or back movements
* Localised tenderness and paraspinal muscle spasm.
* Cord compression if associated epidural abscess
* Depending on location: pharyngeal abscess or mediastinitis

Investigation

* Plain films (appearance depends on age of infection)
  + Loss of disc height (if disc space involved)
  + Irregularity of vertebral endplates
  + Involvement of vertebral bodies (VB)
  + Fusion of vertebral bodies may occur at later date
* MRI
  + Improved imaging of disc space and VB
  + Show if paravertebral, epidural abscess, pharyngeal abscess or mediastinitis
* Blood screen
  + Full septic workup (UTI commonest source)
* Needle biopsy
  + If unable to obtain pathogen from septic workup



MRI of lumbar spine discitis/osteomyelitis.  Sagittal T1-weighted images of the lumbar demonstrate T1-hypointense signal (solid arrows) centered around the L3-4 interspace.



Plain film radiograph of spinal discitis / osteomyelitis. Lateral view of the lumbar spine demonstrates L 3-4 disc space narrowing (arrow) and end-plate irregularity.

Management

* IV antibiotics (Staph aureus commonest organism, then E.Coli) and analgesia
* Immobilisation may help initially with pain relief.
* Open biopsy if unable to obtain organism.
* Drainage of associated collection, particularly if symptomatic

**\*Facet joint infections similar to VB infection +/- epidural abscess**

## Epidural Abscess

Introduction

Are being diagnosed more frequently largely due to increase awareness and improved imaging. Seen in:

* IV drug users
* Diabetics, patient on haemodialysis, infective endocarditis
* Immunosuppressed patients
* Post spinal surgery

Clinical

* Pain -> localised to the area of involvement and tenderness
* Fever
* Limitation of neck or back movements
* Localised tenderness and paraspinal muscle spasm.
* Neurological signs are highly variable. Range from no symptoms/signs to those of significant cord compression. Signs may be due to direct compression or cord infarction secondary to thrombosis.

Investigation

* MRI
  + Should be performed if slightest suspicion.
  + May only initially see no or subtle changes. Should repeat after a few days.
* Blood screen
  + Full septic workup
    - Skin infection (commonest; Staph. Aureus, then Strep.)
    - IV Drug users
    - UTI (E.Coli)
    - Bacterial endocarditis
* Needle biopsy
  + If unable to obtain pathogen from septic workup



Spinal epidural abscess lumbar area



Cervical epidural abscess with spinal cord compression and spinal cord edema

Management

* IV antibiotics
* Surgery if neurological symptoms and/or signs, large collection or unknown organism
* Drainage of asymptomatic and known organism remains controversial. Transfer to a Neurosurgical Unit is indicated in such cases for a definitive decision.
* **This condition cannot be underestimated as misdiagnosis or delay in diagnosis can result in significantly increased morbidity and mortality. Significant back pain and fever must raise the possibility of a spinal abscess..**

## Epidural Haematoma

Introduction

Unusual condition although more frequently diagnosed now because of improved imaging. Causes:

* Vascular Malformation
* Trauma
* Coagulation Disorders
* Anticoagulation
* Cavernoma
* Unknown

Clinical

* Pain -> localised to the area of involvement
* Limitation of neck or back movements
* Neurological signs are highly variable. Range from no symptoms/signs to those of significant cord compression.

Investigation

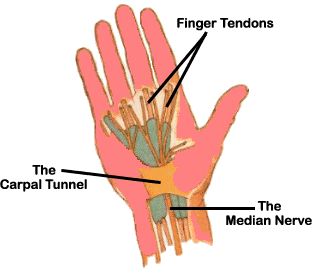
* MRI
  + Evidence of haematoma with or without cord compression
  + Abnormal vessels on cord with vascular malformations
  + May see cord oedema
  + Exclude intramedullary cavernoma
* Spinal Angiogram
  + To delineate if there is a vascular lesion

Management

* Transfer to a Neurosurgical Unit.
* Evacuation of haematoma if neurological symptoms and/or signs, or haematoma causing cord compression.
* Reversal if secondary to anticoagulation.
* Correction of coagulation disorder.
* Surgical removal of underlying lesion and/or embolization.
* **This condition cannot be underestimated as misdiagnosis or delay in diagnosis can result in significantly increased morbidity and mortality. Significant back pain and a patient on anticoagulation, or with a coagulation disorder must raise the possibility of an epidural laceration.**

Peripheral Nerves

**Carpal Tunnel Syndrome**



Introduction

Carpal tunnel syndrome is due to compression of the media nerve as it passes through the carpal tunnel at the wrist. Causes include:

* Repetitive movements
* Pregnancy, due to fluid retention
* Medical condition such as hypothyroidism and rheumatoid arthritis
* Unknown

Clinical

The patient classically presents with nocturnal symptoms

* Wake patient, relief with changing position or shaking hand
* Paraesthesia and/or numbness in lateral 3.5 fingers.
* May be associated with wasting (in particular abductor digit minimi) and weakness over time. Potentially problems with fine motor movements.
* May have positive phalen’s and/or tinel’s sign

Investigations

* Nerve Conduction Studies
  + Mild, moderate or severe changes
* MRI
  + Compression of the nerve in the carpal tunnel; often oedematous.
  + May be associated synovitis, inflammatory changes, cysts etc.

Differential Diagnosis

* Peripheral Neuropathy
* Cervical nerve root compression
* Cervical cord compression.

Management

* Pregnancy. Symptoms should settle after delivery.
* Medical conditions should be treated first
* Mild changes on nerve conduction studies. Management conservatively (eg splints, diuretics, steroid injection) unless causing patient problems
* Moderate and severe usually require surgical decompression by dividing the flexor retinaculum.

**Ulnar Nerve Entrapment**

Introduction

* Common side of an ulnar verve entrapment is at the elbow (tardy ulnar palsy).
* Rarely involves neuroma
* At wrist much less likely
* May have a history of trauma, previous fracture to the area or an underlying arthritic condition such as rheumatoid arthritis

Clinical

* Paraesthesia and/or numbness involving the medial 1.5 fingers of the involved hand.
* In more longstanding cases or severe cases there may be wasting of the hypothenar eminence and the small muscles of he hands (intraosseous)
* Weakness of finger abduction. In longstanding cases the patient may develop a claw deformity of the hand.
* Wasting of hypothenar eminence and intraosseous muscles of the hand

Investigations

* Nerve Conduction Studies/EMG
  + Mild, moderate, severe
* MRI
  + Presence of neuroma. Often used to conjunction with NCS/EMG for loacalisation.

Differential diagnosis

* Cervical nerve root compression

Management

* History suggestive of neurapraxia. Suggest wait 3 months, unless symptoms progressive and/or patient has neurological deficit.
* Moderate to severe ulnar nerve entrapment at the elbow then surgery should be considered. Surgery involves decompression of the ulnar nerve at the elbow with various modifications of this operation including transposition of the nerve and a medial epicondylectomy (if history of trauma or arthritis)

*Ulnar nerve shown in red*

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**Common Peroneal Nerve Palsy**

Introduction

Common peroneal nerve is made up of

* Superficial and Deep component.
* Entrapment of the nerve which most often occurs as the nerve crosses the fibular head or as it penetrates the peroneus longus
* Rarely involves neuroma
* Maybe history of trauma to knee

Clinical

* Weakness of dorsiflexion
* Weakness of foot eversion
* Mild sensory changes on lateral aspect of foot

Investigations

* Nerve Conduction Studies/EMG
  + Mild, moderate, severe
* MRI
  + Presence of neuroma. Often used to conjunction with NCS/EMG for localisation.
  + Cyst or ganglion arising from knee joint

Differential diagnosis

* L5 nerve root compression
* Lumbar canal stenosis
* Diabetic neuropathy

Management

* History suggestive of neurapraxia. Suggest wait 3 months, unless symptoms progressive and/or patient has neurological deficit.
* Moderate to severe entrapment at the fibular head then surgery (decompression) should be considered.
* Excision of neuroma if indicated

