SPECIALTY SUMMARIES

ROUNDUP³⁶⁰

Shoulder & Elbow

Biceps, pressure and instability

The debate surrounding the function of the long head of biceps (LHB) is ongoing, and consequently so is the debate surrounding the treatment of LHB dysfunction. Should we offer patients tenodesis or tenotomy? Without an understanding of the functional role of the tendon it is difficult to establish the best method of treatment. Researchers in London (UK) reasoned that the effects of tenodesis and tenotomy on shoulder stability are not clear. In an attempt to establish what effect these two procedures had, the authors undertook a biomechanical study examining the contribution of intra-articular pressure and the LHB on the stability of the glenohumeral joint. They used a shoulder testing rig capable of testing passive translation in cadaveric shoulders with six degrees of freedom. They tested ten cadaveric shoulders for passive glenohumeral translation in a range of positions with and without joint venting and biceps tendon loading. The biomechanists established the glenohumeral joint to be most lax at 30° of abduction. While venting of the joint increased global instability, the greatest effect was in AP translation (12.5 mm) and supero-inferior translation (7.5 mm). Simulating loading with 20N, the LHB tendon significantly decreased translations in all directions.1 The research team have effectively demonstrated that both negative intra-articular pressure and loading of the LHB

contribute significantly to overall passive stability of the glenohumeral joint. While it is not completely clear to us at 360 what the relevance to clinical decision making is (both tenodesis and tenotomy defunction in the intra-articular component of the tendon) characterising this stabilising function may be useful in patients where glenohumeral instability is an issue.

Chronic acromio-clavicular joint instability

Chronic symptomatic subluxation of the acromio-clavicular joint (ACI) is a very difficult condition to treat and in some ways represents an unsolved surgical problem. There are a number of surgical options including reconstruction (Weaver-Dunn), excision, replacement (Lockdown[™]) and augmentation of the acromioclavicular ligaments. However, as with many new surgical procedures, new complications often arise and newer mini-invasive techniques have resulted, particularly in numerous fractures of the coracoid process. Investigators in Ann Arbor (USA) hypothesised that the orientation of transclavicular-transcoracoid tunnels required for minimimally invasive arthroscopic passage and tendon grafts cannot be reached without significant risk of coracoid cortical breach and subsequent fracture. If this were the case then ligament augmentations in bone sockets would be unable to restore the footprints of the conoid and trapezoid ligaments without risk of coracoid fracture. The researchers employed a novel

study design accessing data from a prospective CT registry. They used the collated data to construct virtual CT models and superimposed these with recognised anatomical tunnel placement. The described transclavicular-transcoracoid tunnel resulted in approximately 7 mm of remaining bone on either side of the tunnel (medial 7.3 ± 1.7 mm, lateral 7.0 ± 1.6 mm). The entry point to anatomic midpoint of the coraco-clavicular ligaments was 9.9 \pm 2.2 mm. Reconstitution of the anatomic ligament position using CT-demonstrated landmarks for the coraco-clavicular ligaments resulted in cortical breach of the coracoid on over 90% of occasions.2 The paper draws attention to the apparently contradictory task of drilling tunnels that are both anatomically located and provide a collinear trajectory that can approximate the coracoclavicular ligaments. We at 360 think this paper highlights the innate difficulties with the transclaviculartranscoracoid tunnel and this proceedure should be approached with caution. We do wonder if looping a graft around the coracoid base is fundamentally a stronger and safer solution although an open approach is of course required.

Chicken or egg: depression and shoulder pain

Unpicking pain, the psychological effects of pain and different experiences of pain as perceived by patients with psychological overtones is something that may never be completely understood. However,

recognition of the psychological aspects of living with pain is something that doctors and surgeons should be more attentive to, particularly in certain subspecialties such as spinal surgery. Researchers in **Daegu** (South Korea) aimed to establish the effects on the psychological wellbeing of patients with three months or more of shoulder pain. They conducted a prospective comparative study (Level II evidence), aiming to establish the prevalence of depression, anxiety, and sleep disturbance in patients with chronic shoulder pain. The outcomes were assessed using scores for pain (VAS), shoulder function (American Shoulder and Elbow Surgeons (ASES) and Korean Shoulder Scale (KSS)), and psychological well-being (Hospital Anxiety and Depression Scale (HADS), and the Pittsburgh Sleep Quality Index (PSQI)). The research team established that the shoulder pain group had significantly higher rates of depression (22.3%), anxiety (19.2%) and sleep disturbance (81.5%) than the normal control group. The authors found no relationship between anxiety, depression and sleep disturbance when subgroup analysis was performed by gender, age, diagnosis or duration of symptoms as increasing VAS for pain score significantly correlated with PSQI scores, and ASES and KSS scores were inversely proportional to the depression and anxiety indices. Interestingly, shoulder pain was the strongest predictor of sleep disturbance.3 Here at 360, we agree with the authors that this study gives a strong association between psychological symptoms and pain. However, it does not establish causation. A larger longitudinal population study is required for this. In other words, we do not yet know if shoulder pain makes you depressed or depression makes shoulder pain worse. We suspect both may in fact be true.

Shoulder replacement and transfusion

In a simple study into transfusion requirements associated with total shoulder replacement, researchers from Tucson (USA) aimed to establish the rate of transfusion in shoulder arthroplasty and examine the risk factors associated with transfusion. The research team performed a retrospective case series review (Level IV evidence) of all patients in their institution who underwent hemiarthroplasty, total shoulder, reverse shoulder and revision shoulder replacements. They used a logistic regression model to establish which clinical variables were associated with risk of transfusion and which were not. The event rate was 7.4% (27 of 339) and unsurprisingly, predictors of blood loss were low pre-operative haemoglobin, high inter-operative blood loss and the use of cemented stems. They found no association with any other factors.⁴ This straightforward study gives a straightforward message: transfusion occurs relatively frequently in shoulder replacement and the risk factors relate directly to blood loss and starting position. Not rocket science, we feel.

Cuff integrity and functionThere is a more complex

relationship between rotator cuff integrity and function than one might first have thought. While small cuff tears are often symptomatic, larger ones may not be. Although much is made of the mid-term results in the literature, there are no longterm series identifying the clinical sequelae of persistent or recurrent cuff tear and function after open repair. A study group from **Helsinki** (Finland) have decided to investigate the problem. They undertook a prospective study to investigate the long-term outcomes of open cuff repair to verify cuff integrity on MRI arthrography and attempt to relate the findings to clinical and functional outcomes. They quantified the rotator cuff status in terms of tendon continuity and fatty infiltration using an MRI arthrography method.

A total of 67 patients who had previously undergone open cuff repair were included in the study with a minimum of 16 years' follow-up. Patients had a mean age of 52 years at the time of surgery. The authors identified a staggering re-tear rate of 94%

(with a mean re-tear size of 3.5 cm × 6 cm), and the remaining four patients had a partial supraspinatus tendon tear. They also identified that cuff arthropathy and secondary tears correlated with both clinical (range of movement) and functional results. Patients with smaller re-tears had better results than those with the larger tears.⁵ Certainly in this series the future hardly looks bright for a 50-something with a degenerate cuff tear. However, it is important to remember that this group of patients had a mean age of 70 years at the time of final follow-up. Here at 360, while we would wholeheartedly agree with the investigators that the best determinant of outcome is re-tear rate, we wouldn't be quite so pessimistic about the outcomes. After all, shoulder arthropathy is fairly common at the age of 70, irrespective of previous injuries.

latropathic plexus injury: the result of clavicular fixation?

Treatment of clavicular fractures is one area where the world can be said to be truly in equipoise; some surgeons and clinicians prefer early fixation for suitable patients while others prefer a 'wait and see' approach. One of the cruxes of the argument from both sides is the balance of functional outcomes and complications with both early and delayed fixation. The current literature suggests a higher complication rate with delayed fixation. However, there is little evidence surrounding the issue of neurological injury in the delayed fixation of clavicular fractures. Researchers in **Stanmore (UK)** draw our attention to the



incidence of iatropathic brachial plexus palsy using a retrospective cohort (Level IV evidence) of patients treated in their institution. The surgeons report a series of 21 patients who have sustained a brachial plexus injury as the result of delayed clavicular fracture fixation over an 11-year-period. In all, 21 cases during exploration of the plexus were found to be adherent to scar tissue on the under surface of the clavicle. Patients typically presented with an injury to the C5/6 nerve roots, upper trunk, lateral cord or suprascapular nerve, and neuropathic pain was pathognomonic in all cases. The majority of patients sustained their injury during fixation between two and four weeks following clavicular fracture. Patients presented with a combination of symptoms; all had some weakness, 90% complained of numbness and just over half of pins and needles, while three quarters complained of pain. All patients were treated with exploration and neurolysis, with 95% having findings consistent with traction injury and a single patient with screws in the plexus. In this

series, neurolysis was successful and improvements were seen in all modalities, although in paraesthesia only 84% of patients had recovered light touch a year later.⁶ Quite sensibly, the authors recommend careful mobilisation of the scar tissue from the underside of the clavicle prior to fixation to reduce the chances of a traction injury from the scar tissue. Early referral to the peripheral nerve injury unit is recommended in cases of acute post-operative palsy. Recognition of plexus palsy as a potential complication of delayed clavicular fracture fixation is important, and to our knowledge here at 360, a similar series has not been published before.

Accuracy of acromioclavicular joint injection

Blind injections in the clinic room are commonplace the world over. The efficacy of these may or may not be limited by their accuracy (there is some support in the rheumatology literature for the practice of intra-muscular rather than intraarticular injections). Orthopaedic clinicians differ in their practice, some preferring blind injections in the clinic room and others wishing to use image-guided injections. A clinical team in New York (USA) designed a cunning little study to establish the accuracy of acromioclavicular joint injections which are commonly performed 'blindly' in the clinic room. They injected their patients blind with a combination of local anaesthetic and radiopaque contrast. Subsequently, radiographs were taken and reviewed by a musculoskeletal radiologist to establish the location of the injection. Injections were classified as intraarticular, partially intra-articular or extra-articular. Under half (43.3%, n = 13) of the injections performed were actually intra-articular with a further 23% partially articular.7 Sadly, one third of patients in this series were completely extraarticular. Despite the subcutaneous location of the joint, it appears as a profession we are probably missing

the bull's eye nearly 60% of the time and missing the target altogether a third of the time.

Tennis not a risk factor for tennis elbow!

■ Lateral epicondylitis (or tennis elbow) is a bit of an enigma. The disease process itself has unclear aetiology and the various treatment modalities have varying reports of efficacy in the literature. A research team in **Derby (UK)** have attempted to shed some light on this poorly understood condition. Using the health improvement database (which is a primary care epidemiology database) and an appropriate statistical method, the authors sought to establish the risk factors for tennis elbow in 4998 patients who were matched on a 1:1 basis for age, gender and treating GP. The median age at onset of tennis elbow was 49 years and multivariant analysis revealed independent risk factors to include rotator cuff pathology (odds ratio (OR) 4.95), De Quervain's tenosynovitis (OR 2.48), carpal tunnel syndrome (OR 1.50), steroid therapy (OR 1.68), and previous smoking history (OR 1.20). Contrary to previous reports in this large series, diabetes mellitus, smoking, trigger finger, rheumatoid arthritis, alcohol intake, and obesity did not have an association with tennis elbow. The authors have performed a large epidemiological study which supports the volume of basic science literature that suggests

that enthesopathies may share a common genetic predisposition and potentially a mechanism mediated through (amongst others) the matrix metalloproteinase pathways.⁸

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