AN INSIDE LOOK AT ‘SWIMMER’S SHOULDER’

PART 1: ANTERO-SUPERIOR INTERNAL IMPINGEMENT (ASII) - A CAUSE OF ‘SWIMMER’S SHOULDER’

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'Swimmer’s shoulder' is a common and debilitating condition in the elite swimmer. Athletes at the highest level swim for up to four hours per day, translating into as many as 1.5 million stroke cycles per arm per year. It is therefore no surprise that the shoulder is the most commonly injured joint in swimmers, with a reported prevalence in elite swimmers of at least one in four and as many as nine out of 10. Also, unsurprisingly, shoulder injuries account for the most lost training time for elite swimmers. A 2013 survey of 109 elite Australian swimmers found that 70% 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. Prevalence data does not appear to show any reduction in shoulder injury burden since the 1980s; this suggests that despite advances in our understanding of this condition, we are missing something regarding shoulder pain in the elite swimmer. This paper aims to review the current concepts thought to be significant in swimmer’s shoulder and present a discussion that challenges long-held beliefs around its aetiology, many of which continue to influence 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 develop the current understanding of this condition.

CURRENT PERSPECTIVES

Broadly, the proposed aetiology of swimmer’s shoulder has followed the fashion of general shoulder conditions of the day. Kennedy and Hawkins first described the term ‘swimmer’s shoulder’ as being synonymous with subacromial impingement. Principally, this referred to mechanical impingement of the supraspinatus and long head of the biceps tendon as they pass under the coracoacromial arch. This concept...
While the subacromial impingement model continues to be accepted as the primary cause of swimmer's shoulder, this paper will show why the traditional external impingement model may not be the main underlying cause and why internal impingement, 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?

Elite swimmers need to produce propulsion throughout as much of the arm stroke as possible while maintaining body alignment that minimises drag. As such, they need to function in high degrees of shoulder elevation and internal rotation (Figures 1, 2 and 3).

More recently, external impingement has been further differentiated into primary and secondary impingement. Primary impingement is described as being due to 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 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 of motion, 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 Hawkins-Kennedy model based on the evidence of the time, but despite this, they remain grounded in the assumption that the subacromial structures are the principal source of symptoms in swimmer’s shoulder. In this paper, we question this central tenet.

The overview of the literature presented below shows that despite a plethora of research into a very common complaint among elite swimmers, we still find ourselves with either insufficient evidence or significant debate over the factors thought to contribute to swimmer’s shoulder. Hill et al 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 injury. In addition, with the overwhelming view that swimmer’s shoulder is a result of secondary subacromial impingement, pathology of structures other than those in the subacromial space have been afforded little or no discussion.

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Coaches often refer to this as the ‘high elbow’ position – i.e. the elbow is physically higher than the hand in the water. To achieve this, the shoulder needs to be internally rotated. Yanai and Hay\(^6\) showed that during a quarter of freestyle stroke time (hand entry, initial catch and early recovery) the shoulder is in positions beyond the normal physiological range of motion, describing them as positions of external impingement. Loss of specific ranges of motion 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 of motion and its relationship to shoulder pain in swimmers have produced conflicting results. Greipp\(^17\) 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. Zemek and McGee\(^23\) identified that elite swimmers had greater shoulder laxity and increased general joint hypermobility compared to recreational swimmers, however drawing a cause and effect relationship between increased laxity or hyperflexibility 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\(^24,25\). McMaster proposed that there may be a subgroup of swimmers in which laxity or hyperflexibility, although considered desirable for performance, exceeds a threshold to become instability resulting in unwanted translation and compromise of the subacromial space\(^6\). Other researchers have also produced evidence to support this concept\(^27,29\). Interestingly, these studies all used 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\(^26\). Furthermore, studies in which a known force was applied to the glenohumeral joint via an instrument to accurately measure displacement, have failed to establish any relationship between laxity and interfering shoulder pain in swimmers\(^4,18,31\). Further doubt is cast over this relationship by a retrospective study conducted by Montgomery et al\(^32\) showing relatively poor return rates for elite swimmers following capsular plication. Instability/hyperflexibility/laxity

As previously stated, the ability to swim efficiently requires a significant degree of flexibility. Therefore, it is not surprising that increased shoulder laxity, or hyperflexibility, is reported to be a common feature in elite swimmers. Zemek and McGee\(^23\) identified that elite swimmers had greater shoulder laxity and increased general joint hypermobility compared to recreational swimmers, however drawing a cause and effect relationship between increased laxity or hyperflexibility 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\(^24,25\). McMaster proposed that there may be a subgroup of swimmers in which laxity or hyperflexibility, although considered desirable for performance, exceeds a threshold to become instability resulting in unwanted translation and compromise of the subacromial space\(^6\). Other researchers have also produced evidence to support this concept\(^27,29\). Interestingly, these studies all used 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\(^26\). Furthermore, studies in which a known force was applied to the glenohumeral joint via an instrument to accurately measure displacement, have failed to establish any relationship between laxity and interfering shoulder pain in swimmers\(^4,18,31\). Further doubt is cast over this relationship by a retrospective study conducted by Montgomery et al\(^32\) showing relatively poor return rates for elite swimmers following capsular plication.

Scapular dyskinesis: often observed, but there’s not much evidence for its relationship with pain

Scapular dyskinesis has been observed in many shoulder conditions. Scovazzo et al\(^33\) 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 scapular 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 coracoacromial arch. Further EMG work by Wadsworth and Bullock-Saxton 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 in which 40 swimmers (20 with and 20 without impingement) were examined for scapular kinematics pre- and post-training, the swimmers with impingement were observed to have significantly decreased upward scapular rotation immediately following training. That said, it is important to acknowledge that these findings do not confirm cause and effect relationships, as these observations could just be the result of pain. Furthermore, studies have shown scapular dyskinesis are present in both asymptomatic and symptomatic shoulders of overhead athletes, and significant variability in scapular patterns within normal subjects, and a high degree of error, in measurement of scapular kinesiology have been observed. Despite the significant attention scapular dyskinesis has received 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. 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 shoulder impingement. Conversely, other research on swimmers with symptomatic shoulders has suggested the exact opposite, reporting deficits in internal rather than external rotation strength. 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 reduced internal and/or external rotation strength, raise debate as to how strength imbalances may contribute to the development of swimmer’s shoulder. Some experienced clinicians argue that strength may not be a relevant factor in swimmers with shoulder pain, and therefore it should be recognised that as with scapular dyskinesis, strength changes are likely to be a consequence of pain rather than the cause of it.

**Load is 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 showed a relationship between hours spent swimming 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 found no association between average swimming distance per week and shoulder pain in swimmers. Work in other sports has shown that it is rate of change in workload that is likely to play a role in the development of injury. Sein et al reported findings in the biceps tendon, biceps anchor, subscapularis, infraspinatus, and the one in five shoulders showed labral tears. 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.

With the incidence of shoulder pain in elite swimmers remaining high, we believe this casts serious doubt that our current understanding and subsequent interventions are adequately identifying and/or addressing key causative factors in swimmer’s shoulder.

SUPRASPINATUS AND SUBACROMIAL BURSA LIKELY NOT THE ONLY VILLAINS!

Given the predominant view that swimmer’s shoulder is the result of secondary subacromial impingement, the literature leaves us with the overwhelming perspective that the primary pathology occurs in the subacromial structures, namely the supraspinatus tendon and bursa, and largely excludes almost all other structures. Sein et al reported that “supraspinatus tendinopathy is the major cause of shoulder pain in elite swimmers”, having found it present on MRI in 69% of 52 subjects. However, there is substantial evidence to suggest that there are other pathologies that are significant in symptomatic swimmer’s shoulder. McMaster 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 in which 11 of 18 swimmers undergoing arthroscopy were found to have labral damage. Similarly, Klein et al 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.

The presentations of labral tears and supraspinatus tendon tears are likely a significant factor in swimmers "swimmer's shoulder". Sein et al reported findings in the biceps tendon, biceps anchor, subscapularis, infraspinatus, and the one in five shoulders showed labral tears. 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.

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profiles performed by the shoulder in elite swimmers.

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 while in abduction. We now know that this unique position under load leads to specific articular changes and pathology that is common in elite throwing athletes. 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. 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 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).

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, the recovery phase is not subject to these conditions due to the shorter lever arm, with low load as the arm is clear of the water, and because movement is assisted by body roll. Interestingly, in a survey on 233 collegiate swimmers cited by Pink and Tibone, 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. As such, a closer examination of the anatomical arrangement of the shoulder's tissues in the catch and early pull phases seemed warranted.

WHAT DO WE KNOW ABOUT THE ELEVATED SHOULDER POSITION?

Although Yanai and Hay 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, 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 impingement positions (Neer and Hawkins-Kennedy positions or flexion with 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 (Figures 7 to 13). Furthermore, the Hawkins-Kennedy position frequently reproduces shoulder pain in swimmers on assessment.
Figure 7: The Neer impingement test position, as described by Neer and Walsh.

Figure 8: A similar hand entry and initial catch phase position is seen in the left arm in freestyle and both arms in butterfly.

Figure 9: The Hawkins-Kennedy impingement test.

Figure 10: The left arm in the early down sweep in the freestyle stroke, note the elevation is forward of the coronal plane with accompanying internal rotation.

Figure 11: Neer and Hawkins-Kennedy positions during MRI.

Figure 12: This posterior view of the shoulder demonstrates the proximity of the supraspinatus footprint to the posterior superior glenoid (in red) in the Neer position, and the proximity of the supraspinatus footprint to the lateral edge of the acromion in the Hawkins-Kennedy position.

Figure 13: These anterior views demonstrate the proximity of the subscapularis footprint (in yellow) to the anterior and anterior superior glenoid in both the Neer and Hawkins-Kennedy positions.
Pink and Tibone\textsuperscript{51} cited a cadaver study conducted by Ekman et al (confirmed unpublished by communication) that examined the anatomy of nine cadaver specimens, where the shoulders were placed in the early pull position of the freestyle stroke. They found five 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, (eight specimens), five contacted the anterior superior glenoid and three the posterior superior glenoid.

Pappas et al\textsuperscript{53} 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 and 15).

Subacromial and intra-articular contact was graded and minimum distances were calculated between the tendons and glenoid, as well as the tendons and the underside of the acromion. With the shoulder placed in the Neer position, none of the subjects 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 the Neer position, and subscapularis contact with the anterior superior glenoid was seen in seven of eight subjects (Figure 16). All subjects showed subscapularis contact with the anterior glenoid in the Hawkins-Kennedy position, with the supraspinatus and infraspinatus contacting the acromion in seven of eight and five of eight subjects, respectively.
respectively. The Hawkins-Kennedy position resulted in significantly greater reductions in subacromial space than the Neer position. Both the Neer and Hawkins-Kennedy positions significantly reduced the distance from the subscapularis to the anterior glenoid, and the supraspinatus to the posterior superior glenoid and acromion. They concluded that the extensive intrarticular contact suggested that internal impingement in these positions may play a role in both tests.

Jia et al. 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 the same range, performing the Neer test in the same manner as the clinical test was performed. Of the 398 patients, 302 showed cuff-glenoid contact and 96 subjects did not show any internal contact. The angle at which the cuff contacted the labrum intraoperatively 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. 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 showed 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 showed consistent soft tissue contact under the coracoacromial arch but again, more tellingly, all subjects exhibited contact between the articular side of the rotator cuff and the anterior superior glenoid.

Gerber and Sebastin 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 three subjects; in 10 subjects there was a combination lesion involving the articular side of the subscapularis and the biceps pulley, and three 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 more than 90 degrees there was mechanical impingement between the biceps pulley region and the superior most part of the labrum. At less than 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 postero-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 performed an arthroscopic study on 10 patients with partial undersurface rotator cuff tears and classic clinical signs of impingement (external impingement). None of the patients showed 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. This group cited the importance of anterior internal impingement as a clinical entity.

Scapular dyskinesis: often observed, but there’s not much evidence for its relationship with pain.
Further to these studies, Graichen et al. 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. These anatomical studies strongly suggest that extensive intra-articular contact does occur between the supraspinatus, subscapularis, long head of the biceps, and the anterior superior and posterior superior labrum (ASII and PSII respectively) during shoulder flexion and internal rotation movements essential for elite swimming and further, that external subacromial impingement of the supraspinatus above 120º of elevation is unlikely.

DISCUSSION
A case for ASII and PSII in swimmer’s shoulder

We believe there is still a lack of clarity as to the aetiology of shoulder pain in swimmers. This is both through examination of the available evidence, as summarised above, as well as our extensive experience managing numerous elite swimmers. Advances 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, the movements particular to swimming, were the positions chosen for investigation.

We have found sufficient evidence to suggest that not only does PSII occur readily in swimmers, but so does the far less recognised and discussed ASII mechanism. We view ASII in the context of the elite swimmer, as analogous with PSII in throwing athletes. Given the evidence presented above and the intimate anatomical relationship of the supraspinatus, long head of the biceps and 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, some of which include tendinopathic change and/or tears in both the anterior cuff (subscapularis and long head of the biceps) and posterior cuff (supraspinatus and infraspinatus).

We believe the internal impingement model plays a far greater role in the aetiology 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, and therefore attracting the vast majority of discussion since first being described by Walch et al. ASII appears to have been a relative sleeper, attracting far less attention and regarded as relatively uncommon. That said, work by Habermayer et al. investigating lesions of the long head biceps pulley and their relationship to subscapularis and supraspinatus tears and ASII, appears relevant to our examination of swimmer’s shoulder. They examined 89 subjects with arthroscopically verified pulley lesions and detailed the classification of four types of lesion:
• Isolated supero-glenohumeral ligament (SGHL) change
• SGHL and supraspinatus change
• SGHL and subscapularis change
• SGHL and both supraspinatus and subscapularis change

They established a strong association between ASII and combined lesions involving the long head of the biceps, its pulley, the subscapularis and supraspinatus. ASII was observed significantly more often in subjects with additional partial articular side subscapularis tears.

Perhaps even more relevant to swimmer’s shoulder, Gerber and Sebasta\(^52\) defined ASII of the deep surface of the subscapularis as a form of intra-articular impingement responsible for painful structural disease of the shoulder. Twelve 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 the biceps pulley and subscapularis, and the anterior superior glenoid driving pathological change.

Although Habermayer et al\(^59\) suggest 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\(^52\) is the potent driver of anatomical change in the anterior cuff (subscapularis) in elite swimmers.

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

**ASII and PSII: a role in tendinopathy?**

Several recent papers discussing the aetiology of tendinopathy have cited the role that mixed loading (articular side and intrinsic compression, shear and tensile loading) along with hypoxic stress may have in the pathogenesis of tendinopathy and tendon injury\(^60-64\). Soslowsky et al\(^64\) showed the devastating effect of overuse 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 damaging than the absolute tendon load itself.

ASII and PSII, not subacromial impingement, provide the principle source of extrinsic articular side compression, which has the potential to compound intrinsic tendon compression and differential shear stress that has been hypothesised to occur at or just proximal to their bony insertions\(^60,63,64\). The shoulder’s extensive range of motion, anatomical arrangement of the cuff tendons as they pass over the humeral head into the greater and lesser tuberosities, and their interdigitated multi-layer collagen matrix, make mixed loading a likely scenario\(^65,66\). Furthermore, we know from studies in other overhead athletes and general populations that intrasubstance and articular side partial cuff failure is more common than bursal side failure\(^67,68\).

**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, PSII and 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 changes seen in elite swimmers. 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 paper makes a sound case for a more focused and robust examination of ASII and PSII to determine their role in the aetiology of swimmer’s shoulder. It is our hope that this will challenge outdated approaches, 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.

**References**

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