Cervical radiculopathy*		
• GREEN		
• Intro:	 Compression or impairment of the nerve root, causing px & Ssx that of Px in one or both UL which corresponds to the dermatome of the corresponds to the dermatome of the correspondence weakness & impaired deep tendon reflexes are common due Neck pain is a common issue, up to 40% of work absenteeism attribution of the correspondence o	responding affected nerve to nerve impingement
 Aetiology (risk factors): 	 Conditions causing compression or irritation of spinal nerve root lead In younger pts (30-40s), disc trauma & herniation are most common of In older pts, degenerative changes become more prevalent 50-60s - disc degeneration is most common cause 70s - foramina narrowing due to arthritic change is a frequent cause Cx radiculopathy less frequent than Lx radiculopathy Incidence rate: approx. 85 / 100,000 C7 nerve root most commonly affected, flooded by C6 Risk factors: manual labour w/ heavy lifting, driving, operating vibrating Chronic smoking Hx increases risk of radiculopathies 	causes
Pathophysiology:	 Primarily involves inflammation Inflammation often caused by <i>acute herniation</i> of a Cx disc pressing of a line of the nerve changes, such as osteophyte Direct compression of the nerve root causes px, numbness, tingling, and the nerve root causes px. 	s or disc dehydration, affecting the nerve root
Clinical presentation:	 Pts present w/ radicular px or weakness Inquire: occupational risk factors, Hx of trauma, & px patterns Typically unilateral, but B cases are rare B presentations can complicate physical Dx Cases of trauma or B involvement necessitate advanced imaging for 	accurate Dx
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Cervical radiculopathy* (cont)	
Physical examination:	 Reflexes, compare B Reflexes usually reduced Reduced muscle strength, innervated by the affected nerve (major sign) Spurling test: compresses foramina to Dx radiculopathy (px radiates down ipsilateral side) Cx distraction: in some cases may relieve Ssx
Diagnosis:	 X-rays are first step CT used in traumatic scenarios MRI is the preferred modality Electromyography is useful in confirming dysfunction of the affected nerve
Management:	 Around 85% resolve within 8-12 weeks NSAIDs Cx pillows Acupancture Nerve flossing SMT / STW
• Ddx:	 Brachial plexus injury in sports Cx disc injuries Cx discogenic px s. Cx facet s. Cx spine sprain RC injuries Strain injuries

link text

Pancoast syndrome		
• YELL	ow	
•	- Pancoast s. should be distinguished from Pancoast tumour itself	
Intro:	 Entails: ipsilateral shoulder & arm px, paresthesia, paresis, atrophy of the thenar muscles, & Horner's s. (ptosis, miosis, anhidrosis) 1° bronchogenic carcinoma is the most frequent cause of Pancoast s. 	
	- Manifests as radiating parascapular px, atrophy of intrinsic hand muscles, & a lung apex density w/ localised rib & vertebrae destru-	
	ction	



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Pancoast syndrome (cont)
Aetiology (risk factors):	 - 1° caused by tumours in the superior sulcus of the lung, mostly non-small cell lung cancer (NSCLC) - NSCLC accounts for 80-85% of all lung cancer cases, w/ Pancoast s. making up 3-5% of these - Squamos cell carcinoma used to be most common type of NSCLC associated w/ Pancoast s. - Other malignancies can also cause it - Rarely, being tumours cause it - Lung cancer is 2nd most common cancer & is the leading cause of oncological mortality globally
Pathophysiology:	 Pancoast or superior sulcus tumours cause Pancoast s. Ssx inc. shoulder & arm px due to compression of the brachial plexus Initial Ssx often misDx as MSK Tumour extension can lead to C8-T1 radiculopathy (px & paresthesia of the dermatomes) Weakness of intrinsic hand muscles affects fine motor skills & handgrip Involvement of sympathetic trunk & Cx ganglion can cause facial flushing & sweat Harlequin s. may occur w. contralateral flushing & sweating due to hyperactive sympathetic reaction
Clinical presen- tation:	 Encompasses Ssx related to tumours affecting the lung apex Ssx arise due to brachial plexus & associated structures involvement 1° Ss: shoulder or arm px & paresthesia along the medial half of the 4th & 5th finger, hand, arm, & forearm (C8-T1 radiculopathy) Pulmonary Ssx, e.g. SOB, develop as the tumour progresses to involve more of the lung
Physical examin- ation:	 Ipsilateral facial flushing & sweating due to involvement of sympathetic trunk & Cx ganglion Horner s. (ptosis, miosis, anhidrosis) may also develop w/ further disease



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Pancoast syndrome (cont)		
• Diagnosis:	 - Chest x-ray: initial screening, shows increased size of apical cap or lung mass - CT: provides additional info on tumour extent, satellite nodules, mediastinal adenopathy; crucial for staging - MRI: done after Dx & before surgery to identify vascular, brachial plexus involvement - CT-guided core biopsy: Dx test of choice due to outer tumour location 	
Complicat- ions:	 Surgical: atelectasis (partial lung collapse), px, chest wall deformity, frozen shoulder, CSF leak, prolonged air leak, injury to the brachial plexus Chemotherapy: side effects of the drugs Radiation: alopecia, nausea, vomiting, leathery skin, poor wound healing 	
• Manage- ment:	 Good prognosis: early-stage Dx Poor prognosis: advanced disease, poor performance status, & weight loss Standard care procedure: chemo-radiation followed by surgical resection Contraindication to surgical resection: Presence of mets Involvement of ipsi/contralateral mediastinal nodes or supraclavicular nodes Involvement of VB >50% Involvement of oesophagus &/or trachea Involvement of brachial plexus above T1 nerve root 	
• Ddx:	- Other malignancies either 1°, or even being tumours are known to cause Pancoast s. - Even apical lung infections or abscesses can cause Pancoast s. if they involve the chest wall & surrounding structures	
link text		

Thoracic of	Thoracic outlet syndrome (TOS)*		
• GREEN			
• Intro:	 Encompasses various conditions involving compression of neurovascular structures in the Tx outlet 5 types: venous, arterial, traumatic, true neurogenic, disputed neurogenic 		
	- Tx outlet: 1st rib, scalenes, & clavicles		
	- Imaging helps in Dx		



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Thoracic outlet syndrome (TOS)* (cont)			
Aetiology (risk factors):	 Caused by increased pressure in Tx outlet, often due to anatomical abnormalia (e.g. tumours, cysts), or fibrous muscular bands from overuse Past trauma & neck positioning are common causes, leading to impingement 2° causes: trap deficiency or clavicle #, which can decrease the outlet space & Neurogenic TOS: most prevalent variant, constituting over 90% of cases F>M & individuals w/ poor muscle development or posture Incidence rate: 3-80 / 1000 	of vessels or nerves	
Pathophys- iology:	 Extra ribs from 7th vertebrae are common culprits Extra ribs from 7th vertebrae are common culprits Neck trauma preceded 80% of neurological TOS cases, while 20% were 1° caused by anatomic variants B TOS reported w/ B Cx ribs as 1° cause Soft tissue components (fibrous muscular bands & tumours/cysts), also contribute to TOS Athletes w/ repetitive motions inv. extreme ABD & ER (swimmers) are susceptible to TOS Classic presentation in swimmers inc. px, tightness, or numbness in the neck or shoulder area when their hand enters water Other susceptible athletes: baseball, water polo, & tennis players Clinical Manifests w/ variety of Ssx depending on its cause 		
Clinical presentation:			
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Thoracic outlet syndrome (TOS)* (cont)		
Physical examination:	 Quick overview of pt's posture Check symmetry & ROM of both arms initially Special tests: Neurological exam to evaluate n. compression Brachial plexus compression test Spurling's test Adson maneuver for suspected arterial compression Roo's stress test Costoclavicular test 	
Diagnosis:	 Physical exam 1st, further imaging confirms Dx Chest or Cx x-ray: 1st imaging step, providing crucial anatomical info US only for venous TOS Venous dopplers for detecting compression of subclavian / other veins 	
Complications:	 Rare complications Ischemic change could manifest if vascular compromise occurs Most complications arise from surgical intervention (iatrogenic n. injury, pneumothorax, bleeding complications) 	
Management:	 Excellent prognosis (90% of cases resolve Ssx w/ conservative care) Lifestyle modifications - avoiding repetitive postural stress & workstation modification SMT - Cx, Tx, & 1st rib STW - scalenes & pec minor Exercises phase 1: Cx retractions, ulnar n. floss, scalene stretch, corner pec stretch Exercises phase 2: resisted shoulder retraction Surgery in case of severe compression not responding to conservative care 	
• Ddx:	 Pec minor s. (PMS) - commonly confused w/TOS Brachial plexus injuries Cx spine injuries Cx radiculopathy SIS Elbow or forearm overuse injuries AC joint injury Nondescript px disorders (due to vague nature of TOS Ssx) 	
link text		

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Complex regional pai	n syndrome (CRPS)*
• YELLOW	
• Intro:	 Neuropathic px disorder w/ persistent, disproportionate px beyond typical healing times Ssx inc. sensory, motor, & autonomic abnormalities Often follows trauma, #, or surgery, but spontaneous cases also occur Diagnostic criteria: Budapest criteria 2 types: no nerve trauma & known nerve trauma (clinically indistinguishable, favouring distal extremities)
• Aetiology (risk factors):	 CRPS can occur due to various types or degrees of tissue trauma, inc. even w/o injury or due to prolonged immobilisation Common causes: #, surgery, sprains, contusions, crush injuries, & seemingly minor interventions like intravenous line placement Psychological distress during physical injury may influence the severity & prognosis Incidence varies (higher rates in Netherlands compared to US) F>M, peak incidence 61-70 age group Upper extremities are more frequently involved than lower extremities # are the most common trigger (44-46% of cases) Vasomotor Ssx, e.g. swelling, temperature, & colour changes, are common Dx tests: 3-phase bone scans & autonomic testing Risk factors: asthma, ACE inhibitor use, menopause, osteoporosis, Hx of migraine, & smoking
Pathophysiology:	 Multifactorial mechanisms Inflammatory changes Immunological changes Peripheral sensitisation Central sensitisation & neuroplasticity Autonomic changes
Clinical presen- tation:	 Allodynia: non-painful stimuli causing px Hyperalgesia: exaggerated px from usually painful stimuli vasomotor dysfunction: skin colour & temperature changes Sudomotor dysfunction: swelling & sweating changes Motor Ssx: weakness, reduced ROM, tremor, dystonia in affected extremity
Dubas	

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Complex regional pain syndrome (CRPS)* (cont)				
 Physical e 	xamin Neuropsychologic	- Neuropsychological deficits: executive functioning, memory, word retrieval		
ation:	- Constitutional Ss	- Constitutional Ssx: lethargy, weakness, disruptions in sleep architecture		
	- Cardiopulmonary	- Cardiopulmonary inv .: neurocardiogenic syncope, atypical chest px, chest wall muscle dystonia leading to SOB		
	- Endocrinopathies	: low serum cortisol, hypothyroidism		
	- Urologic dysfunction: increased urinary frequency & urgency, urinary incontinence			
	- GI dysmotility: nausea, vomiting, diarrhoea, constipation, indigestion			
	Psychosocial factor	Psychosocial factors:		
	- Associated w/ worsening depression & anxiety			
- Poor function & diminished quality of life				
	- No specific perso	nality or psychopathology predictors		
- Px-related behaviour & catastrophic thinking in pts w/ sign		our & catastrophic thinking in pts w/ significant	t comorbid psychological burden or poor coping	
mechanisms				
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Complex regional pain syndrome (CRPS) ^{**} (cont)		
• Diagnosis:	 Budapest criteria A. They should report continuing px disproportionate to the inciting event B. They should report at least 1 Ssx in 3/4 following categories: Sensory: reports of hyperalgesia &/or allodynia, Vasomotor: reports of temperature asymmetry &/or skin colour changes &/or skin colour asymmetry, Sudomotor/edema: reports of edema &/or sweating changes &/or sweating asymmetry, Motor/trophic: reports of decreased ROM &/or motor dysfunction (weakness, tremor, dystonia) &/or changes (hair, skin, nails) C. Additionally, they must display at least 1 sign at the time of evaluation in 2 or more of the following categories: Sensory: evidence of hyperalgesia (to pinprick) &/or allodynia (to light touch or deep somatic pressure), Vasomotor/edema: edema &/or sweating changes &/or sweating asymmetry, Sudomotor/edema: edema &/or sweating changes &/or sweating asymmetry, Motor/trophic: evidence of temperature asymmetry &/or skin colour changes &/or asymmetry, Sudomotor/edema: edema &/or sweating changes &/or sweating asymmetry, Motor/trophic: evidence of decreased ROM &/or motor dysfunction (weakness, tremor, dystonia) &/or trophic changes (hair, skin, nails) D. Finally, there is no other Dx that better explains the Ssx & Sx 	
Complicat- ions:	 Dystonia Cognitive executive dysfunction Adrenal insufficiency Gastroparesis IBS 	
• Manage- ment:	 Early treatment may improve prognosis Reported cases of spontaneous improvement Treatment goal: px & discomfort improvement, functional restoration, & disability prevention PT & exercise improve ROM, function & reduce disability through endorphin release Px education NSAIDs / pharmacotherapy Behavioural therapy (related to depression) Invasive interventions 	

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Complex regiona	nal pain syndrome (CRPS)* (cont)	
• Ddx:	 Arterial insufficiency Gillian-Barre s. Hysteria Monometric amyotrophy Multiple sclerosis Peripheral atherosclerotic disease Phlebothrombosis Porphyria Poliomyelitis Tabes dorsalis 	
link text		
Bummer or Sting	nger*	
• YELLOW		
• Intro:	 Common injury in contact sports Reflects upper Cx root or peripheral nerve dysfunction injury Occurs due to over-stretching of upper trunk of brachial plexus or compression of C5/C6 nerve root Recurrences ar frequent & can result in permanent neurological deficits Typically graded as Grade I or Grade II nerve injury 	
Aetiology (risk factors):	 - 1° observed in collision or contact sports (e.g. American football, ice hockey, & rugby) - Affects 50-65% of collegiate American football players - High recurrence rate requires attention to minimise the problem 	
Pathophys- iology:	 3 primary mechanisms: Forceful blow causing depression of shoulder & lateral FX of the neck to the contralateral side, leading to traction of the upper roots of the brachial plexus A direct blow to supraclavicular fossa or Orb's point causing a percussive injury Head forced into hyperEXT, ipsilateral side FX towards trauma side → narrowing of intervertebral foramen at Cx spine, nerve root compression (common in high-level athletes) 	
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Clinical	- Immediate, acute traumatic onset of px/ burning/paresthesia/pins & needles/weakness
presentation:	- Typically presents w/ Ssx circumferentially radiating down the arm
	- Reports recent Hx of trauma to the area
	- Common in young athletes competing in contact sports
	- Previous Hx of burners
Physical examination:	- Shacking of the upper extremity
	- Holding upper extremity close to their body
	- Atrophy or asymmetry in the neck
	- Shoulder depression
	- Atrophy of deltoid or supraspinatus
	- Altered motor patterns when using the shoulder
	- Palpation: tenderness, muscle spasm, vertebral tenderness
	- ROM: possible decrease in neck & shoulder mobility
	- Strength: deltoid (ABD), supraspinatus (ABD - full can), infraspinatus (ER), biceps (elbow FX), pronator teres (forearm
	pronation), triceps (elbow EXT), & ADD digits minimi (ABD of 5th digit)
	- Sensation: burning, paresthesia, pins & needles (usually present circumferentially)
	- Reflexes: triceps & brachioradialis
	- Special tests: Spurling's test & Tinel test (supraclavicular fossa)
Diagnosis:	- Usually through clinical examination & past medical Hx
	- EMG & NCS: able to determine where the lesion is & its severity
	- X-rays: indicate or rule out bone injuries
Manage-	- Length determined by severity of injury
ment:	- For some recovery may take minutes, for other weeks to months
	- Commonly reoccur (up to 87%)

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Bummer or Stinger* (cont)

Ddx:	- Necessary to rule out Cx #, dislocation, or spinal cord injury
	Alternative/associated Cx injuries inc:
	- Assessment & management of concussion
	- Transient quadriplegia - B Ssx
	- Muscular strain/ligament strain - unlikely to have neurological involvement
	- Brachial neuritis - insidious onset
	- Radiculopathy - differences in acute presentation



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