An Inside look at 'Swimmers Shoulder': Antero-superior Internal Impingement (ASII) 'A Cause of 'Swimmer's Shoulder'

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"Swimmers shoulder" is a common and debilitating condition in the elite swimmer. Athletes at the highest level swim for up to 4 hours per day, translating into as many as 1.5 million stroke cycles per arm per year[28]. It is no surprise that the shoulder is the most commonly injured joint in swimmers, with the prevalence in elite swimmers reported to affect at least 1 in 4 and as many as 9 out of 10.[14,44,57] Also, unsurprisingly, shoulder injuries account for the most lost time to training for elite swimmers.[44] A survey conducted in 2013 on 109 of Australia's elite swimmers found that 70% of them had experienced shoulder pain at some time in their career with 28% indicating that it was a recurrent issue, and 21% noting it was ongoing at the time of the survey.[9] Prevalence data dating back to the 1980's does not appear to be showing any reduction in the shoulder injury burden, and to us, this suggests that despite advances in our understanding of this condition, we are missing something in regard to shoulder pain in the elite swimmer. This paper aims to review the current concepts thought to be relevant in 'swimmer's shoulder' and present a discussion that challenges long held beliefs around its etiology, many of which continue to influence our clinical assessment and management decisions. By examining literature from various areas of study, we have derived a hypothesis that we believe warrants further investigation and has the potential to further our understanding of this condition.

Current perspectives

Broadly the proposed etiology of 'swimmers shoulder' has followed the fashion of general shoulder conditions of the day. Kennedy and Hawkins[34] first described the term 'swimmers shoulder' as being synonymous with subacromial impingement. Principally, this referred to mechanical impingement of the supraspinatus and long head of biceps tendon as they pass under the corocoacromial arch. This concept was consistent with the work of Neer[47,48,49] that described the first anatomical theory for subacromial impingement (external impingement), in the general population. Neer described the mechanical impingement of the supraspinatus and long head of biceps against the anterior third of the corocoacromial arch, during shoulder elevation. Rathbun and McNab[53] conducted vascular studies that identified zones of relative avascularity in the supraspinatus and the long head of biceps. Using this research, Kennedy and Hawkins then hypothesized that the relative "avascular zones" of the two tendons made them more susceptible to failure as they were subjected to repeated "wringing out" during the swimming stroke[34]. These two concepts formed the founding principles that continue to be widely accepted in the etiology of 'swimmers shoulder' nearly 50 years later.

More recently, external impingement has been further differentiated into primary and secondary impingement. Primary impingement is described as being the result of local anatomical variations, such as bony osteophytes narrowing the subacromial space. Secondary impingement is theorised to be the result of functional reductions in the subacromial space, and is thought to better account for presentations in the younger athletic populations. Consequently, research over the past 30 years has investigated several factors thought to be implicated in secondary impingement and their potential relationship to shoulder pain in swimmers. Some leading theories include loss of specific shoulder ranges, instability/hypermobility/laxity, scapular dyskinesis and/or altered muscle activity patterns, and rotator cuff strength and/or imbalances. Thus, we have witnessed several iterations of the Kennedy-Hawkins model based on the evidence of the time, but despite this, they remain grounded in the founding assumption that the subacromial structures are the principle source of symptoms in 'swimmers shoulder'. In this paper, we question this central tenet.

The overview of the literature presented below demonstrates that despite a plethora of research into a very common complaint amongst elite swimmers, we still find ourselves with either insufficient evidence or significant debate over the factors thought to contribute to 'swimmers shoulder'. Hill et al[29] conducted a systematic review of the risk factors for shoulder pain and injury in swimmers and found that no studies identified any risk factors predisposing a swimmer to pain and injury with a high degree of certainty. Clinical joint laxity and instability, internal/external rotation range, previous history of pain and competitive level were determined to have a moderate level of certainty, and all other risk factors held a low level of certainty that they predisposed swimmers to pain and injury. In addition, with the overwhelming view that 'swimmer's shoulder' is a result of secondary subacromial impingement, pathology found in structures other than those in the subacromial space have been afforded little or no discussion[1,18,54,57].

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While the subacromial impingement model continues to be accepted as the primary cause of 'swimmers shoulder', our paper will show why the traditional external impingement model is flawed and why internal impingement, and specifically anterior superior internal impingement is a key mechanism leading to pathology in these athletes.

What does the literature tell us?

Range of motion – "too little, too much or somewhere in between?"

To move quickly through a dense medium such as water, swimmers need to produce propulsion throughout as much of the arm stroke as possible whilst maintaining body alignment that minimises drag. As such they need to function in high degrees of shoulder elevation and internal rotation. (Figures 1, 2 & 3)

Figure 1

Freestyle left hand at entry & early catch in high forward elevation

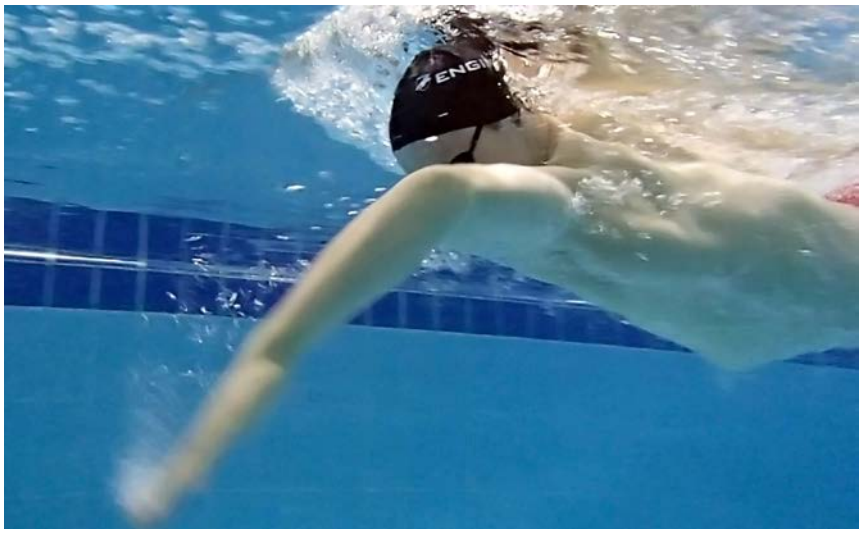

Figure 2

The left arm in early down sweep in high forward elevation and internal rotation, "High Elbow"

Figure 3

Butterfly in early catch showing high forward elevation and internal rotation

Coaches often refer to this as the "high elbow" position – i.e. the elbow is physically higher than the hand in the water. Note that to achieve this, the athlete needs to produce significant amounts of

shoulder internal rotation. Yanai and Hay[71] demonstrated that during a quarter of freestyle stroke time (hand entry, initial catch and early recovery) the shoulder is in positions that are beyond normal physiological range, and described them as positions of external impingement. Loss in specific ranges is therefore thought to increase the potential for impingement during these phases of the swimming stroke. Although this seems a reasonable argument based on the physical requirements of the swimming stroke, papers examining range and its relationship to shoulder pain in swimmers have produced conflicting results. Greipp[24] found a strong association between a lack of shoulder flexibility, specifically horizontal abduction and internal rotation in supine, and the incidence of 'swimmer's shoulder'. Walker et al[67] however, found no relationship to losses in internal rotation, but rather reduced or excessive shoulder external rotation was related to an increased risk of interfering shoulder pain with swimming. While other studies examining various ranges of movement, including internal and external rotation range, have found no correlation between shoulder range and pain in elite swimmers[3,5,30]. Further to these studies, Blanch[7] hypothesised that there may be an ideal window of shoulder range for elite swimmers and that too little or too much range may be a source of increased risk of developing shoulder pain.

Instability/Hyper-flexibility/Laxity.

As previously stated, the ability to swim efficiently requires a significant degree of flexibility. Therefore, it is not so surprising that increased shoulder laxity, or hyper-flexibility, is reported to be a common feature in elite swimmers**.** Zemek and McGee[72] identified that elite swimmers had greater shoulder laxity and increased general joint hypermobility than seen in recreational swimmers, however drawing a cause and effect relationship between increased laxity or hyper-flexibility and shoulder pain in swimmers is not so clear. Instability has been described as a structural or functional deficit that results in pathological or unwanted translation of the humeral head on the glenoid giving rise to pain and/or a compromise in function[6,38]. McMaster proposed that there may be a subgroup of swimmers in which laxity or hyper-flexibility, although considered desirable for performance, exceeds a threshold to become instability resulting in unwanted translation and compromise of the subacromial space[40]. Other researchers have also produced evidence to support this concept[2,55,69]. Interestingly, these studies all utilised manual clinical tests to group subjects with and without instability, which raises some questions over their findings given recent evidence regarding the validity of these tests to accurately diagnose instability[27]. Furthermore, studies in which a known force was applied to the glenohumeral joint via instrument to accurately measure displacement, have failed to establish any relationship between laxity and interfering shoulder pain in swimmers.[10,57,67]. Further doubt is cast over this relationship by the retrospective study conducted by Montgomery et al[46] showing relatively poor return rates for elite swimmers following capsular plication.

Scapula Dyskinesis: often observed, but there's not much evidence for its relationship with pain.

Scapula dyskinesis has been observed to be present in many shoulder conditions. Scovazzo et al[56] performed an EMG study assessing the activity of 12 muscles around the shoulder in swimmers with and without pain and found impaired output in three scapula stabilisers (serratus anterior, upper trapezius and rhomboids) in individuals with shoulder pain. They hypothesised that reduced lateral rotation of the scapula during elevation, resulting in a functional reduction in subacromial space, compromises the cuff tendons and bursa as they pass under the corocoacromial arch. Further EMG work by Wadsworth and Bullock-Saxton[65] found upper and lower trapezius temporal patterns were more variable, and the serratus anterior temporal pattern was more delayed in swimmers with shoulder pain. Additionally, in a recent observational study by Su et al[62] in which 40 swimmers (20 with and 20 without impingement) were examined for scapula kinematics pre and post training, the swimmers with impingement were observed to have significant decreases in scapula upward rotation immediately following training. That said, it is important to acknowledge that these findings do not confirm cause and effect relationships, as such observations could just as easily be the result of pain[35,68]. Furthermore, studies have demonstrated scapula dyskinesis presenting in both asymptomatic and symptomatic shoulders of overhead athletes, and others have observed significant variability in scapula patterns within normal subjects and a high degree of error in measurement of scapula kinesis[11,61]. Despite scapula dyskinesis having undergone significant examination over the past 20 years, its exact role in shoulder dysfunction and specifically 'swimmer's shoulder' is still not clear.

Rotator Cuff Strength and Ratios, conflicting results and are more likely secondary to pain.

Several studies examining shoulder rotation strength have suggested that an increase in internal relative to external rotation strength may predispose a swimmer to shoulder dysfunction and symptoms[4,5,42,55,69]. It is theorised that this imbalance, occurring due to the predominance of internal rotation loading during the swimming stroke, leads to an inability of the rotator cuff to control humeral head positioning, increasing the likelihood of secondary external impingement. Conversely, other research on swimmers with symptomatic shoulders has suggested the exact opposite, reporting deficits in internal rather than external rotation strength[3,63]. These studies have been performed on relatively small numbers and the findings have not been consistent to all age groups examined. Nevertheless, these findings, in combination with more recent research indicating that swimmers with symptomatic shoulders have both reduced internal and/or external rotation strength, raise debate as to the contribution that strength imbalances may have in the development of 'swimmer's shoulder'[9]. Some experienced clinicians argue that strength may not be a relevant factor in swimmers with shoulder pain[7], and therefore it should be recognised that as with scapula dyskinesis, strength changes are likely to be a consequence of pain rather than the cause of it.

Load, not well researched in swimmers, but is likely a significant factor.

Given the high volume of training associated with elite swimming, and the recent increasing attention on load, some literature is now suggesting load is a principle factor in the development of injury. Sein et al[57] demonstrated a relationship between hours swum and mileage per week, and the presence of shoulder tendinopathy. They found 85% of supraspinatus tendinopathy could be predicted in their cohort if subjects swam more than 15 hours or 35 kilometres per week. Contrastingly, Walker et al[67] found no association between average swimming distance per week and shoulder pain in swimmers. Work in other sports has demonstrated that it is rate of change in workload that is likely to play a role in the development of injury[21,31,32]. Percentage change in short-term weekly workloads versus the chronic workload (previous 4 week moving average) was found to be predictive of increased incidence of injury. With this relatively recent understanding, further research to establish its relationship to injury in swimmers is still required.

Supraspinatus and subacromial bursa, not likely the only villains!

Given the predominant view that 'swimmer's shoulder' is the result of secondary subacromial impingement, the sum of the literature leaves us with the overwhelming perspective that the primary pathology occurs in the subacromial structures, namely the supraspinatus tendon and bursa, to the exclusion of almost all other structures[1,7,18,57.] Sein et al[57] reported that 'supraspinatus tendinopathy is the major cause of shoulder pain in elite swimmers', having found it present in 69% of the 52 subjects' MRIs performed in their study. However, there is substantial evidence to suggest that there are other pathologies that may be significant in the symptomatic swimmer's shoulder. McMaster[39] first described damage to the anterior labrum as a painful lesion in swimmers, and this was later supported by the findings of Brushoj et al[12] in which 11 of 18 swimmers undergoing arthroscopy were found to have labral damage. Similarly, Klein et al[37] conducted an MRI investigation on elite water polo players and found changes in the labrum, cuff tendons, long head of biceps and bursa in the non-throwing shoulder. In the paper by Sein, despite the focus on supraspinatus, other findings included those in the bicep tendon, biceps anchor, subscapularis, infraspinatus, and 1 in 5 of shoulders showing labral tears[57]. As the relevance of these anatomical changes is not well understood, they appear to have received little or no discussion. Furthermore, anecdotally we have recently seen several high-profile Australian swimmers retire due to subscapularis, not supraspinatus, failure. This presentation appears to be reflected in recent dynamometry findings, with some athletes demonstrating significant deficits in internal, not external rotation strength as is traditionally accepted, suggesting anterior cuff (subscapularis) and posterior cuff (supraspinatus and infraspinatus) involvement in 'swimmer's shoulder'[9].

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Combined shoulder elevation and internal rotation has been overlooked

As stated earlier, we are of the view that past research into the aetiology of 'swimmers shoulder' has attempted to examine relationships or perspectives that were largely derived from the study of shoulder pain in general populations, to establish if these relationships apply in the context of the elite swimmer. We believe this is fundamentally flawed as it fails to adequately account for the unique movement requirements and loading demands undertaken by the shoulder in this population of athletes.

The one thing common to all swimming strokes, and a prerequisite for swimming fast, is that athletes must repeatedly load the shoulder in high degrees of elevation and internal rotation. There is no other sport that demands tolerance to either the volume or the magnitude of range in this combination of movements. The only other example that comes close is the elite baseball pitcher who performs only a fraction of the movement volume (albeit at very high intensity) but in an almost polar opposite direction to that of swimming, in that baseball pitchers are required to achieve large amounts of external rotation whilst in abduction. We now know that this unique position under load leads to specific articular changes and pathology that is common in elite level throwers. In fact, our understanding of the throwing shoulder has progressed significantly over the past 15 years thanks largely to the observations of both the behaviour of the shoulder in these positions, and the unique articular findings seen in these athletes[13]. Similarly, we believe closer examination of swimming related positions may be useful in developing our understanding into the aetiology of shoulder pain and injury in swimmers.

Yanai and Hay[71] identified two phases of the swimmer's stroke cycle where the shoulder was under most stress and theoretically vulnerable to injury. They referred to these periods as 'positions of subacromial impingement'. The authors noted that as much as 25% of the freestyle stroke time (ST) was spent in positions of impingement; initial catch and early pull (15% of ST), and recovery phase (10% of ST) (figures 4, 5 & 6)

"Combined shoulder elevation and internal rotation has been overlooked"

Figure 4

The left arm is in the catch position, (full forward elevation): As described by Yanai[70] the force of the water acts over the long lever of the arm producing significant torque into further shoulder flexion resulting in a position of impingement.

Figure 5

The left arm is now in early pull: still in a high degree of elevation with increasing internal rotation

Figure 6

The left arm is in early recovery: the shoulder is in abduction and internal rotation, the position Yanai[70] describes as impingement

In initial catch and early pull positions, the force of the water acting on the arm over a long lever, produces large torques into shoulder flexion. They proposed that these torques were sufficient to take the shoulder beyond its normal physiological range of forward elevation and into a position of 'impingement'. Contrastingly, recovery phase, is not subject to these conditions because it is a shorter lever arm, with low load as the arm is clear of the water, and movement is assisted by body roll. Interestingly, in a survey on 233 collegiate swimmers cited by Pink and Tibone[52], 70% of swimmers noted their pain occurring in the first half of the pull phase of freestyle, much like the initial catch and early pull phases described in Yanai and Hay[71]. As such, a closer examination of the anatomical arrangement of the shoulder's tissues in the catch and early pull phases seems warranted.

What do we know about the elevated shoulder position?

Although Yanai and Hay[71] examined the kinematics of the shoulder throughout the swimming stroke and identified initial catch, early pull and recovery as positions of impingement, only one study (Ekman et al unpublished) cited by Pink and Tibone[52] has specifically investigated the anatomical relationships of the shoulder when placed in a swimming specific position. Other relevant studies, looking at swimming like positions, have either examined anatomical relationships with the shoulder in Neer and Hawkins Kennedy positions or investigated specific patient cohorts whose shoulder symptoms were exacerbated when placed in classic Neer and Hawkins Kennedy impingement positions (flexion or flexion and internal rotation). Although these test positions have long been accepted as clinical tests for external subacromial impingement, they also bear striking similarities to the initial catch phase performed during all strokes, particularly freestyle, butterfly and backstroke and early pull phase[52] (figures 7-13). Furthermore, the Hawkins-Kennedy position frequently reproduces shoulder pain in swimmers on assessment[52]. These studies have been cadaver, MRI or arthroscopy in method[22,33,50,60,64].

Figures 7 & 8 - demonstrate the Neer impingement test position. Neer[48] Hawkins and Kennedy[26]
Figures 9 & 10 - highlight the similarities these positions have with hand entry and initial catch, left arm in freestyle and both arms in butterfly.

Figure 7

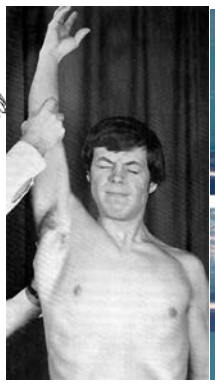
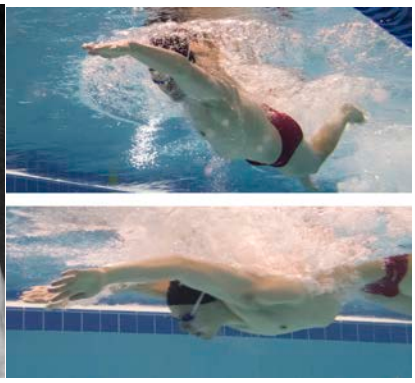


Figure 8



Figures 9

Figure 10

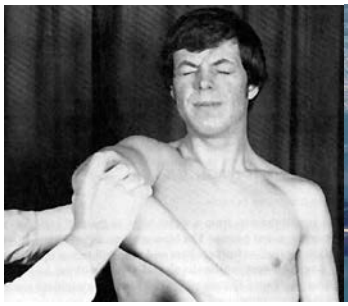


Figure 11

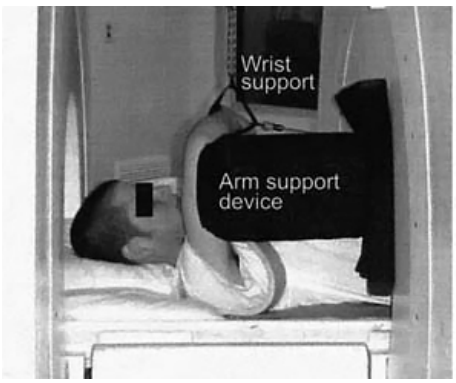
Figure 12

Figure13

Figure 11
Hawkins Kennedy impingement test, Hawkins and Kennedy[26]
Figure 12 & 13
the left arm is in early down sweep in the freestyle stroke, note the elevation is forward of the coronal plane with accompanying internal rotation.

Pink and Tibone[52] cited a cadaver study conducted by Ekman et al (confirmed unpublished by communication) that examined the anatomical relationships of nine cadaver specimens, where the shoulders were placed in the early pull position of the freestyle stroke. They found 5 subjects exhibited bursal and intra-articular contact with the rotator cuff with a further two specimens demonstrating intra-articular contact only. Only two of the subjects with intra-articular contact also exhibited greater tuberosity contact with the acromion. Of those with intra-articular contact, (8 specimens) 5 contacted the anterior superior glenoid and 3 with the posterior superior glenoid.

Pappas et al[50] performed an MRI study looking at the anatomical arrangement of the shoulder's structures in eight subjects placed in both Neer and Hawkins-Kennedy positions, (figures 14 & 15).



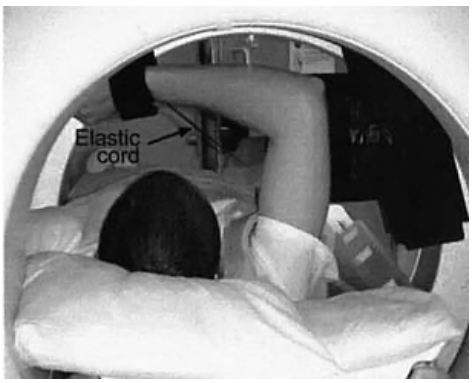
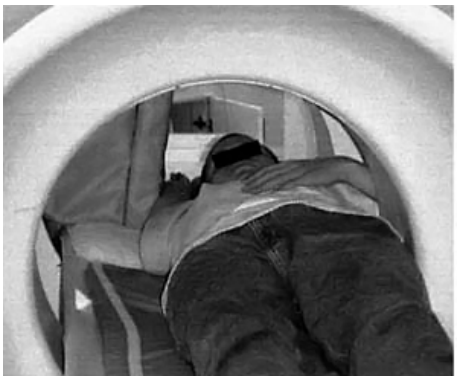


Figure14



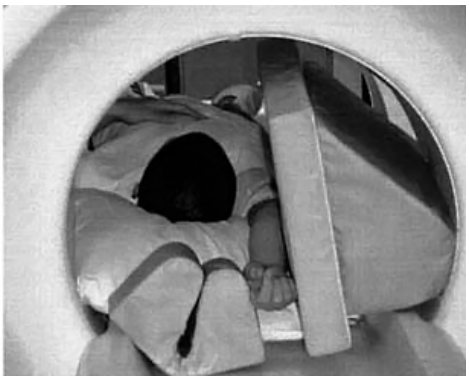
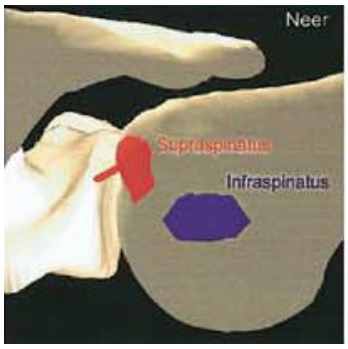


Figure 15

Figure 14 & 15 - Neer and Hawkins Kennedy positions in the MRI. Pappas et al[50]

Subacromial and intra-articular contact was graded, and minimum distances were calculated between tendons and glenoid, and tendons and the underside of the acromion. With the subjects' shoulders placed in the Neer position not one had contact between the cuff and the underside of the acromion. Intra-articular contact between supraspinatus and the posterior-superior glenoid was seen in all shoulders in Neer's positions, and subscapularis contact with the anterior-superior glenoid was seen in 7 of 8 subjects (figures 16 & 18). The Hawkins-Kennedy position resulted all subjects demonstrating subscapularis contact with the anterior glenoid, (figure 19) and with the supraspinatus and infraspinatus contacting the acromion in 7 of 8 and 5 of 8 subjects respectively (figure 17). The Hawkins Kennedy position resulted in significantly greater reductions in subacromial space than did

the Neer position. Both the Neer and Hawkins-Kennedy positions significantly reduced the distance of the subscapularis to the anterior glenoid and the supraspinatus to the posterior-superior glenoid and acromion. They concluded that the extensive intra-articular contact suggested that internal impingement in these positions may play a role in both tests.



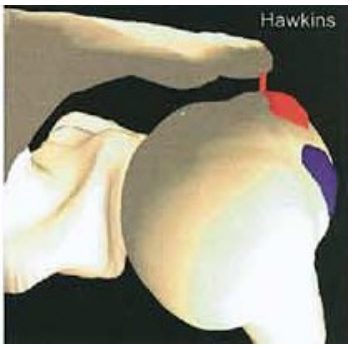


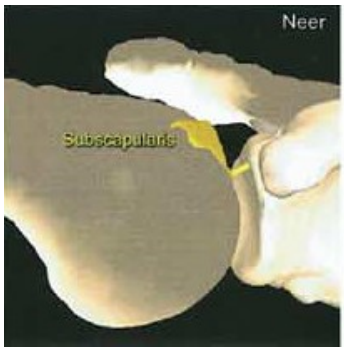
Figure16 **Figures 17**

Figure16

This posterior view of the shoulder, demonstrates the proximity of the supraspinatus footprint to the posterior superior glenoid (in red) in Neer position. Pappas et al[50]

Figure 17

This highlights the proximity of the supraspinatus footprint to the lateral edge of the acromion in Hawkins-Kennedy position. Pappas et al[50]



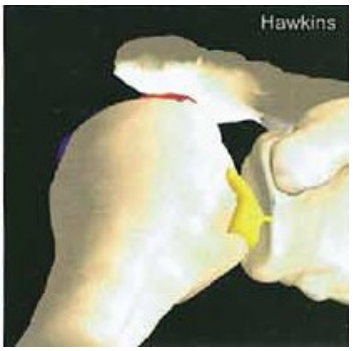


Figure 18 **Figure 19**

Figure 18 & 19

These anterior views of the shoulder demonstrate the proximity of the subscapularis footprint (in yellow) to the anterior and anterior superior glenoid in both the Neer and Hawkins-Kennedy positions. Pappas et al[50]

Jia et al[33] prospectively examined 398 patients with a Neer test, recording the angle of shoulder flexion at which maximum pain occurred. During subsequent arthroscopy, the patient's shoulders were taken through range performing the Neer test in the same manner as the clinical test was performed. Of the 398 patients, 302 demonstrated cuff-glenoid contact and 96 subjects did not demonstrate any internal contact. The angle at which the cuff contacted the labrum intra-operatively was not statistically different to the flexion angle at which the patients noted pain in the clinical preoperative Neer test. They concluded that a positive Neer sign most often corresponds to contact between the cuff and the glenoid. The authors did note that extra articular impingement was not examined in these same positions in this study.

Valadie et al[64] investigated the intra and extra-articular relationships of the rotator cuff during the Neer, and Hawkins-Kennedy impingement tests (n=5 and 4 respectively) using fresh frozen cadaver specimens. All subjects in the Neer position demonstrated soft tissue contact between the articular side of the rotator cuff tendons, and the anterior superior glenoid rim along with the medial acromion. Subjects placed in the Hawkins Kennedy position demonstrated consistent soft tissue contact under

the corocoacromial arch but again more tellingly, all subjects exhibited contact between the articular side of the rotator cuff and the anterior superior glenoid (figures 20 & 21)

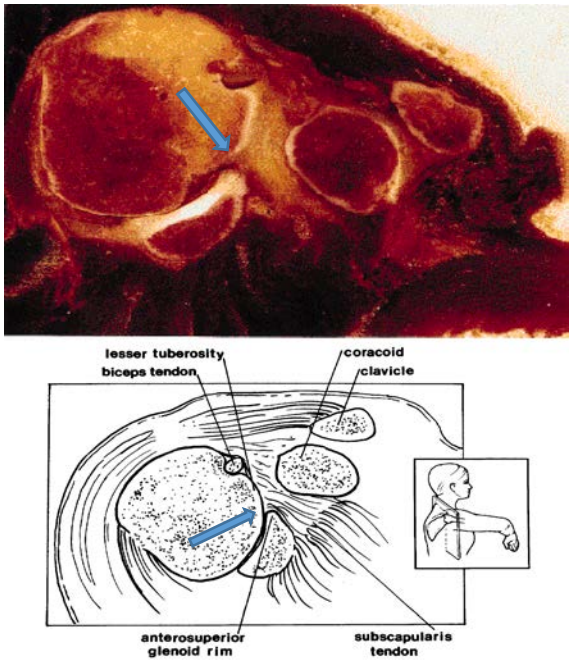


Figure 20

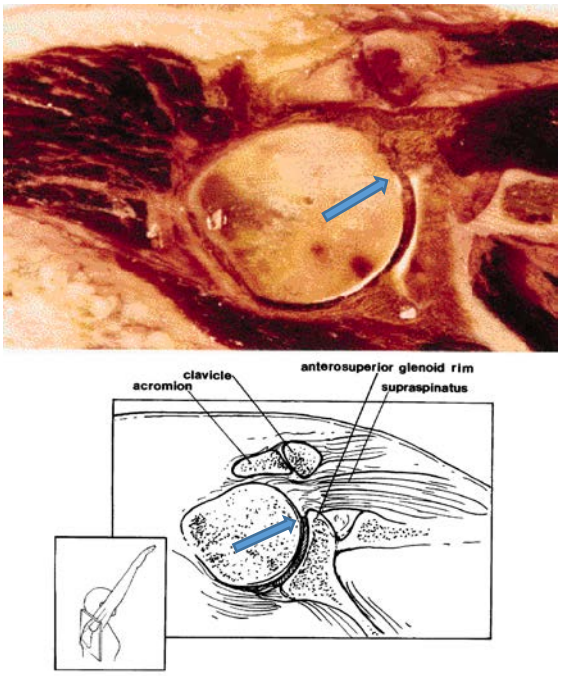


Figure 21

Figure 20 & 21 - The blue arrow highlights contact of the underside of the subscapularis and supraspinatus tendons with the anterior superior glenoid in the Neer and Hawkins-Kennedy positions. Valadie et al[64]

Gerber and Sebasta[22] performed arthroscopic evaluation of 16 patients all of whom had primary unexplained shoulder pain that was provoked by flexion and internal rotation. None of the patients had any signs of instability. Ten of 13 had confirmed partial subscapularis lesions on preoperative MRI investigation. At arthroscopy, isolated lesions of the biceps pulley were noted in 3 subjects, in 10 subjects there was a combination lesion involving the articular side of the subscapularis and the biceps pulley, and 3 subjects had an isolated articular side subscapularis tear. The most painful movement of forward flexion and internal rotation was emulated and evaluated during arthroscopy. In flexion with internal rotation above 90 degrees there was mechanical impingement between the biceps pulley region and the superior most part of the labrum. Below 90 degrees the contact was between the insertion of the subscapularis and the anterior labrum. These researchers concluded that in addition to the internal impingement described by Walch[66], posterior-superior internal impingement (PSII), that a further type of internal impingement, antero-superior internal impingement (ASII) occurs between the deep fibres of subscapularis, the biceps pulley and the anterior-superior labrum when in flexion and internal rotation.

Struhl[60] performed an arthroscopic study on 10 patients with partial undersurface rotator cuff tears and classic clinical signs of impingement (external impingement). At arthroscopy, none of the patients had any evidence of subacromial impingement and when the arm was placed in the

Hawkins-Kennedy position all subjects exhibited contact between the undersurface of the cuff at the site of their tears and the anterior-superior labrum (figures 22 & 23). This group cited the importance of anterior internal impingement as a clinical entity.

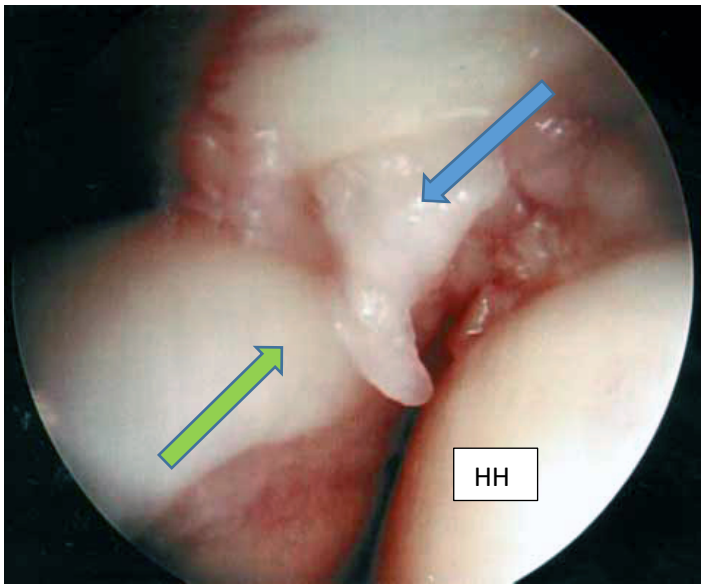


Figure 22

Arthroscopic view of the shoulder from the posterior portal with the shoulder in forward flexion and internal rotation.
Anterior internal impingement is seen occurring between the articular side of the rotator cuff (blue arrow) and the superior labrum (green arrow). HH – Humeral Head. Struhl[60]

Figure 22

Further to these studies, Graichen et al[23] investigated the changes in subacromial space width changes associated with abduction and rotation. They found that beyond 120º of abduction the entire supraspinatus tendon footprint was medial to the region corresponding to the narrowest acromion-humeral dimension, (figures 23 & 24).

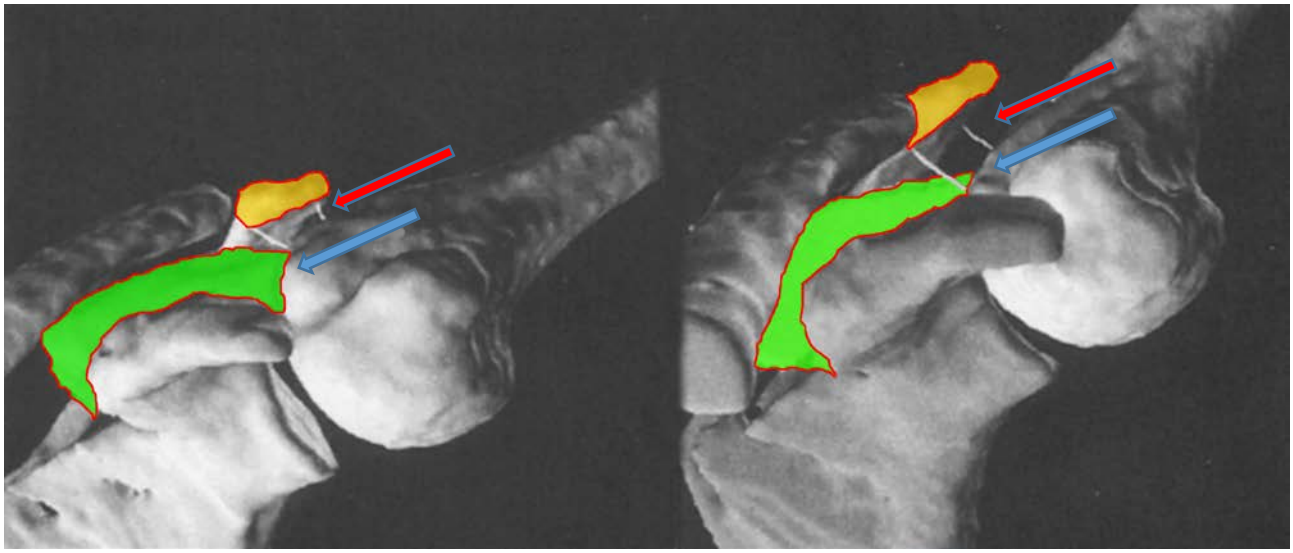


Figure 23 **Figure 24**

Figure 23 & 24 – the blue arrow's show the supraspinatus footprint (green) medial to the acromion and the region corresponding to the narrowest acromio-humeral width (red arrow) at 120° (figure 23) and 150° (figure 24) of abduction. Adapted with Graichen et al[23]

These anatomical studies strongly suggest that extensive intra-articular contact does occur between the supraspinatus, subscapularis, long head of biceps and the anterior superior and posterior

superior labrum (ASII and PSII) during shoulder flexion and internal rotation, movements essential for elite swimming and further, and that external subacromial impingement of the supraspinatus above 120º of elevation is unlikely.

“These anatomical studies strongly suggest that extensive intra-articular contact does occur between the supraspinatus, subscapularis, long head of biceps and the anterior superior and posterior superior labrum (ASII and PSII) during shoulder flexion and internal rotation”

Discussion

A Case for ASII & PSII in Swimmer’s Shoulder

We believe there is still a lack of clarity as to the etiology of shoulder pain in swimmers. This is both through examination of the available evidence to date, as summarized above, as well as our extensive experience managing many elite swimmers. Advances made in the understanding of the throwing shoulder through an examination of the positions unique to this action (abduction and maximum external rotation), led us to believe the application of this approach to ‘swimmer’s shoulder’ would be of significant value. The requirement for repeated high degrees of elevation and internal rotation, movements peculiar to swimming, were the positions chosen for investigation.

The authors have done this and have found sufficient evidence to suggest that not only does PSII occur readily in swimmers, but so does the far less recognized and discussed ASII mechanism. We view ASII in the context of the elite swimmer, as analogous with PSII in throwers. Given the evidence presented above and the intimate anatomical relationship of the supraspinatus, long head of biceps, its pulley, and subscapularis tendons, we argue that repeated articular side contact of all these structures is likely during elevation and internal rotation (as is achieved in elite swimming) and as such ASII and PSII have the potential to drive changes across the entire rotator cuff and interval. This model appears far better placed to account for the mixed presentations that have been observed in some of our elite national level swimmers[9], some of which include tendinopathic change and/or tears in both the anterior cuff (subscapularis and long head of biceps) and posterior cuff (supraspinatus and infraspinatus)[12,37,57]

We believe the internal impingement model plays a far greater role in the etiology of ‘swimmer’s shoulder’ than those mechanisms associated with the external impingement model and that this

mechanism can also account for the intra-articular findings observed in the swimmer's shoulder, namely labral damage and SLAP lesions.

ASII explained

Internal impingement is a well documented phenomenon in overhead athletes with PSII being described as the principle manifestation, attracting the vast majority of discussion since first being described by Walch et al[66]. ASII appears to have been a relative sleeper, attracting far less attention and regarded as relatively uncommon[35]. That said, the work by Habermayer et al[25] investigating lesions of the long head biceps pulley and their relationship to subscapularis, supraspinatus tears and ASII, is a body of work that appears relevant to our examination of 'swimmer's shoulder'. They examined 89 subjects with arthroscopically verified pulley lesions and detailed the classification of 4 types of lesion 1) Isolated supero-glenohumeral ligament (SGHL) change, 2) SGHL and supraspinatus change, 3) SGHL and subscapularis change and 4) SGHL and both supraspinatus and subscapularis change. They established a strong association between ASII and the combined lesion involving LHB, its pulley, subscapularis and supraspinatus. ASII was observed significantly more often in subjects with additional partial articular side subscapularis tears.

Perhaps even more relevant to the swimmers' shoulder is the work by Gerber and Sebasta[22]. They defined ASII of the deep surface of the subscapularis as a form of intra-articular impingement responsible for painful structural disease of the shoulder. 12 of their 16 patients were engaged in manual labour involving regular overhead activity and they concluded that repetitive forceful internal rotation in flexion above the horizontal plane results in impingement between the long head of biceps pulley and subscapularis and the anterior superior glenoid, driving pathological change.

Although Habermayer et al[25] suggests that progressive failure of the cuff structures and the associated biceps pulley leads to ASII, we would suggest the contrary. We argue that elite swimmers are serious overhead workers, moving repetitively under load, in high degrees of forward elevation and internal rotation and that ASII as described by Gerber and Sebasta[22] is the potent driver of anatomical change in the anterior cuff (subscapularis) in elite swimmers.

As has been established with PSII, internal cuff impingement occurring in ASII, is likely to be normal physiological contact. However, in the context of the elite swimmer who performs these movements more than a million times per shoulder per year under load, we hypothesize that it has the potential to precipitate a pathological response.

ASII & PSII, a role in tendinopathy?

In recent times, several papers discussing etiology of tendinopathy have cited the role that mixed loading (articular side contact and intrinsic compression, shear and tensile loading) along with

hypoxic stress may have in the pathogenesis of tendinopathy and tendon injury[8,16,17,45,57,59]. Soslowsky et al[59] demonstrated the devastating effect that overuse tensile loading combined with compression had on rat supraspinatus tendons.

Although the absolute tensile loads borne by the cuff tendons are relatively low next to those experienced by the lower limb tendons, we would suggest that repeated mixed loading in the shoulder during the swimming stroke is far more devastating than the absolute tendon load itself and that ASII and PSII provide a principle source of extrinsic tendon shear and compression.

The shoulders range, along with the rotator cuff's unique anatomical arrangement (interdigitated multilayered collagen matrix) passing over and around the humeral head and into the tuberosities, make mixed loading a likely scenario[15,19]. Our view is that ASII and PSII have the potential to compound the intrinsic tendon compression and differential shear stress that has been hypothesized to occur, at or just proximal to their bony insertions[8,16,19]. This view gains further support by other studies citing the higher incidence of intrasubstance and articular side cuff failure relative to bursal side failure, in overhead athletes and general populations[20,51].

"with a model that acknowledges and accounts for a broader range of pathology, we see the potential to sub classify different manifestations of swimmers' shoulder, leading to more tailored management approaches"

Conclusion

We are not suggesting that external impingement, as previously described in the literature, does not occur at all in elite swimmers, rather that ASII and PSII's contribution to mixed tendon loading, along with elite training volumes, play a far more potent role in the pathogenesis of swimmer's shoulder. We believe that this model readily accounts for the diverse range of pathological change we see in elite 'swimmers shoulder'. Further, with a model that acknowledges and accounts for a broader range of pathology, we see the potential to sub classify different manifestations of swimmers' shoulder, leading to more tailored management approaches.

This discussion paper lays a sound case for a more focused and robust examination of ASII and PSII to determine their role in the etiology of 'swimmer's shoulder'. It is our hope that this can challenge out dated thinking to advance the understanding of 'swimmer's shoulder' and propel further research, and in turn lead to new approaches that improve the management of this very common and challenging injury.

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