Clinical Weekly - 140th Edition

#JournalTuesday - by Abi Peck

Sham surgery versus labral repair or biceps tendodesis for type 2 SLAP lesions of the shoulder: a three-armed randomised clinical trial

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1. **What is a SLAP lesion?**
2. **What causes a SLAP lesion?**
3. **What is a Bankart lesion and how does this differ?**
4. **What are the most common interventions for this lesion and what does it involve?**
5. **How successful was surgery compared to SHAM for the three groups mentioned in this study?**
6. **Could this study be useful clinically?**

#ClinicalSkillsFriday - by Josh Featherstone

Cranial Nerve 3 – Oculomotor nerve

General anatomy and motor function

The oculomotor nerve is entirely motor in function and therefore provides us with the ability to raise our upper eyelid and move our eyes upwards, downwards, medially and enable constriction of the pupil and for the eye to accommodate on objects.
It is closely integrated with both the 4th and 5th cranial nerve to enable ocular motility and alignment.
It consists of 2 motor nuclei:
1. The main oculomotor nucleus is situated within the mid brain and its nerve cells innervate most of the extrinsic muscles of the eye that enable movement
2. The accessory parasympathetic nucleus receives a stimulus that enables both the accommodation reflex and light reflex to occur.

Diseases of oculomotor function

Infranucleur disorders of oculomotility is marked by abnormal eye movements or paresis of the nerve at levels below the oculomotor nucleus. An array of disorders and diseases can cause this to occur and some are listed below:
-Occupying space lesions such as growths and tumours
-Trauma – if oculomotor paresis follows then neuro-radiological imagining is warranted
-Neurological disorders such as myasthenia gravis
-Subarachnoid damage – as the nerve is situated in the brainstem, it is therefore susceptible to aneurysms and therefore paralysis of the 3rd cranial nerve.

Oculomotor function testing for therapists

With the patient sitting in front of you, ask them to follow your finger with their eyes both upward, downward, medially and laterally without their head moving. Whilst doing so assess the smoothness of eye movement, and speed of eye movement as well as any nystagmus of the eyes when at rest. As mentioned above, the occulomotor nerve also has a parasympathetic supply for the pupil of the eye that are responsible for pupillary actions therefore it is also important to assess pupillary responses, dilation and constriction. If interested on how to test this, then please refer back to the last edition of the weekly in which assessment of pupillary action was covered.

References:

Butler DS (2000) 'The sensitive nervous system' Australia: Noigroup publications
Herzau V (2007) 'Infranuclear disorder of ocular motility' In: Scheifer U, Wilhelm H, Hart W Clinical neuro-ophthamology (137 – 152) Germany: Springer
Snell RS (date) 'The cranial nerve nuclei and their central connections and distributions' In: Snell RS Clinical Neuroanatomy (332 – 370) Philadelphia: Lippincott Williams and Wilkins

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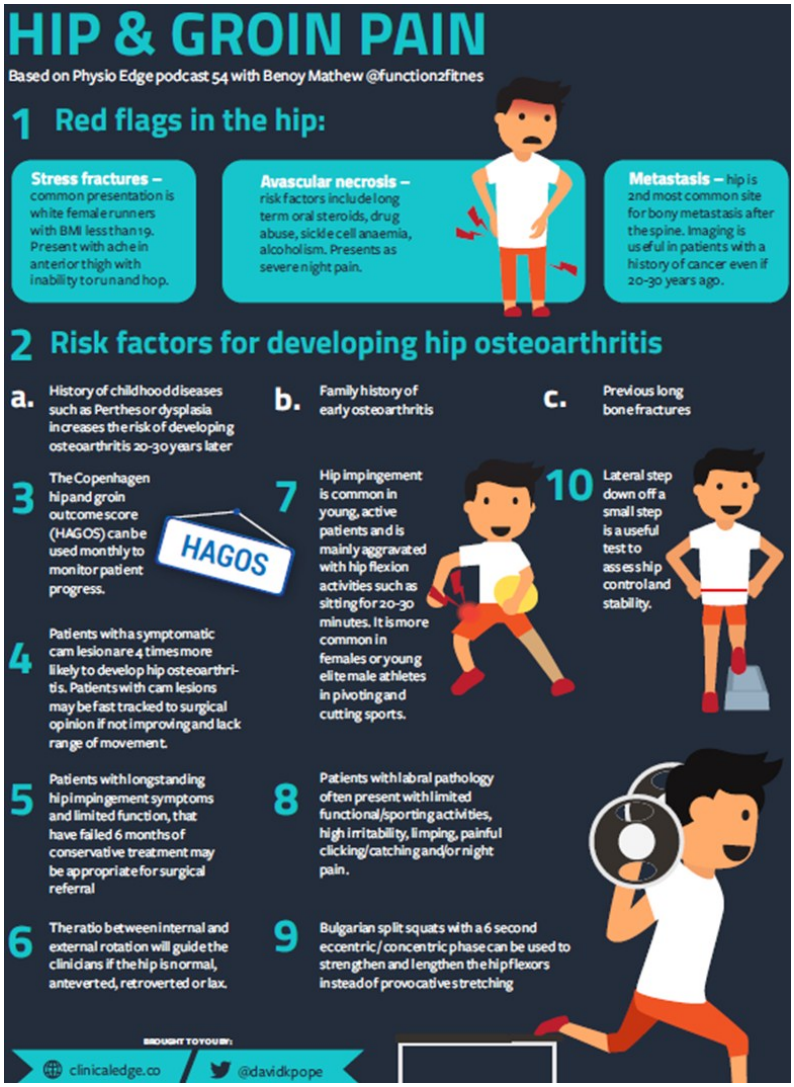
#NewsOfTheWeek - by Liz Wright

1. Hip and groin pain with Benoy Matthew. Free resource with links to webinar and handouts (part 1 episode 53 and 2 episode 54)– simply sign up with your email. Topics discussed in part 2: how you can assess the hip and groin, tests to identify the source of your patient's hip and groin pain, identifying hip joint involvement, how to start hip and groin exercises, when to incorporate hip strengthening exercises, exercise progressions, how to strengthen while you lengthen the hip flexors, when and which plyometric exercises. http://bit.ly/2qWNQtf



2.Neuromechanical deficits 1-2 years after Achilles tendon repair.
Peter Malliaras critiques a recent study from Oda et al. in which the neuromechanical behaviour of the repaired AT 1-2 years after repair was investigated, particularly whether the tendon is more complaint (less stiff) and how this influences the calf muscle activation, force and stretch shorten cycle. The findings indicate that the repaired AT is more compliant and overstretches in the braking phase of hopping (soleus works harder here, possibly because of the increased compliance). Probably as a consequence of greater compliance, the tendon is not able to utilize stored energy for positive work in the propulsive phase (i.e. the stretch shorten cycle). A limitation to consider is that they compared unaffected side vs. affected, and we know that the unaffected may suffer motor output deficits too to some degree. Additionally we don't know what rehab the subjects had and it may not have involved loaded progressive seated and standing calf raises. (Maybe this would help to restore muscle-tendon function, though maybe it would not).
http://bit.ly/2szCYDG

#TWEETOFTHEWEEK- Movement is life…

3.Spotting the Signs: Extra-Articular Manifestations in Rheumatology.
Is rheumatology spoken about frequently enough in the physio world? For many rheumatology conditions there are considerable delays to diagnosis. The British Society for Rheumatology published a policy outlining the importance of early diagnosis and proposed a '12 week window of opportunity' to positively affect a patients care. More recently a national clinical audit (Ledingham et al., 2017) confirmed that most with early inflammatory arthritis wait too long from symptom onset to treatment. Intra-articular features suspicious of synovitis and systemic inflammation (e.g. erythema, heat, oedema, pain, stiffness, restricted ROM) are often noted. However, extra-articular manifestations are often less well known. E.g. psoriasis rashes; IBD; eye inflammation, inflammation of entheses (insertional Achilles etc); nail abnormalities, rheumatoid nodules, anaemia, dry ears +/- mouth. Read the full article to appreciate key points in inflammatory pattern recognition and prevalence rates. http://bit.ly/2szeeeB

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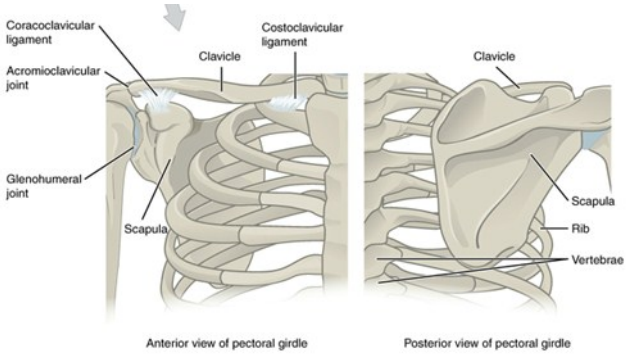
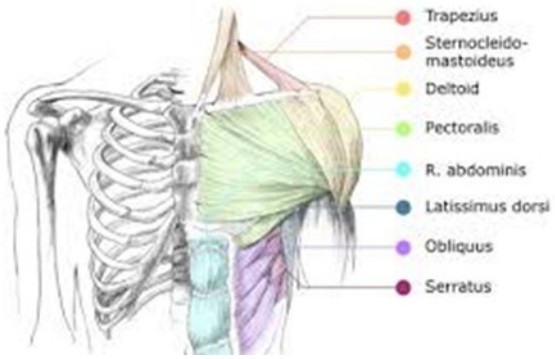
#FractureOfTheWeek by Sam Ackerley

Clavicle Fracture

Accounts for 2-10% of all fractures.
69-82% occur in the mid-shaft or the middle/outer third.
This is due to two factors: firstly this is the thinnest part of the bone, and secondly, it is the only part of the bone not reinforced by attached musculature and ligaments.



Symptoms
Sagging of the shoulder downward and forward
Inability to lift the arm because of pain
A grinding sensation when you try to raise the arm
A deformity or "bump" over the break
Bruising, swelling, and/or tenderness over the collarbone



Articulations
The clavicle articulates with acromion at the acromioclavicular joint laterally and the sternum at the sternoclavicular joint medially.
Attachments
Muscles: pectoralis major, sternocleidomastoid (clavicular head), deltoid, trapezius, subclavius
Ligaments
Acromioclavicular ligament, coracoclavicular ligament, sternoclavicular ligament, costoclavicular ligament.
Blood supply
Nutrient branch from the suprascapular artery

Mechanism of injury
Typically, fractured clavicles occur as the result of a direct blow to the shoulder. Falls onto the shoulder or onto an outstretched arm can cause this.
Population
They are common in very young or very old patients.

Clinical tests
In a clavicle fracture, there is usually an obvious deformity, or "bump," at the fracture site. Gentle pressure over the break site will bring on pain.

Rehab/ Treatment
-Immobilisation and a sling (Collar and cuff) or figure-of-8 dressing while the injury heals.
-Pain medication
-Physiotherapy: maintaining arm ROM and preventing stiffness. Post healing addressing loss in ROM and strength.
Surgery: where there is significant displacement, angulation, shortening (>2 cm) or comminution, internal fixation either with plate-and-screw fixation. Internal fixation is advisable in patients who are at risk of non-union (e.g. elderly).

It is common for clavicle fractures to be displaced due to a combination of the weight of the upper limb pulling the distal fragment down and the sternocleidomastoid pulling the medial fragment upwards.

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