CERVICAL SPINE PAIN V.I.N.D.I.C.A.T.E.R

**Vascular Visceral referral Infective Neoplasms**

-Angina *GIT* -Vertebral osteomyelitis -Pancoast tumor

-MI -Liver pathologies -Meningitis -Cervical cord tumors

-AAA -Pancreas pathologies -Lyme disease -Esophageal cancer

 -Oesophagus -Acute pyogenic infection -Thyroid cancer

**Rheumatological** -Diaphragm *-*Tuberculosis-Cervical bone tumours

-RA (instability) -Spleen -Multiple Myeloma

-PMR -Crohns -Hodgkin’s lymphoma

-PAN -UC -Secondary’s from breast, lung, kidney,

-GCA prostrate

-Polymyositis

-Fibromyalgia

-Psoriatic

-Reiters syndrome

-Enteropathic

-SLE

-Whipples

-AS

**Congenital/Developmental** **Repiratory** **Headaches**

-Scoliosis -Hyperventilation -Cervicogenic

-Cervical Rib -Apex tumour/Bronchial carcinoma -Tension

-Sprengels shoulder -COPD -Migrainous

-Kleppel feil syndrome -Pneumothorax -Cluster

-Marfans -Sinusitis

**-**Hypermobility

-Turners syndrome

-Buergers syndrome

**Trauma-**

-Fractures/Osteoporosis/Falls/Domestic

-Whiplash

**CERVICAL PAIN-MUSCULOSKELETAL/NEUROLOGICAL**

**Musculoskeletal** **Neurological** **Musculoskeletal Referred**

-Disc herniation/radiculopathy -Cervical Myelopathy -TMJ

-Annular strain -VBI -Shoulder complex i.e. Glenoid/ 1st rib

-Spondylosis DDD -Radiculopathy? -Thoracic spine

-Sponyloarthrosis -Thoracic Outlet -Functional i.e postural fatigue, myofascial

-Stenosis -TIA

-Spondylolosis -Motor Neuron Disease

-Capsular strain -Multiple Sclerosis

-Meniscoid entrapment/facet lock -Myasthenia Gravis

-Osteoarthritis

-Torticollis

-Ligament sprain

-Muscle strain

-Cervicogenic headaches

-Tension headaches

 -

**Cervical Spine- Musculoskeletal**

**Annular strain**

*Definition*

Annular tears or properly called ‘Annular fissures’ are separations between annular fibers, avulsion of fibers from their vertebral body insertions or breaks through fibers that extend radially,transversely or concentrically. There are 3 types of recognized tears.

A/ Circumferential tears

B/ Radial tears

C/ Rim tears.

The annulus fibrosus outer one third is innervated by the recurrent meningeal nerve and the vertebral nerve (the former deriving from the ventral ramus and the latter from the autonomic chain). If fissuring is extensive and fault lines occur from the nucleus than chemical spillage can cause pain if it reaches outer one third of the annulus, alternatively mechanical compression from loading in the outer third can stimulate mechanorecptor and free nerve endings to cause referred pain. Pain is usually somatic referred and non dermatomal unless there is nerve root irritation or compression. It can be unilateral or bilateral depending on the extent of the damage

-C5,C6 and C6,C7 most common accounts for 75% of discogenic pain (emedicine). C7 is the most common nerve root involved (emedicine)

Incidents peak around 45-50 yrs old**.**

*Cause*

Degenerative changes occur mostly at C5,C6/ and C6/C7 because most mobile areas of neck and because under most compressive loads (because of head). Degenerative changes causes loss of nutrition and water so disc loses height and annulus has to load more. The loss of shock absorbing capacity affects the zygopophysial joints. Loss of disc height and degenerative facets causes risk of nerve entrapment as IV is encroached

Predisposing factors include

-Direct Trauma e.g. falls, motor vehicle accidents, whiplash and sports injuries

-Repetitive micro trauma such as repeated flexion

-Prolonged compression (sitting) and poor posture e.g. increased cervical lordosis

-Sudden unguarded movements

*Clinical Presentation*

Symptoms: Patient usually presents with diffuse non dermatomal pain and paresthesia that may radiate down shoulder, interscapular area or midback (location of pain may indicate what level and what area of annulus is affected ***Cloward 1959 “Cervical Diskography”. Full radial tears may cause interscapular pain and dermatomal arm pain as nerve root is affected***



Agg: Pain may be aggravated by prolonged sitting posture or with repeated or prolonged flexion, arm movements, coughing, sneezing, defecation

Observation: Guarded movements, head positioned away from lesion

Palpation: Local tenderness, muscle spasm and guarding, hypertonicity and joint restriction

Tests:Spurling’s test may reproduce pain. Axial distraction test may relieve pain. Arm abduction test may relieve symptoms. Neurological Exam P and E

*Management*

In acute phase calm down muscle spasm without overstretching or irritating joint. Cervical traction may be useful. Increase the mobility in hypo mobile segments to off load overloaded disc, also improve thoracic mechanics e.g. kyphosis. Patient management may be cryotherapy, ergonomics, posture and positional awareness. Strengthening and stretching exercises in later phase of recovery

**DD: DD-Ensure following red flags are not present**

-**Cancer/Malignancy** (*Ask questions about history of previous cancer, unexplained weight loss, pain with bed rest, night pain, fever, lethargy)*

-**Infection** (*Ask questions related to potential infection such as history of recent surgery, history of fever/chills, history of IV drug use)*

**-VBI** *Ask questions about syncope (drop attacks), vertigo, nausea and vomiting, CN involvement such as diplopia, dysarthria, facial numbness*

-**Trauma with underlying fracture**

(*Ask about past incident history, whiplash’s, RTA’s, falls, sports injuries)*

-**Osteoporosis with underlying fracture**

(*Ask about any previous trauma’s/fractures, family history and if female menopause times)*

-**Conditions with spinal instability e.g RA**

*Any previous whiplash’s, Symptoms for RA include polyarthralgia, morning stiffness, fever, fatigue, malaise, hand joint pains and systemic signs*

-**UMN signs/symptoms e.g. cervical myelopathy, multiple sclerosis (***C M:Weakness of stiffness in the legs, unsteady gait, clumsy or weak hands, signs include sensory loss, Lhermittes sign, ankle clonus, + Babinski’s, absent reflex at C5/6 with hyper reflexion at C7 MS: Visual impairement, Bladder dysfunction, loss of balance/dizziness, hyper reflexia, spasticity, + Babinski’s*

**Cervical Facet Capsular strain**

*Definition*

Facet joints can be irritated in many ways. Mostly pain is caused by extension/rotation movements; however flexion and meniscoid entrapment can also cause facet pain. Capsular irritation in extension can be caused when for example the left zygopophyisial facet joint gets impacted into the laminar (instead of gliding) the contralateral (right) inferior articular process is forced backwards into rotation, resulting in capsular disruption. This may happen in an acute traumatic event. Alternatively capsular extension pain can occur in prolonged disc degeneration/spondil oarthropathy; a loss of disc height means there is more weight bearing and greater impaction of the zygophophyisial facet joints causing cartilage pain. Pain is generated from capsular compression stressing nerve endings and chemical inflammation irritating nerve endings. Another capsular strain mechanism is rotation. Rotation is usually limited by the impaction of the zygopophysial joint opposite the direction of movement e.g. right zygopophisial joint impacts during rotation left. If the torque is strong enough the contralateral e.g. left facet joint gets gapped by being drawn backwards and medially, this can disrupt capsule mechanics, tear or avulse the capsule ( a trauma could do this e.g. whiplash)

 Irritation or damage to the facet joint capsule may cause cranial, cervical, upper shoulder or back pain referral. It is estimated that 26-65% of neck pain complaints have a cervical facet component



*Cause*

1. Whiplash (the prevelance of cervical facet pain after whiplash injury is 60% most common at levels being C2,C3 and C5/C6

2. Cervical disc injuries (disc pain at the same spinal level is associated with facet pain in -40%of cases)

3. Osteoarthrosis

4. Repetitive stress/poor posture (occupational)

*Clinical Presentation*

Symptoms: Pain is usually unilateral and felt in the neck and sometimes radiates to the shoulder or mid scapular areas. PT’s usually reports a dull achy pain although may be sharp pain or pinch when turning head towards painful side or during extension. Pain usually does not go beyond elbow or radiate below upper T spine. Patient has limited ROM in more than 1 direction

Observation: May be slight antalgic neck posture as neck is flexed and laterally side flexed away from painful facet, may have increased cervical lordosis and increased thoracic kyphosis

Palpation: TTP over facet joints affected with muscle guarding and hypertoniicity in surrounding musculature

Active: Usually quadrant testing movements painful but flexion can also aggravate as capsule is stretched. However active motions usually limited and painful in most directions

Tests: Spurlings more likely painful as facet joints approximated

*Management*

-In acute phase reduce inflammation with ice. May also respond well to traction. ST to reduce inflammation, in later stages deep tissue work to improve ROM. In later stage hypomobile facets respond well to manipulation. Improve posture via stretching and strengthening exercises

-DD’s include: Annular strain, muscle strain, radiculopathy, infection, neoplasm, aneurysm

**Cervical Spondylosis DDD**

*Definition*

Progressive loss of articular cartilage with disc space narrowing, subchondral sclerosis, osteophyte formation and facet joint degenerative changes. Early trauma causing endplate damage can lead to rapid acceleration of degenerative changes as proteoglycans and water are lost from nucleus pulposus causing loss of disc height and subsequent increased loading on the annulus, facet joints and vertebra bodies. As disc becomes unstable, osteophytes are layed down on the anterior and lateral vertebra bodies to help create more stability and support. However, consequently this can lead to increased neck stiffness and muscle loading and pain: in the unfortunate few osteophyte growth, ligamentum flavum hypertrophy and disc sclerosis can affect the spinal cord creating ‘Cervical Myelopathy’

*Cause*

1/ Traumatic structural damage e.g. whiplash, fall, sporting injury, previous surgeries, fractures

-Loss of nuclear hydrostatic pressure as a result of endplate fractures/inner annular disruption causes a shifting of axial loads to the annulus fibrosus, ring apophysis and facet joints. Or ligamentous damage causing laxity and instability in neck

 2/ Structural inheritance/ metabolic conditions

-Much greater risk of developing neck pain if parent has it .There may be genetic weaknesses in collagen framework of disc, blood supply, disc metabolism, disc size, torso size e.g. diabetes, osteoarthritis

3/ Occupation

-Seated jobs causing microtrauma to neck structures e.g. repetitive flexion, or extension or prolonged loading on neck structures e.g. sheer forces

4/ Smoking

-Smoking increases disc degeneration by 20% (Battie,1991). Smoking damages the already compromised capillary beds which reduces nutrient supply to the discs causing further dehydration.

5/ Glycation

-Is a biochemical reaction when sugars come into contacts with proteins (like disc collagen) in an avascular environment. In the absence of oxygen (avascular) sugars start to rub against and bind proteins with the collagen. This makes the collagen more brittle and ‘sticky’ weakening and aging discs

*Clinical Presentation*

Symptoms: Gradual onset of slowly developing dull aching neck pain. Morning stiffness and aggravation of pain by repetitive use or lifting

Observation: May have increased lordosis or loss of lordosis

Passive: Decreased ROM, hard end feel passively especially with loss of sidebending (osteophyte formation), clicking and crunching sounds (instability). Posterior and lateral muscle guarding

Active: Limited in most directions, painful

Tests-Spurlings (for IV encroachment). Watch out for Lhermette sign (suggests osteophyte pressing on spinal cord). Look for signs and symptoms of cervical myelopathy

*Management*

Most patients respond well to heat, traction to decompress joints, soft tissue relaxation (hypertonic muscles as a result of joint instability) or segmental facilitation, mobilization of segments (be wary of manipulation because of osteophyte formation)

Long term

-Strength and stretching

-Postural awareness

-Cardiovascular fitness improvemnets

-Nutrition

**Cervical Radiculopathy**

*Definition*

Cervical radiculopathy is a disease process marked by nerve compression from herniated disk material or arthritic bone spurs. This impingement typically produces neck and radiating arm pain or numbness, sensory deficits, or motor dysfunction in the neck and upper extremities specific to a dermatomal distribution. In 60% of nerve roots C7 is most commonly affected (C6/C7) and in 25% of cases C6 (C5/C6).

*Cause*

-In young patients sporting injuries involving forced extension, lateral bending or rotation, or with sudden or progressive loading/sustained postures in flexion or extension. In elderly patients nerve compression occurs from IV narrowing from disc degeneration and subsequent osteophte formation. Risk factors include:

1/ Heavy manual labour require repetitive lifting

2/ Driving or operating vibrating equipment

3/ Contact sports

4/ Prior trauma e.g. whiplash, falls, DDD

*Clinical Presentation*

Symptoms: Patient tends to present with unilateral neck pain with interscapular and radicular arm pain. Pain is described as a deep aching to burning sensation, the arm pain may be described as “numbness, tingling, sharp, shooting or electrical pain” Patient may complain of sensory changes, of muscle weakness in the arm/hand during functional activities. Patients pain may be provoked by certain neck movements such as flexion/extension/rotation/SB and worse in weight bearing positions e.g. seated at desk, driving car, reading book, lifting objects. PT may also have more pain when coughing, sneezing

Observation: Head tilt and neck posture (antalgic)

Palpation: If acute inflammation present, tissues feel boggy, spasmed and hot. If chronic protective hypertonicity present

Active: May be limited and painful in flexion (annulus bulging into nerve root) or limited in extension/ rotation/ side bending to same side as IV is closed down and nerve root compressed

Tests: Spurlings may acerbate symptoms. Neurological: When sensory fibers are blocked numbness is the symptoms and sign, when motor fibers are blocked weakness ensues. Diminished reflexes occur as a result of either sensory of motor block. Radiculopathy is only confirmed by objective neurological signs, if there is no sensation loss, motor loss or reflex changes but pain pattern is electrical lancinating and dermatomal the patient is said to have radicular pain and is more conducive to nerve root irritation rather than compression.

*Management*

In acute phase goal is to reduce pain and decrease spasm, inflammation, encourage healing process, deal with compensations as a result of altered postures, unload segment, manage patient to avoid further injury. Get patient to embark on cryotherapy routine for inflammation control. Manual therapy techniques would include pump and drainage methods to decrease inflammation without affecting healing process e.g. harmonic techniques, rhythmic articulations, gentle rhythmic tractions. No prolonged stretches in early phase, be careful with X-fibre and may disrupt healing better longitudinal or pump techniques, articulation in midrange only. Take load of segment by ensuring other cervical segments doing their job, also treat upper T-spine to reduce load to lower cervical segments. PT activity management: if desk orientated may have to encourage regular breaks from desk, encourage movement from thoracic spine (bottom up approach) to mobilize neck.

**Torticollis**

*Definition*

Rotation and lateral bending of the head due to SCM muscle dystonia ( involuntary sustained or spasmodic contractions involving co-contraction of the agonist and the antagonist). Also known as wryneck torticollis can be either acquired or congenital.

*Cause*

-The etiology of congenital torticollis is unclear, it is suspected that birth trauma or intrauterine malposition is said to be the cause of damage or fibrosis to the SCM muscle. This results in a shortening or excessive contraction of the SCM-the head is laterally flexed to the ipsilateral side and contralaterally rotated. Sometimes a SCM tumor is the cause and a palpable lump can be felt, gradually is disappears by 8 months but person is left with fibrotic muscle. Another congenital cause is Klippel Feil Syndrome.

-Acquired causes occur in childhood or adulthood. Acute conditions lasting 1-4 weeks may be due to awkward head position, draughts, colds or stress. Cervical disc degeneration may also cause this or previous neck trauma. More serious causes include posterior fossa tumours (which can compress nerves to neck muscles), infections in the posterior pharinx, glands, lymph nodes, sinuses, mastioids, jaw ear, teeth and scalp can all cause muscles spasm. Also the use of certain drugs such as antipsychotics.

*Clinical Presentation*

Symptoms: Neck stiffness/tightness/burning, shoulder pain, headaches, back pain

Observation: Persons head will be tilted to one side with visible hypertrophy of SCM and possibly other shoulder girdle muslces

Palpation: Muscle hyptertophy and hypertonicity in neck and shoulder girdle muscles e.g. SCM, levator scapulae, scalenes, trapezius and spleni musculature. Multiple cervical joint restrictions and myofascial trigger points

Active: Person will be reluctant to move neck

Tests: Neurological P and E

DD: Meningitis, osteomyelitis, tumours, trauma such as fractures, cervical disc disease (herniation)

*Management*

-Gentle soft tissue massage to hypertonic muscles, manipulation to help improve joint movement, prescribed stretching exercises. Long term: stress management, CBT (if psychosocial cause)

**Headaches**

*Cervicogenic*

*Definition*

-Cervicogenic headaches are headaches characterized by chronic hemicranial pain that is referred to the head from either bony structures or soft tissues of the neck (muscles, joints, fascia, neuro-vascular structures). Cervicogenic headaches are often linked with past trauma e.g. whiplash but may occur in the absence of trauma. Primary sensory afferents from roots C1-C3 converge with afferents from occiput and trigeminal afferents (via trigeminalcervical nucleus) on the same second order neuron in upper cervical spine results in anatomical structures innervated by cervical roots C1-C3 as potential sources of cervicogenic headaches. The convergence of pathways allows bilateral referral of painful sensations between neck and trigeminal sensory receptive fieleds of face and head. Pain is referred from nerves, muscles, ligaments, and joint capsules and vertebra

*Causes*

-Traumatic e.g. whiplash, falls onto neck/shoulder, direct compression or external blow to head neck

-Poor posture e.g. ergonomics

-Repetitive or sustained activities

-Sudden twisting

-Lifting

-Stress

-Aging and degenerative conditions

*Pathophysiology*

-C1 dorsal ramus or suboccipital nerve innervates the O/A joint so any restriction to joint can refer to occipital region

-C2 spinal nerve root and its dorsal root ganglion have a close proximity to the lateral capsule of the atlantoaxial joint (C1-C2) innervating both it and C2-C3, therefore trauma or pathological changes around these joints can be a source of referred pain (facet referred pattern). Secondly, emerging from the dorsal ramus of C2; a branch called the greater occipital nerve (sensory) passes below obliquus capitas inferior, thus any tension/restriction in muscle can irritate nerve causing referred pain over back of head (can also refer to eye). The greater occipital nerve can also be irritated aponeurosis attaching into mastoid

-C3 dorsal ramus innervates C2-C3 apophyseal joint, this joint is vulnerable to acceration/deceleration injuries of the neck e.g.whiplash.

*Clinical Presentation*

-Unilateral head or face pain, pain is localized to orbital, frontal, temporal or occipital regions, sometimes behind the eye

-Pain is moderate to severe in intensity

-Pain is generally deep dull ache and non-throbbing in nature, head pain is triggered by neck movements, awkward neck postures, pressure to suboccipital muscles or valsalva, cough or sneeze

-Occasionally person may experience other symptoms such as dizziness, nausea, light-headiness

-Reduced active and passive neck movements with neck stiffness

-Aggravated by prolonged postures e.g. sitting, driving or specific neck movements, patients may feel tight band of muscles into neck

*Management*

-Cervicogenic headaches respond very well to manipulation and mobilization, also consider massage to hypertonic musculature e.g. upper trapezius, splenius, suboccipitals and SCM, consider also T-spine mechanics

-Rule out pathological headache or red flags e.g. neurological deficits, new onset of headaches after 50, previous cancer, thunderclap headache, symptoms of GCA (jaw claudication, visual disturbance, fever, weightloss, proximal myalgia’s, temporal artery tenderness/pulselessness), systemic symptoms such as fever, weighlotss, progressively worsening headaches, red eye/halo around lights (glaucoma)

***Tension Headaches***

*Definition*

-Tension type headaches are the most common cause of primary headaches. The origin of pain has long been attributed to ischaemia of head and neck muscles in acute states. There is increased myofascial pain sensitivity in chronic tension type headaches states possibly caused by sensitization of second order neurons at the level of the dorsal horn caused by prolonged nocieceptive stimuli from pericranial myofascial tissues. The average age of onset is 25-35 years and peaks at 30-39 with women slightly being more affected then men.

*Causes*

-Stress and maladaptive coping strategies

-Depression and anxiety

-Neck pain

-Eye strain

-Sleep disturbances

-TMJ dysfunction

*Clinical Presentation*

-TTH is characterized by a bilateral pressing tight band like pressure around the whole head

-It is usually of mild to moderate intensity and occurring in short episodes. The headache is not associated with migraine type symptoms such as nausea, vomiting or severe photophobia or phonophobia

-Typically they start soon after waking and worsen as the day progresses, the headaches may last 30 minutes to several days but very rarely wake patient from sleep

-Respond well to analgesics

-Palpation may reveal tender hyperirritable bands of muscle around cervical spine e.g. trapezius, suboccipitals, posterior neck musculature

***Cluster Headaches***

*Definition*

-Cluster headaches are primary neurovascular disorders. They are thought to bee related to dysfunction in the posterior gray matter of the hypothalamus. There are also altered neurological circuitry in the trigeminalvascular system affecting the branches of trigeminal nerve (this may explain pain) along with vascular dilation (increased blood flow in temporal artery). They are defined by duration as episodic or chronic.

1/ Episodic: occurs in clusters lasting from 7 days to 1 year, clusters separated by pain free intervals lasting at least 2 weeks (usually a cluster lasts 2 weeks to 3 months)

2/ Chronic: occur for more than a year without remission or with remission lasting less than 2 weeks

Cluster headaches tend to effect males 6:1 ratio of ages 30-50

*Causes*

-Hypothalamic hormonal influences disrupting circadian rhythm

-Disrupted auto regulation of cerebral arteries (hypoxemia)

-Disorders of serotonin metabolism

-Poorly understood may have a link to genetics. Triggers include

-Alcohol (symptoms start 1 hr post drink)

-Strong smelling substances such as solvents, perfumes and petrol

-Hot room or hot bath

-Stress/bright lights

*Clinical Presentation*

-Pain is located always unilaterally in an orbitotemporal distribution and can cause stabbing pain into the eye, it is sudden and severe and often described as excruciating, piercing, boring, penetrating, throbbing

-Symptoms include lacrimation (tearing), nasal congestion, ptosis, facial flushing and pain into nostril and cheek

-It is an excruciating type of pain peaking within a few minutes and usually subsiding within 5minutes to 1 hour

-Symptoms are distinctive. Attacks usually occur at the same time of the day yet are more common at night often awakening patients from sleep at same time e.g. 1am, 4am, 7am.

-Patients are usually agitated preferring to restlessly pace the floor rather than sleep in a quiet darkened room (migraine)

-Symptoms occur daily in clusters e.g. over 6-12 weeks within a year and is usually seasonal (in uk December, January and February are particularly provocative)

DD’s include TMJ syndrome, temporal arteritis, migrane, cervicogenic headaches

***Primary disorders- Clinical Presentation***

*Migraines*

*Definition*

-Migraines are chronic common episodic heads pain thought to be a neurovascular disorder. Altered central neuronal processing (activation of brain stem nuclei, cortical hyper excitability) and involvement of the trigeminovascular system (triggering neuropeptide release) seems to be the causes for painful inflammation in the cranial vessels and dura matar. Migraines commonly begin in puberty or young adult hood, waxing and waning in frequency and severity over years, often diminishing after age 50. Migraines tend to affect males and females equally peak onset is 20-40 yrs but can occur any age

*Causes*

* Genetics-Primary cause
* Menstrual cycle (pregnancy/ovulation)
* Nutrition (drinking red wine/skipping meals)
* Stress
* Exposure to bright or florescent lights
* Strong odours
* Weather changes
* Head trauma
* Medications (oral contraceptives, vasodilators
* Lack of sleep

*Clinical presentation*

-Often attacks are heralded by prodrome (a sense that migraine is beginning) this may include mood changes, loss of appetite, nausea or combination

-An aura procedes attacks in about 25% of patients (Aura’s are temporary neurologic disturbances that can affect sensation, balance, muscle co-ordination, speech or vision lasting minutes up to one hour). Most aura’s involve visual symptoms such as flashing lights/ zig zags or scotomas. ***Paresthesias and numbness typically starting in one hand and moving to ipsilateral arm and face, speech disturbances, transient brain stem dysfunction (ataxia, confusion) are less common but can occur with aura***

-Symptoms typically last from 4-72 hours

-Symptoms include nausea (occasional vomiting), photophobia (sensitivity to light), phonophobia (sensitivity to sound) e.g person prefers lying in dark quiet room

-Classic migraine symptoms are located in frontal region, described as pulsatile or throbbing and usually unilateral, aura is present

-Common migraine symptoms are located fronto, temporal, occipital described as a dull ache but with a stabbing pain in eye (like a bunch of needles) often bilateral, no aura.

-Migraines can sometimes be associated with vertigo and dizziness (further investigation may be required)

-Migraines can also be brought on post exertion (strenuous workout in gym or after sex) symptoms subside in 10-15 minutes

-Fluctuating oestrogen levels are a potent cause of migraines, many women have migraines during menstruation, pregnancy and menopause

***Cervical Myelopathy***

*Definition*

Cervical spondylotic myelopathy is the most common cause of spinal cord dysfunction in older persons. The aging process results in degenerative changes in the cervical spine that in advanced stages can cause compression of the spinal cord. Symptoms often develop insidiously and are characterized by neck stiffness, arm pain, numbness in the hands and stiffness or weakness of the legs with unsteady gait. It may also be characterized by Lhermittes sign- an electric shock like sensation down the centre of the back following flexion of the neck). Cervical myelopathy develops slowly usually from a stenosis, slow degenerative changes usually causing this stenosis, which may be compromised by structurally narrow canals in the first place. It is common in people older than 55 yrs.

*Cause*

Spondylosis or degenerative changes in the spine: Osteophytic spurs develop at respective endplate collapse in an attempt to stabilize spine, they do this by increasing the weight bearing surface of end plates and decrease the forces placed on them. Alongside this the disc calcifies in an attempt to further stabilize the vertebra. Also the ligamentum flavum may stiffen and buckle into the spinal cord dorsally which can cause direct compression of the spinal cord. Also bear in mind that a tumour can act similarly to a degenerative disc. During neck flexion the spinal cord may be stretched over the osteophyte whilst during extension the ligamentum flavum may buckle into the spinal cord. Therefore to recap there is

* Narrowing of the spinal cord via degenerative changes
* Dyamic compression over osteophtes or ligamentum flavum via flexion and extension
* Spinal cord ischemia as blood vessels on the outer cord are disrupted

*Clinical Presentation*

Cervical myelopathy presents as a slow insidious onset. The person may begin with neck stiffness, which is unilateral or bilateral, arm and shoulder pain and possibly stiffness or clumsiness when walking, eventually there may be tingling into the hand or numbness, stiffness in the legs and impaired co-ordination and weakness and clumsiness in the hands, occasionally there is sphincter symptoms such as slight hesitancy on urination

-These symptoms are due to both LMN and UMN signs. There may be nerve root impingement at C5/C6 causing loss of brachioradialis reflex, dermatomal loss of sensation and if severe weakness and muscle wasting in C6 muscles, whilst at C7 there may be a brisk triceps reflex, slight weakness, ankle clonus and positive babinski’s sign

Management

-Neurological assessment- It is important to correlate cervical spondylitic changes with sensorimotor abnormatilites identified on examination to confirm suspicion. If this is the case refer out for MRI CT scan, also need to rule out tumour

-There are important DD’s to consider:

1/ Multiple Sclerosis-

2/ Motor neuron disease (ALS)-The absence of extremity sensory abnormalities should alert the physician to ALS

3/ Spinal cord tumours-intrinsic/extrinsic

4/ Syringomyelia

5/ Subacute degeneration of the spinal cord e.g. Vit B12 deficiency, hereditary spastic paraplegia

**Vertebral Basilar Insufficiency**

*Definition*

Vertebrobasilar insufficiency (VBI) or vertebral basilar ischemia (also called Beauty parlor syndrome (BPS) refers to a temporary set of symptoms due to decreased blood flow in the posterior circulation of the brain. The posterior circulation supplies blood to the medulla, cerebellum, pons, midbrain, thalamus, reticular formation and occipital cortex (responsible for vision). Therefore, the symptoms due to VBI vary according to which portions of the brain experiences significantly decreased blood flow. Any reduction in blood flow can cause a myriad of symptoms. If the blood supply to the brainstem is affected than cranial nerves (vision, hearing, speech) can be affected. If blood supply to the reticular formation is affected then history of drop attacks is noted (reticular formation switches on your conscious cortex and is responsible for extensor tone). If in the cerebellum then ataxia and dizziness can occur. Lesions usually occur from atherosclerotic changes in the vertebral artery or its branches; there are 3 vulnerable areas

1/ The proximal part of the vertebral artery as it begins it’s journey from the subclavian artery (turbulence),

2/ Vertebral artery as it winds around the lateral masses of the atlas (turbulence and tortuous route)

3/ Posterior inferior cerebella artery is the first branch of the vertebral artery. It is an end artery to the lateral medulla with no anastomosis and is therefore more at risk for ischemia (it may also be more prone to damage in neck manipulations). Ischemia of the lateral medulla leads to ‘Wallenberg Syndrome’

In the united states one fourth of strokes and TIA’s occur in the vertebral basilar distribution



*Causes*

-Connective tissue disorders e.g. Marfans, vascultis

-Trauma e.g. whiplash, neck manipulation, falls, sporting injuries (tears in the intima)

-Atherosclerosis (males, smoking, diabetes, hypertension, hypercholestrol) atheroma causing stroke

-Cervical spondylosis (arteries kink more as neck is shortened)

*Clinical Presentation*

*VADN memonic*

-Vertigo is the primary symptom described as a swimming or swaying sensation. As this is CNS vertigo symptoms are long lasting and not transient, cannot be easily suppressed by position

-Vascular pain in the ipsilateral posterior location accompanied with occipital headache

-VII or fascial pain is an onion like distribution

-Bells palsy

-Vomiting (centre is in medulla)

-Ataxia (if cerebellum affected)

-Drop attacks (reticular)

-Diplopia (cranial nerves ischemia-more than one nerve likely to be affected)

-Dyphagia (CN 9/10)

-Nystagmus (cerebellum)

-Nausea/Numbness (facial)

*Clincial features of Wallenberg syndrome*



**Most common form of brainstem ischemia.**

1/ Ataxia (balance and co-ordination)

-Cerebella peduncle becomes ischaemic. The inferior cerebellum peduncle is involved. This gives you ipsilateral ataxia and problems with co-ordination and balance on that side

2/ Vestibular nuclei

-Involvement of the vestibular nuclei causes vertigo, nausea, vomiting and nystagmus. May also cause drop attacks

3/ Descending sympathetics

-Involvement of sympathetics creates an ipsilateral horners syndrome

4/ Nucleus Ambiguus

-The nucleus ambiguus supplies special visceral efferent stimuli to the branchial muscles e.g. larynx, pharynx and soft palate or CN nerves CN9, C10, CN11

Therefore symptoms include dysphagia (difficulty swallowing, dysarthria (slurring of words). Ask patient to stick out tongue and say arrggghh. Hoarse voice

5/ Spinal Trigeminal tract nucleus

-Involvement of CN5 creates ipsilateral absence of pain and temperature

6/ Spinothalamic tract

-Contralateral absence of pain and temperature (neck downwards)

**Thoracic Outlet Syndrome**

*Definition*

Thoracic Outlet syndrome or TOS is a neurovascular condition where there is compression of the neurovascular bundle leading to mixed neurovascular symptoms, or separate neural or vascular symptoms. There are 3 main sites of compression

1/ ‘Supraclavicular lesion’ Between Anterior and middle scalenes near 1st rib brachial plexus and subclavian artery can be compressed

2/ ‘Costoclavicular lesion’. Between first rib posteriorly and the clavicle and subclavius muscle anteriorly

3/ ‘Infraclavicular lesion’. Between pec minor and costo-coracoid membrane (fascia)



*Cause*

* Presence of cervical rib
* Overuse, hypertonia of scalenes (stressed, asthmatic)
* 1st rib dysfunction (1% of population have these and only problematic in 10% of these)
* Clavicle fracture (callus bone formation)
* Poor posture (desk bound)or overtraining of anterior muscles in gym e.g tight subclavius, pec minor, scalene
* Pancoast Tumour
* Elongated C7 TP
* Scoliosis
* Large Breasts
* Sleeping pattern (arms above head)

*Clinical Presentation*

Usually unilateral numbness tingling and parethesia of upper limb and shoulder or constant ache and pain down medial aspect of arm and hand along forearm into ring and little finger. Patient may complain of hand coldness, colour changes may be seen, there may be swelling or arm. Remember if blood flow is impaired then metabolites in blood build up causing diffuse P and N. Adsons test is the key test for TOS. It is a provocative test designed to incriminate where the lesion is .Patient sits down and takes arm palpating the raidial pulse. Patient rotates head to same side as lesion whilst pulse is felt. If there is an absence of pulse or pulse gets fainter then test is positive for scalene involvement. If no change get person to take a big breath in this will elevate the first rib. If there is compression at 1st rib and subclavius you may get a positive test there. If no symptoms, to incriminate pec minor abduct patients arm and traction shoulder. Other test includes Roo’s test.

**Multiple Sclerosis**

*Definition*

Multiple sclerosis (MS) is an immune-mediated inflammatory disease that attacks myelinated axons (white matter) in the central nervous system (CNS), destroying the myelin and the axon in variable degrees.Thus the key areas that get affected are the heavily myelinated areas such as the lateral/anterior column (spinothalmic tract, spinocerebellar tracts, corticospinal, dorsal columns, cerebellar peduncles, optic nerve tract or any areas close to CSF

*Cause*

* Hormonal changes- Pregnancy can be a trigger for MS
* Previous viral infections
* Vitamin D deficiency- Lack of sunlight. People further from equator tend to be effected more
* Stress may affect ns
* Genetics

*Clinical Presentation*

-Person tends to be between 20-40, doesn’t tend to effect elderly population

-Symptoms tend to be relapsing/remitting with very mild symptoms 2x per year, whilst other people have progressive multiple sclerosis where it starts strong and gets worse and worse (males mostly)

-Symptoms affect different part accordingly:

*Optic Nerve dysfunction*

-Blurry hazy, grainy vision as myelin around optic nerve gets damaged. This nerve is surrounded by CSF and meninges. Myelinated fibres from cones being lost affects colour, if in rods then peripheral vision may be lost, ability to judge light and dark, shape affected. Vision may be changed or lost for a few wks but then returns again (suggests that structural damage is limited) myelin repairs (may be put on steroids during inflammatory period). Test: Peripheral vision is usually first to go, PT develops diplopia, if macula affected then loss of colour

*Cerebellum dysfunction-*

-Cerebellum signs include drunken and reeling gait, trunk ataxia, intention tremor, dysarthria, downgoing plantars, pendular reflexes, hypotonia, rebound phenomena, speech changes. Cerebellum symptoms are macrographia, changes in dexterity, patient feels unbalanced, dizzy. Loss of co-ordination and control, patient overshoots target (dysmetria). Test: Rebound phenomena, dysmetria test, rhombergs test

*Dorsal Column involvement*

*-*Dorsal column does advance sensations e.g. fine touch, vibration and position sense. If Fasiculus Gracilus damaged then loss of position sense in legs. Person may have difficulty with gait (stamping gait). Test: Joint Position sense or Rhombergs

*Spinothalamic Tract involvement*

*-*Spinothalamic tract carries in anterior and lateral columns both crude touch, pain and temperature

Patient may mention that that can’t feel hot water on skin or that there cuts don’t hurt. There can also be pain from demyelinating process or secondary postural musculoskeletal compensations from spasticity. Test: Pin prick test to classify area of loss (contralateral side)

UMN’Corticospinal tract

-Corticospinal tract in the anterior column is the major pyramidal descending motor tract (extrapyramidal tracts include reticulospinal, rubrospinal, tectospinal, cerebellaspinal, vestibulospinal. PT with UMN lesion may complain that their legs feel stiff, with lots of spasm, keep tripping up, feel clumsy and out of control. Test: Plantar response will be upwards, Tone: Hypertonia (spasticity), Clonus, postural changes as extrapyramidal tracts affected as well as balance. Attention span, memory, concentration, planning and problem solving all affected

**Motor Neuron Disease**

*Definition*

MND Is a CNS degenerative condition affecting the anterior horn cells in the spinal cord, CN nuclei in the brain stem, and motor cells in the frontal and temporal lobes causing premature apoptosis of cells (axons as a result die off to e.g corticospinal tract). ALS Amyotrophic Lateral Sclerosis is the most common type of motor neuron disease. In its classic form it affects both UMN and LMN.

-Amyotrophy refers to the atrophy of muscle fibers, which are denervated as their corresponding anterior horn cells degenerate (LMN component). Lateral sclerosis (lateral corticospinal tract and corticoreticular spinal tract dying off) refers to hardening of the anterior and lateral columns of the spinal cord as motor neurons in these areas degenerate and are replaced by fibrous astrocytes

-Therefore LMN anterior horn cells degenerate in the spinal cord and brainstem (known as the corticobulbar pathway). The UMN governed by the corticospinal/corticoreticular tract degenerate and die therefore cells in the spinal cord, brain stem and cortex die (premotor, primary motor)

*Cause*

90% are idiopathic. 10% are genetic defined by family history of the disease or test positive for the genetic mutation caused by the disease.

-Environmental factors such as smoking have been linked with ALS

Symptoms usually present between the ages of 50-70 yrs

*Clinical Presentation of ALS*

In ALS you tend to get UMN and LMN signs and symptoms. The first symptoms tend to be in the arms. The LM neurons tend to die in the cervical enlargement. Therefore you get LMN signs (atrophy, reduced tendon reflexes, fasciculations, wasting). You may also get damage to the Corticobulbar tracts that supply the LMN’s in the pons and medulla therefore patient may complain of

-Difficulty swallowing/choking on liquids/foods

-Difficulty speaking/hoarseness, quieting voice

However in the legs you tend to get UMN signs ie. Spasticity (clasp knife type), hyperreflexia, positive Babinski, clonus

As the disease progresses there may be breathing difficulties. Also as the frontal cortex gets damage there may be difficulties with cognition, i.e. difficulty formulating ideas, loosing concentration, and emotional manifestations such as involuntary laughing or crying

The other key finding is there will be no sensory losses because the dorsal columns or the antero lateral ascending columns are not affected so all sensations will be preserved

**Congenital/Developmental**

*Klippelfeil syndrome*

Involves congenital failure of segmentation of cervical vertebrae; results from failure of normal segmentation of cervical somites at 3-8 weeks's gestation; result is multiple fused cervical segments; spectrum of deformity from fusion of 2 vertebrae to involvement of entire C- spine; fusion of **C-2 & C-3** is most common; familial Klippel-Feil-syndrome gene locus on the long arm of chromosome 8. Consistently associated with congenital anomalies of other systems; congenital scoliosis majority require treatment; Sprengels deformity; (33%) Failure of scapula descent . Attached to cervical spine by omovertebral bone or fibrous band

*Clinical Presentation*

-Presentation: Abnormal head position, webbed short neck, Torticollis, restricted Cervical ROM.. Patients with extensive fusions present earlier cosmetic deformity. Instability/Hypermobility at unfused levels. Cord compression in congenitally anomolous, narrow canal in young adults. Other associated anomalies include scoliosis, spina bifida, anomalies of the kidneys and ribs, respiratory problems and heart malformations

-Classified into 3 categories

Type1-Fusion of C2 and C3 with occipitilization of the atlas, adontoid peg hypermobile narrowing spinal cord and brain stem

Type 2- Long fusion below C2 with an abnormal cervical occipital junction

Type3- A single open interspace between 2 segments

*Chiari Malformation*

*Definition*

**A Chiari malformation (sometimes called an Arnold Chiari) means that the lower parts of your brain have been pushed downwards towards your spinal cord, so they are below the entrance to your skull.** Most patients will have a type 1 Chiari malformation, the least serious form of the disease. This is where the lowest part of the back of the brain (the cerebellar tonsils) drops down into the top of the spinal canal. Type 2 and 3 Chiari malformations are less common and more serious. They are associated with spina bifida (a birth defect involving problems with the development of the spine) and adults will also have hydrocephalus (a build-up of fluid in the brain). Usually, the lower parts of the brain are contained in a space within the skull, above the level of the foramen magnum (opening at the base of the skull). If you have a type 1 Chiari malformation, these brain parts are pushed downwards because they are too big for the skull. When parts of the brain are pushed out of the skull towards the spinal cord, this can cause pressure at the base of the brain and block the flow of cerebrospinal fluid (CSF) to and from the brain

*Cause*

Recent studies suggest linkage to chromosomes 9 and 15.It is hypothesized that Chiari type I originates as a disorder of para-axial mesoderm, which subsequently results in formation of a small posterior fossa. The development of the cerebellum within this small compartment results is overcrowding of the posterior fossa, herniation of the cerebellar tonsils, and impaction of the foramen magnum. This theory is consistent with the observed association of Chiari I and other hereditary mesodermal connective tissue disorders, such as Ehlers-Danlos syndrome

*Clinical Presentation*

Symptoms include: headaches, usually at the back of the head – these are brought on, or made worse by, exercise, straining, laughing or bending over, neck pain, dizziness and balance problems numbness or tingling in the arms or legs, blurred vision and involuntary movement of the eyes (nystagmus) swallowing problems, hearing loss and tinnitus. feeling sick and vomiting insomnia depression

**Sprengels Shoulder**

*Definition*

Sprengels shoulder is a congenital elevation of the scapula first described by Eulenburg1 in 1863. It was Sprengel2 who recognized that the deformity was caused by failure of the scapula to descend. The high and rotated scapula causes an ugly cosmetic deformity with a widening of the base of the neck. The superomedial border of the rotated scapula causes a lump at the base of the neck. Decreased abduction may also be noted. This deformity is very often associated with a scoliosis and a Klippel-Feil syndrome, which may exaggerate the deformity. The scapula is usually hypoplastic and rotated so that the inferior pole abuts the thoracic spinous processes. This is usually associated with a very straight clavicle, and an omovertebral bone is present in up to 50 percent of patients and can be palpated as a chondro-osseous bar rather than a fibrous cord in 25 percent of patients. Scapulothoracic movement is further limited by fibrous bands beneath the subscapulars associated with muscular deficiencies. Glenohumeral movement is normal, but scapular rotation is limited such that the glenoid faces down and hence limits abduction. Surgery is used to correct

**Cervical Rib**

*Definition*

A **cervical rib** in humans is an extra rib or fibrous band which arises from the seventh cervical vertebra. Sometimes known as "neck rib”, their presence is a congenital abnormality located above the normal first rib. A cervical rib is present in only about 1 in 500 (0.2%) of people and in even rarer cases, an individual may have two cervical ribs. A cervical rib can sometimes compress subclavian artery/vein and nerves as they pass over the first rib, causing TOS. Most symptoms are relieved conservatively but in the unlucky few surgery may be required to remove rib.

