Shoulder Pain in Swimmers

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Additional information is available at the end of the chapter

1. Introduction

Shoulder pain is the most important symptom that affects competitive swimmers, with a prevalence between 40 – 91% [1-3], and it constitutes a special syndrome called the "swimmer's shoulder".

This syndrome, described by Kennedy and Hawkins in 1974 [3] consists in discomfort after swimming activities in a first step. This may progress to pain during and after training. Finally, the pain affects the progress of the athlete [4]. Some researchers have demonstrated that an important proportion of competitive swimmers have shoulder pain that interferes with training and progress of their abilities. The percentage of athletes with swimmer's shoulder is proportional to the age, the years of practice and the level of competition. Swimmers with interfering pain might not progress in training and thus will not compete as effectively [5].

One of the first reports of this problem was in the 1972 Olympic Games in Munich; Kennedy noticed a high incidence of shoulder pain among swimmers of Canadian group: of 35 competitive swimmers, there were 43 orthopaedic consultations, with 16 specific-related to shoulder (37%), being the most frequent problem [4].

Kennedy had performed a cross-Canada survey involving all competitive swimmers (5000 yards per day). A total of 2496 swimmers were included, reporting a 3% (81 swimmers) shoulder complaints, caused primarily by the freestyle and butterfly strokes and occasionally by the backstroke [4].

2. Epidemiology of shoulder pain in competitive swimmers

The epidemiology of shoulder pain in competitive swimmers has been studied by many researchers. The estimation of prevalence of shoulder pain is very difficult because it is

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related with the subjective experience of pain, memory factors, level of training and the definition of pain considered by the researchers. It is important to establish the difference about the type of evolution of pain (acute, sub-acute, chronic or history of pain) and to differentiate pain of exercise-induced soreness.

As mentioned above, Kennedy et al [4] found a prevalence of 3% of anterior shoulder pain in competitive swimmers. In later surveys, the prevalence has been reported as much higher from 15% to 80% [1].

McMaster and Troup [5] in 1993 performed one of the largest descriptive studies on shoulder pain in competitive swimmers, consisting in a survey questionnaire self administered under classroom-style supervision to a group of 1262 USA swimmers. They included group demographics, training profiles and out-of-water training techniques. They clearly defined the pain as that which interfered with training or progress in training as opposed to post-exercise muscle soreness. Specifically, they questioned about the current experience of pain and the history of pain at any time during the swimming career. With these definitions, the prevalence of history of pain was 71% for male swimmers and 75% for female swimmers. The prevalence of actual pain is less than history of pain (17% in males and 35% in women) [Table 1].

Richardson et al [6] in 1978 performed a survey and physical examination to 137 competitive swimmers. They found a prevalence of history of pain of 52% in "elite" swimmers and 57% in "championship" group (World Champion Team group). In the overall group, a greater percentage of men, as compared to women, complained about shoulder problems (46 vs. 40%). When individual groups were considered, the "elite" women had the greater number of complaints [Table 1].

Bak et al [1] in 1994-1995 season performed detailed interviews and clinical examinations (probably, the most detailed descriptive study of shoulder pain in competitive swimmers) to 36 Danish swimmers. 33 swimmers had unilateral shoulder pain and 13 had bilateral pain. Thirteen swimmers were National Team members (half of the subjects with bilateral complaints were National Team swimmers).

Author - year	Participants (n)	Age	Gender (female - male)	Acute Pain	Sub acute pain (2-week)	History of pain
McMaster et al - 1993	1262	19,5	Not described	9,4 – 35%	Not described	38 – 75%
Richardson et al - 1980	137	14 – 23	83 - 54	Not described	Not described	52 - 57%
Bak et al - 1997	36	17 (12 - 23)	22 - 14	Not described	Not described	91,66%
Contreras et al - 2010	40	17,96 ± 4,11	16 - 24	20%	46,67%	80%

Table 1. Descriptive studies of shoulder pain prevalence in competitive swimmers.

Our research group performed a descriptive study in 2008-2009 [7] to a group of 40 competitive swimmers from the "Universidad de Chile". In our study, the prevalence of history of shoulder pain is 80%. A 20% presented actual pain and the 47% a two week pain. These results are comparable with the international surveys [Table 2].

Years of practice	Meters per day	Weight work hours per week	Stretching time (minutes)	Use of implements	Preferred stroke	Preferred contest
6,07 (3,69)	4716,67 (1297,77)	2,72 (0,96)	7,72 (6,67)	73,33%	Freestyle 73,33%	Sprint 56,67%

Table 2. Training data; the values are expressed in mean (SD) or as percentages.

The survey method for data collection has inherent limitations to correlate cause and effect relationships. But competitive swimmers are very sensitive to their shoulder problems and their ability to effectively train. They have the opportunities to compete against other swimmers and to perform timed trainings.

3. Shoulder Biomechanics in swimming

Swimming requires several different shoulder motions, most being performed during circumduction in clockwise and counter-clockwise directions with varying degrees of internal and external rotation and scapular protraction and retraction [8].

Competitive swimmers used four types of strokes: freestyle or front crawl stroke, breaststroke, backstroke, and butterfly stroke. The fastest, most popular and most widely used stroke for training is the freestyle stroke [9]. The power for this stroke comes 80% from the pull and 20% from the kick [9].

The freestyle stroke pull-cycle can be divided in four phases [10]:

1. Early pull-through: beginning with the hand entry into the water and ending when the humerus is perpendicular to the axis of the torso.
2. Late pull-through: beginning at the completion of early pull-through and ending as the hand leaves the water.
3. Early recovery: beginning at hand exit and ending when the humerus is perpendicular to the water surface.
4. Late recovery: beginning at the completion of early recovery and ending at hand entry.

During the entry and beginning of the pull phases, the glenohumeral joint is in forward flexion, and the humerus is in abduction and internal rotation [9]. During the end of the pull, the joint is extended and the humerus is in adduction and internal rotation [9]. During the recovery period, the arm is in abduction and internal rotation, moving from extension to flexion above the water [9].

The backstroke is considered the complement to the freestyle stroke, and the arm actions involve the same four phases; however, power comes 25% from the kick and 75% from the pull [9].

The butterfly stroke is performed with the arms in the same phase of the stroke at one time. During the entry, both shoulders are flexed, abducted, and internally rotated. During the pull-through phase, the shoulders move into extension, and in the recovery, the arms are brought above the water from extension to flexion while abducted and internally rotated. The power for this stroke comes 30% from the kick and 70% from the pull [9].

The breaststroke has a fifty-fifty split from where the power is initiated. In the pull phase, the arms move into adduction, internally rotated, and are always below the water surface. During the recovery, the arms return in a circular pattern, always under the water surface [9].

In 1991 Marilyn Pink performed the most detailed electromyographic and cinematographic analysis of freestyle stroke [10]. In the pull-through phase, they recognized three different phases: the first phase was reaching forward and gliding. From the point that the hand entered the water to the point of maximal elbow extension, there was no actual pulling. Pulling began after the reach.

Reach began as the hand entered the water (predominance of phasic activity in the upper trapezius, rhomboids, supraspinatus, and the anterior and middle deltoids). The serratus anterior was upwardly rotating and protracting the scapula while the upper trapezius was elevating it and the rhomboids were retracting it.

Therefore, the hand followed an S-shaped curve during the pull-through phase (pectoralis major is the responsible for the initial powerful adduction and extension of the humerus). When the humerus is perpendicular to the body, latissimus dorsi continued the pulling by shoulder extension (internal rotation is given by subscapularis). Also, the serratus anterior was acting to move the body over the arm and through the water and upwardly rotate the scapula to maintain glenohumeral joint congruency. When the latissimus dorsi finished its activity, the posterior deltoid fired to lift the shoulder out of the water.

Finally, in the recovery position (much shorter), the activity noted at the end of pull-through in the middle deltoid and supraspinatus is maintained. The rhomboids fired to retract the scapula.

Pink et al highlighted that the subscapularis and the serratus anterior continually fire above 20% MMT (manual muscle test). Thus, these two muscles would appear to be susceptible to fatigue [10].

4. Etiology

The term ''swimmer's shoulder'' covers a spectrum of consecutive or coexisting pathologies, with rotator cuff–related pain to be the most common finding [11].

Kennedy and Hawkins [4] proposed that the avascularity zones of the supraspinatus and bicipital tendon in the adducted position of the arm are the explanation of swimmer's shoulder. When the shoulders are abducted, all of the vessels of the tendons are almost completely filled. However, when the arm is at the side in the adducted position, there is a

constant area of avascularity extending 1 cm. proximal directly to the point of insertion of the supraspinatus and in the intracapsular portion of the bicipital tendon when it passes over the head of the humerus [4].

Bak reported that the main factor in the development of a swimmer's shoulder seems to be the high training volume during growth in the absence of a well-designed and balanced dryland training program, affecting the muscular balance and the scapular motion [11].

A clear consensus is lacking as to the causes of shoulder pain in swimmers. A general medical assumption has been that swimmer's shoulder is a rotator cuff pathology [12]. Kennedy and Hawkins explain this phenomenon based on the differential vascularity of the supraspinatus and bicipital tendons [4]. Other reports suggest that the impingement is produced by glenohumeral instability or muscular imbalance of the scapular stabilizers (secondary impingement). [9,13,14] Indeed, the muscular electric activity is different in the shoulders with pain during the swimming [10,15].

Essentially, there are various causes or contributor factors accepted to cause shoulder pain in swimmers. The intrinsic mechanism has been defined as a tendon injury that originates within the tendon from direct tendon overload, intrinsic degeneration, or other insult. The extrinsic mechanism has been defined as tendon damage caused by injury of the tendon through compression against surrounding structures, specifically the coracoacromial arch. Among these are: overuse, overload, bony configuration, hypovascularity, muscular imbalance, scapular dyskinesis, joint stability, flexibility, stroke technique, training errors, performance level and coaching factors [9].

Brushøj et al [16] in 2007 reported the arthroscopic findings of 18 competitive swimmers. The most common finding at arthroscopy was labral pathology in 11 (61%) shoulders. Of these, five had signs of posterior superior impingement, two in combination with subacromial impingement. The second most common finding was subacromial impingement (28%). Only two swimmers had isolated inflammation of the bicipital tendon.

4.1. Overuse

The repeated movement of the shoulder can cause micro injury to different structures under risk during swimming. The elite swimmers may log up to 8000-20000 meters per day average using the freestyle arm stroke for most of the distance [9]. At an average of 8-10 arm cycles per 25 meters, a swimmer completes over one million shoulder rotations each week [17]. Richardson and Jobe [6] calculated 396000 strokes per season in male competitive swimmers and declared that it is remarkable to them that an even greater number of shoulder problems do not develop.

Murphy [18] calculated that swimming is equated to running for energy expenditure in a ratio 1:4 in that running 4 miles is equivalent to swimming 1 mile.

This type of training predisposes swimmers to overuse injuries of the shoulder. Consequently, shoulder pain is directly proportional to the age, volume of training, and the ability of the

swimmer (level, training duration and years of practice) [6]. To maintain proficiency, swimming requires a great amount of work. Rest from training quickly translates into detraining [5]. Accordingly, the cause of pain is a combination of overuse and overload [11].

4.2. Impingement and supraspinatus tendinopathy

Swimming involves repetitive overhead movement [19]. Jobe et al [20] hypothesized that repetitive and forceful overhead activity causes a gradual stretching out of the anteroinferior capsuloligamentous structures leading to mild laxity, instability and impingement.

The supraspinatus is the major rotator cuff muscle responsible for securing the humeral head in the glenoid, and its tendon is susceptible to tendinopathy in swimming [19]. The normal tendon appears yellow-white. Microcopically, quiescent rows of tenocytes can be seen interspersed among the compact parallel bundles of collagen fibres. In supraspinatus tendinopathy, the tendon appears grey, dull and oedematous. Microscopically, the tissue appears disrupted and hypercellular with fibroblastic cells in varying states [19].

Sein et al [19] reported in a group of 52 competitive swimmers that were imaged by MRI a 69% supraspinatus tendinopathy with no association between preferred swimming stroke. Tears of the supraspinatus tendon were found in three swimmers: two were reported as having a delaminated intrasubstance tear, and one had a partial 3 mm articular side tear. The bicipital tendon was normal in 46 imaged shoulders (3 unstable bicipital anchor and 3 bicipital sheat effusion) and two had subscapularis tendinopathy. One had infraspinatus tendon thickening, but no change was reported for the teres minor tendon.

Seint et al [19] found that the swimmers' supraspinatus tendon thickness correlated significantly with their level of training, years in training and hours per week in training. Competitive swimmers who trained for more than 15 h/week were twice as likely to have tendinopathy as those who trained less. Similarly, elite athletes who swam more than 35 km/week were four times more likely to have tendinopathy as those who swam fewer kilometres. Also, all swimmers with increased tendon thickness had impingement pain and supraspinatus tendinopathy. In fact, a positive impingement sign correlated strongly with the presence of supraspinatus tendinopathy. A positive impingement sign had 100% sensitivity and 65% specificity for diagnosing supraspinatus tendinopathy and failed to correlate with other shoulder lesions.

Sein et al [19] based in the results of their research, proposed a new model for swimmer's shoulder. In this model, repetitive movement causes tendinopathy with an associated increase in tendon thickness. Tendinopathy leads to pain when the thickened tendon and associated bursa are repeatedly squashed under the bony arch of the acromion during swimming as in impingement testing.

4.3. Scapular dyskinesis, muscular imbalance and secondary impingement

Normal scapular motion is required for adequate shoulder function and to prevent the development of pain [21]. Visible alterations in scapular position and motion patterns have

been termed scapular dyskinesis [22,23]. SICK scapula (Scapular malposition, Inferior medial border prominence, Coracoid pain and malposition, and dysKinesis of scapular movement) is a recently recognized muscular overuse syndrome and it is prevalent in sports like tennis, volleyball and baseball [22,23]. The biomechanics of this disorder is the unbalance of scapular stabilizers generated by the unappropiate overtraining of one arm. In sports like swimming, where both arms are used equally, this pathology must be uncommon, but in competitive swimmers with an overtraining of shoulder muscles, a small asymmetry factor (hand or breath side dominance) can establish a difference between sides. However, this problem is not yet clarified. In our study, we found an important prevalence of asymmetry factors in competitive swimmers [7].

Visually, findings of dyskinesis have been reported as winging or asymmetry [23]. The lateral displacement of the scapula from the thoracic midline has been considered as a marker of scapular dyskinesis [21]. However, clinical measures of scapular position based on side-to-side differences of linear measures have lacked reliability [24]. In the case of the compromise of both scapulas, these methods are less reliable. Our research team recently developed a new technique to evaluate the scapular position and rotation based on digital photography. The exactitude, precision and reliability obtained in the evaluation of this technique accomplished the highest clinical standards [Figure 1].
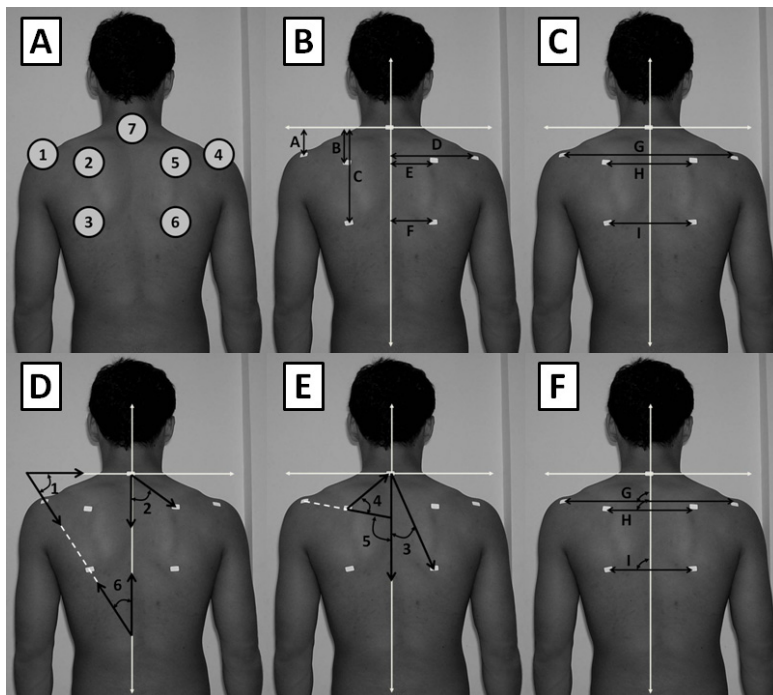

Figure 1. Digital photographic evaluation. (A)Anatomic points (B) Vertical and lateral movements (C) Interscapular distances (D,E,F) Rotation movements.

The relationship of scapular dyskinesis and swimmer's shoulder has been barely researched. Bak et al [1] had evaluated the scapulothoracic instability by observing the scapulohumeral rhythm. They found a severe lack of coordination in 33% of the symptomatic shoulders, compared with 9% of nonsymptomatic shoulders (statistically significant). Crotty et al [25] had evaluated swimmers pre-exercise and post-exercise with Kibler's Test, but there weren't significant differences. Madsen et al [26] evaluated the prevalence of scapular dyskinesis at 4 time intervals during a swim training session (scaption and wall push-up) in seventy-eight competitive swimmers with no history of shoulder pain; scapular dyskinesis was seen in 29 shoulders (37%) after the first time interval (1/4 of a training session), in another 24 (cumulated prevalence, 68%) after one-half of the training session, and in an additional 4 swimmers (cumulated prevalence, 73%) after three-quarters of the training session. During the last quarter of the training session, another 7 had dyskinesis, resulting in a cumulated prevalence of 82%.

The prevalence of asymmetry risk factors and scapular dyskinesis by visual and conventional clinical methods is high in competitive swimmers, considering that this sport presumably uses both scapulas equally. A large group is right-handed, and this correlates with right breathe side predominance, probably because it gives a longer and calmer breathe. This could be an explanation of the important prevalence of scapular asymmetry, because the one-sided movement of the head overuses the elevator muscles of scapula (upper trapezius, rhomboideus and sternocleidomastoideus), raising the risk to develop muscular unbalance. Concordantly, Smith et al [27] found higher electromyographic activity of upper fibers of trapezius than lower in competitive swimmers. However, the association of asymmetry and scapular dyskinesis in the development of shoulder pain is not clearly with this type of methods.

Competitive swimming predisposes to changes in the positioning of the scapula. Our research group found a protraction and lateral movement of the inferior angle of scapula with associated depression on both sides [Figure 2]. Kibler and McCullen [23] suggested that too much protraction will cause impingement as the scapula rotates down and forward. Also, the incapability to elevate the acromion can be a secondary source of impingement. Probably, the overdevelopment of internal rotators muscles (pectoralis major, pectoralis minor and serratus anterior) is the cause of these anatomical changes [7]. Also, scapular stabilizers fatigue reduces motion along two of three scapular axes [28], reducing retraction. This results in protraction and secondary impingement [29], because lower trapezius and serratus anterior muscle fatigue decrease acromial elevation. Su et al [14] suggest that the scapular kinematics of swimmers with shoulder impingement syndrome may not have changed until after they practiced swimming and fatigued the shoulder muscles. Kibler and McMullen [23] recognized this as possible muscular unbalance etiology the directly injured from direct-blow trauma; microtrauma-induced strain, leading to muscle weakness; become fatigued from repetitive tensile use; or are inhibited by painful conditions around the shoulder. In fact, they consider that the serratus anterior and the lower trapezius muscles, pivotal muscles in swimming, are the most susceptible to the effect of the inhibition.
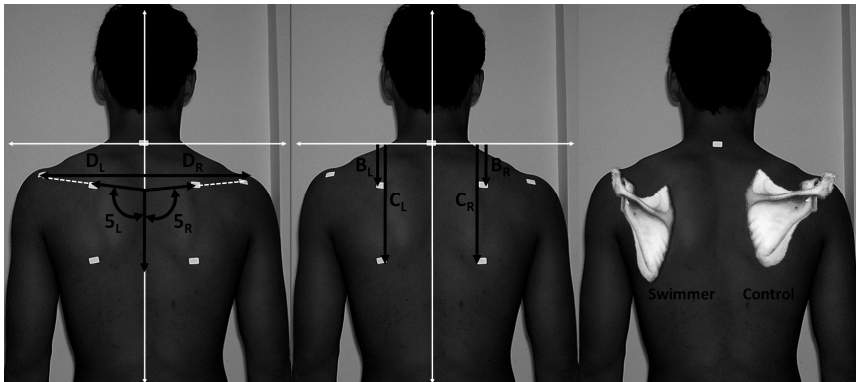
Figure 2. Significant differences between distances and angles of swimmers compared with control group.

Remarkable are the modifications in the positioning of the scapula in swimmers with shoulder pain. In our research, we have found differences in competitive swimmmers with control group and opposite variations in swimmers with and without shoulder pain. We found an elevation of both scapulas associated with retraction. Loss of protraction creates functional anteversion of the glenoid. This increases the degree of impingement between the posterior superior glenoid and posterior rotator cuff by moving the posterior aspect of the glenoid closer to the externally rotated and horizontally abducted arm [23]. Our data and other research suggest an association between scapular malposition and malrotation and swimmer's shoulder [Figure 3]. Pain has been shown to alter propioceptive input from Golgi tendon organs and muscle spindles, predisposing to muscular unbalance [23].
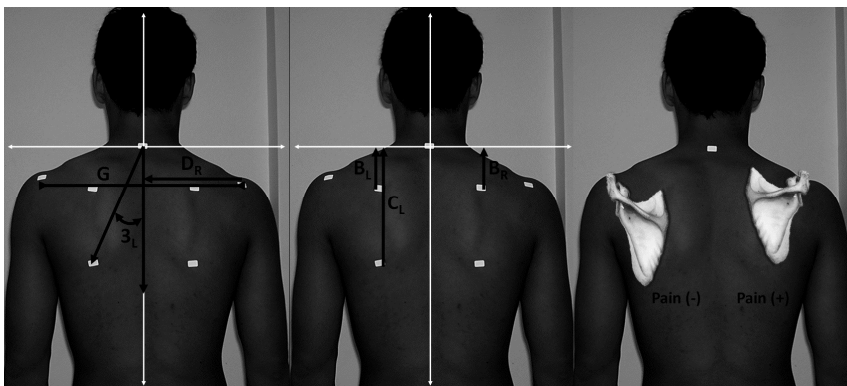

Figure 3. Significant differences between distances and angles of swimmers with pain compared with swimmers without pain.

4.4. Shoulder instability and range of motion

The primary stabilizer of the shoulder joint is the capsulolabral complex (static stabilizer). The rotator cuff muscles function dynamically as secondary stabilizers by contracting in a

coordinated and synergistic fashion to contain the humeral head throughout abduction. The deltoid functions in a force-couple with the internal rotator and external rotator muscles to maintain the humeral head centered in the glenoid during arm elevation [30].

Imbalances of the rotator musculature, excess capsular laxity, or loss of capsular flexibility, have all been implicated as etiologic factors in both glenohumeral instability and impingement syndrome [30].

Warner et al [30] prospectively evaluated 53 subjects: 15 asymptomatic volunteers, 28 patients with glenohumeral instability, and 10 patients with impingement syndrome. They found that impingement syndrome is associated with posterior capsular tightness and a relative weakness of the external rotators and that anterior instability is associated with the findings of excessive external rotation, and a relative weakness of the internal rotators.

Bak and Magnusson [31] examined fifteen competitive swimmers allocated into two groups. The first group consisted of seven swimmers with unilateral shoulder pain related to swimming and the control group consisted of eight swimmers with no present or previous history of shoulder pain. They found that internal range of motion was reduced in painful shoulders compared with pain-free swimmers, although without significance. No differences in external range of motion were detected.

McMaster et al [32] evaluated shoulder laxity and interfering pain in competitive swimmers. The total study group of swimmers represented 80 shoulders at risk for possible pain. Fourteen swimmers (35%) noted significant interfering shoulder pain at the time of the assessment. Clinical examination assessed the sulcus sign and anterior and posterior manual provocation tests in the sitting and recumbent positions. The statistical analysis revealed a positive correlation at the 95% confidence level between the clinical examination score and the presence of interfering shoulder pain. McMaster proposed that the shoulder laxity may be a common denominator in the causation of significant interfering shoulder pain in the swimming athlete [32].

Burkhart et al [33] proposed that the essential lesion that affects these athletes is an acquired loss of internal rotation resulting from tightness of the posteroinferior capsule. They called it glenohumeral internal rotation deficit (GIRD) and defined it as "the loss in degrees of glenohumeral internal rotation of the dominant shoulder compared with the nondominant shoulder." Torres and Gomes [34] found that competitive swimmers mean GIRD was 12 degrees ± 6.8 degrees.

The main finding of our study is the decrease in the range of internal and external rotation of the glenohumeral joint of competitive swimmers compared to a healthy control group [Table 3]. This could be explained by repeated microtrauma in the soft tissues, which can ultimately lead to failure of the supporting structures. In fact, it has been shown that alterations in the glenohumeral rotation range, in addition to the continuous and ongoing training an elite athlete, can be modified with only one training season. In female athletes, there was a significant decrease in internal rotation after one season [35].

Variable	Competitive	Control group
IR Right	62,47 ± 12,4*	73,2 ± 9,74*
IR Left	67,3 ± 12,36*	76,87 ± 12,03*
ER Right	86,47 ± 14,72*	105,6 ± 10,24*
ER Left	84,67 ± 13,8*	107,2 ± 12,13*

Table 3. Glenohumeral rotation. The values are expressed in mean ± SD. * p < 0,05.

However, the etiologic impact of shoulder instability always has been discussed. Borsa et al [36] evaluated with ultrasound the glenohumeral joint displacement under stressed and non-stressed conditions in 42 competitive swimmers. They were unable to identify significantly greater glenohumeral joint displacement in elite swimmers compared to controls, and elite swimmers with a history of shoulder pain were not found to have significantly more glenohumeral joint displacement compared to swimmers without a history of shoulder pain.

4.5. Impingement, overuse, scapular dyskinesis, shoulder instability and supraspinatus tendinopathy: Biomechanical and molecular pathways to explain "swimmer's shoulder".

The biomechanics of the glenohumeral joint is the most complex and least understood of all joints. Allows range of motion than any other joint can be achieved, but with a cost: instability. Throughout this chapter, has given extensive and relevant evidence about the delicate muscular balance and the impact of instability on optimum performance of this joint. However, we face a difficult problem: swimming. This sport represents a challenge to both the glenohumeral joint stability and muscle balance, but certainly the most important problem is the overuse.

In the past ten years, many studies have tried to elucidate this problem, using rat models of supraspinatus muscle overuse, which is the most injured muscle in this sport. This animal model has been used to evaluate the role of intrinsic injury factors (acute insult) and extrinsic injury factors (subacromial impingement) on rotator cuff injury. The overuse exercise rat model consisted of treadmill running at 17 meters per minute, at a 10° decline, for 1 hour per day, 5 days per week, resulting that approximately 7500 strides per day is consistent with the number of strokes an competitive swimmer may take during a typical training protocol [37,38].

Soslowsky and Carpenter [37] with the use of a rat model, designed one of the first studies to elucidate the pathophysiology of the effect of overuse in the supraspinatus muscle. They measured the effects of an overuse running regimen on 36 rats after 4, 8, or 16 weeks of exercise and compared them with a control group who were allowed normal cage activity.

Histologically, cellularity was increased, and cell shape changed from elongated spindle shaped cells to more rounded plump cells; collagen fibers in the overuse groups were less aligned with respect to the longitudinal axis of the tendon.

Geometrically, cross-sectional area was significantly greater and continued to rise over time. The cross-sectional area increased significantly between 4 weeks and 16 weeks.

In a previous research, Carpenter et al [38] evaluated intrinsic (acute injury: bacterial collagenase) and extrinsic factors (impingement: Achilles tendon allograft was passed underneath and wrapped around the acromion) with overuse of supraspinatus tendon in the same model. The supraspinatus tendons which were subjected to a combination (intrinsic or extrinsic factor) plus overuse, exhibited an increase in histologic grade compared with overuse alone. Also, the shoulders that received the combination of alterations had an increased supraspinatus tendon area with respect to the contralateral overuse-alone tendons. This study demonstrates that detrimental changes in the supraspinatus tendon can be incited by combinations of overuse and intrinsic injury, overuse and extrinsic compression, and overuse alone.

Changes in cell shape, organization of collagen and cross-sectional area are the result of the activation of molecular pathways in response to the mechanical stress generated by overuse.

Several studies have addressed the major biochemical changes in tendon matrix composition in human tendinopathy [39]. Predominantly consisting of collagen type I (95%), there are many other matrix constituents (proteoglycans and noncollagen glycoproteins). The called ''minor'' collagens are implicated in a number of important processes including collagen fibril formation, regulating the ultimate diameter of the fibrils and mediating interactions with the surrounding cells and matrix.

Riley et al [40] shown that degenerate tendons have a small but significant reduction in the total collagen content relative to the tissue dry weight. This was partly because of an increase in the non-collagen glycoprotein content, as well as increases in matrix proteoglycan [40]. The type and distribution of collagen also changed, with an increase in the proportion of type III collagen, which was found associated with the type I collagen fibril bundles, thought to be intercalated into the fibrils, suggesting that the original fibrils had been extensively remodelled, resulting in a greater proportion of small diameter and randomly organized fibrils [39]. In tendinopathy, also there is a generalized increase in sulfated glycosaminoglycan, the majority of which was chondroitin sulfate [39]. Archambault et al [41] in the same rat overuse model found that supraspinatus tendon had increased expression of well-known cartilage genes such as Col2a1, Aggrecan and Sox9.

The tenocyte matrix equilibrium is regulated by the interaction of matrix-degrading matrix metalloproteinases (MMP) and tissue inhibitors of metalloproteinases (TIMPs) [39]. Most studies have focused on collagen degradation occurring in the extracellular environment mediated by MMP. Thornton et al [42] exposed a rat model to intermittent cyclic hydrostatic compression (to simulate impingement injury). Levels of MMP-13, MMP-3 and TIMP-2 mRNA were evaluated, finding increased expression of MMP-13 in the supraspinatus tendon. Ruptured human supraspinatus tendon have been demonstrated to have increased MMP-13 mRNA expression [43]; the unique upregulation of MMP-13 mRNA levels may be related to matrix turnover and, as such, could support the impingement injury theory for rotator cuff tendinopathy [42].

The balance of production and destruction found in the tendon matrix has been widely studied. However, as the mechanical stress is converted into biochemical signals that ultimately produce an imbalance at the level of MMP/TIMPs has been studied less. Szomor et al [44] evaluated the regulation of NOS (nitric oxide synthase), a potent regulator of tendon degeneration and healing. With the same animal model of supraspinatus tendon overuse, they found that the mRNA expression of all three NOS isoforms (inducible, endothelial and neuronal) increased in the supraspinatus tendons as a result of overuse exercise. Nitric oxide (NO) is a diatomic, highly reactive, free radical; high levels are often associated with degradative processes, including modulation of the activation of metalloproteinase enzymes, cytotoxicity (apoptosis, tenocyte death) and induction of pro-inflammatory cytokines [44].

De Castro Pochini et al [45] studied the effect of overuse with the same model of rat supraspinatus over the mechanoreceptors. On histologic evaluation, they found a typical response to overuse (cellularity was increased and cell shape changed from elongated spindle-shaped cells to more rounded plump cells). Supraspinatus tendons also were evaluated with immunohistochemistry using S100 protein antibodies, finding that the group of rats that ran showed significantly higher expression of proprioceptors than the group of rats that were not subjected to physical activity. They declared that the increase of mechanoreceptors for sure may not be indicated on increase of proprioception but is rather an indication of different pattern of tendon receptors following overuse physical activities.

It is important to consider, in addition to the response of the extracellular matrix, cell response to overuse. Research has found an imbalance between proliferation and apoptosis [46,47]. Scott et al [48] using the rat overuse model, found that tendinosis was present after 12 weeks of downhill running and was characterized by tenocyte rounding and proliferation, glycosaminoglycan accumulation and collagen fragmentation. His research group found a correlation between the proliferation index in tenocytes and local IGF-1 expression and phosphorylation of IRS-1 and ERK-1/2.

Apoptosis or programmed cell death is mediated by the activation of caspases (cysteine-containing aspartate proteases) and is involved in the stress-induced cascade of tendinopathy [49]. Yuan et al [49] studied the levels of apoptosis at the edges of torn supraspinatus rotator cuff tendons from patients with rotator cuff tear. Apoptosis was detected by in situ DNA end labelling assay and DNA laddering assay. The percentage of apoptotic cells in the degenerative rotator cuff (34%) was significantly higher than that in controls (13%).

Following oxidative and other forms of stress, one family of stress proteins that is often upregulated are heat shock proteins (HSPs). HSPs play a protective role as molecular chaperones in cells by facilitating the folding, intracellular transport, assembly, and disassembly of other proteins. In addition, HSPs protect cells from oxidative damage and protect cells from apoptosis [46,47]. Millar et al [46] found upregulation of HSP 27 and HSP 70, cellular FLICE-inhibitory protein receptor and caspase 8 while downregulation of Poly(ADP-ribose) polymerase, Type-2 angiotensin II receptor and Hypoxia inducible factor

1 occurred in rat supraspinatus tendon subjected to daily treadmill running for 4 weeks. Also, they found that the expression levels of caspases 3 and 8 and HSPs 27 and 70 was higher in the torn edges of supraspinatus of patients undergoing arthroscopic shoulder surgery when compared to matched subscapularis tendon.

Other researches have demonstrated different possible pathways to trigger apoptosis from overuse stimuli. Arnoczky et al [50] found that cyclic strain resulted in an immediate activation of JNK (c-Jun N-terminal kinase), which peaked at 30 min and returned to resting levels by 2 h. This activation was regulated by a magnitude-dependent but not frequency-dependent response and appeared to be mediated through a calcium-dependent mechanotransduction pathway. While transient JNK activation is associated with normal cell processes, persistent JNK activation has been linked to the initiation of the apoptotic cascade [50]. Also, JNK plays an important role in tendon matrix degradation, possibly through upregulating of MMP-1 [51].

In summary, in light of the evidence previously discussed, we can say that the swimmer's shoulder is a multifactorial disease. The main factor that differentiates the swimming of other predisposing factors for shoulder pathology is overuse. All other factors associated with shoulder pain in swimmers (scapular dyskinesis, shoulder instability, impingement syndrome) are secondary and modulate the final effect of overuse.

Sein et al [19] demonstrated that muscle supraspinatus tendinopathy is the pathological basis of swimmer's shoulder and associated factors that generate it.

Overuse, together with the effect of intrinsic and extrinsic factors are the etiology of muscle supraspinatus tendinopathy. Overuse and other factors activate different biochemical signals (HSP, JNK, mechanoreceptors, NO) to generate alterations in the balance between MMP/TIMPs, which in turn alter the composition and architecture of the tendon matrix. Furthermore, these biochemical signals affect the balance between cell proliferation and programmed cell death. If the stimulus produced by the overuse continues, it would develop muscle supraspinatus tendinopathy and finally shoulder pain [Figure 4].

5. Diagnosis and clinical management

5.1. Diagnosis

Kennedy and Hawkins [3] based their clinical syndrome named "Swimmer's shoulder" in the repetitive mechanical impingement of the supraspinatus and the bicipital tendon produced by pull and over-arm recovery. In their original paper [3], they reported that "...diagnosis is usually not difficult. Discomfort is first noticed only after swimming activities. This may progress to pain during and after training and even finally to pain which affects performance of the stroke..." In the physical examination, they described point tenderness over the great tuberosity and over the anterior acromion; a painful arc of abduction maximum at 90° degrees; and impingement signs (Neer or Hawkins). If the bicipital tendon is compromised, there will be tenderness to palpation and a positive Yergason and Speed tests.
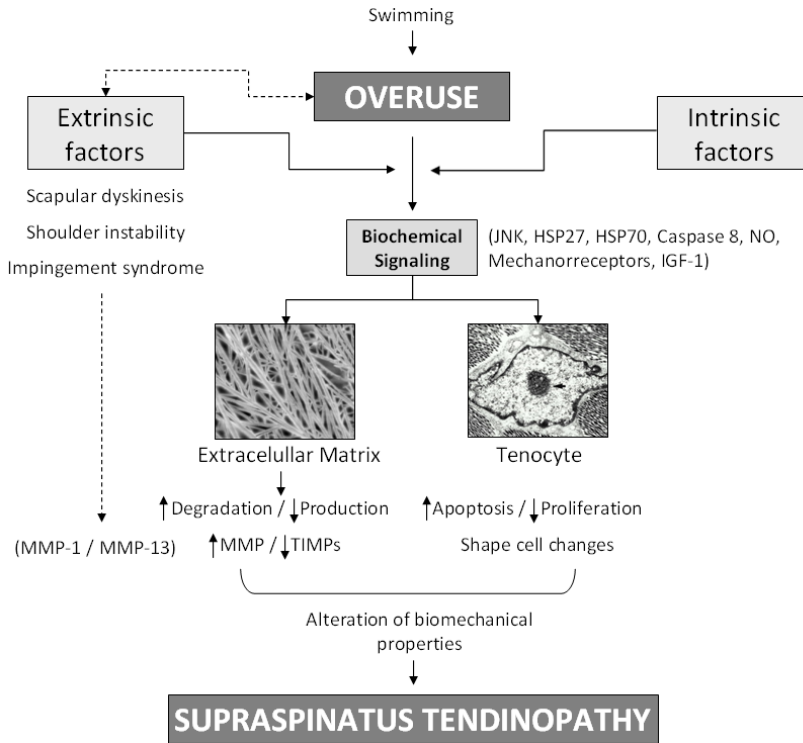
Figure 4. Etiology for supraspinatus tendinopathy.

Bak identified five main categories of swimmer's shoulder [11] [Table 4]. Types A, B, and C may represent different stages of the same condition. The first 4 types nearly always have scapular dyskinesis present.

Type A	Isolated external impingement with subacromial bursitis and increased amount of fluid in the supraspinatus tendon. Normal morphology of acromion. Possible enlarged coracoacromial ligament. No hyperlaxity or instability. Scapular dyskinesis present in most cases.
Type B	Isolated internal impingement without instability. Labral wearing/fraying and minor partial articular side supraspinatus tendon lesions. Scapular dyskinesis present in most cases.
Type C	Complex impingement with both extra-articular and intra-articular pathology. Nearly always minor instability. Scapular dyskinesis present in all cases.
Type D	Isolated minor instability. Often with bilateral hyperlax shoulders. Rarely pain. Scapular dyskinesis is always present.
Type E	Other pathologies, that is, acromioclavicular joint meniscus tear/arthritis (may be related to weight training). Scapular dyskinesis may be present.

Table 4. Types of Swimmer's Shoulder according to Bak's description.

5.2. Preseason assessment

The exact activities that predispose to altered shoulder biomechanics and tissue damage are not fully understood. Most of the research has been done in swimmers that already have the impairment, and the results are extrapolated to design preventive programs [8].

It is consensus that swimmers at high level that have more than five sessions per week should perform dry-land exercise in order to prevent lesions.

Some authors recommend a program to prevent shoulder injury that might lead to pain and dysfunction appears warranted and might include exposure reduction, cross-training, pectoral and posterior shoulder stretching, strengthening, and core endurance training [52].

5.3. Training errors

A rapidly increase in the hours or distance per day is a classic training error. The high level of repetitions can led to fatigue and is the start of the pathological way to swimmer's shoulder, so if the swimmer progression is too aggressive or if he has reached a plateau and some discomfort has appear. A modification of the swim distance may need to be done and/or an increase in the dry-land activities to prevent a progression of an injury. And in some cases a short period of rest out of the water is advisable.

Another training error is to gain more muscle abusing of hand paddles. The increased surface area and resistance tend to over stress the shoulder muscles leading to early fatigue and the imbalance discussed before. If a kickboard is thought to use to rest the pain in the shoulder, it is not a good idea, because they tend to put the shoulder in a disadvantage position for the subacromial space, which is in full elevation and internal rotation.

Yanai and Hay [53] propose: (a) decrease the amount of internal rotation of the arm during the pull phase. (b) Improve early initiation of external rotation of the arm during the recovery phase. (c) Improve the tilt angle of the scapula.

Improper techniques are a common cause of shoulder problems. The coach should seek for increased body roll with scapular retraction to aim optimal strength and endurance of rotator cuff and scapular stabilizers improving a flexibility of pectoral minor in the recovery phase an early pull-through [54].

It has been study that an excessive body roll led to cross the mid-line with the hand during the pull-through phase. This position tends to compress the subacromial space. The arm must stay close to the plane of the scapula in order to reduce the stress in that area [18]. The optimal body roll allows a greater length of the adductors, medial rotator, scapular protractors and abdominal oblique muscles in the beginning of the pull-through phase [55]. In the other hand, an absent body rolls forces the shoulder to a greater extension, abduction and medial rotation compromising the subacromial space.

In other sport and disorder of the shoulder there is a factor that should be considered in prevention and for intervention: improvement of core stability [56,57]. In swimming, the

abdominal and lumbar muscles are the base of the kinetic chain for the propulsion, and should be exercise.

5.4. Treatment strategies

The first time the swimmers experience pain, usually complaints in the subacromial region. The symptoms are related to an inflammatory condition (bursitis, tendonitis) and labeled as impingement syndrome. As we have learned, impingement is a consequence of a subtle or evident imbalance in the shoulder that produces an antero/superior migration of the head by imbalance forces or tissues that can be corrected.

According to Bak [11], when the pain is only at swimming (phase 1), the first strategy is to active rest, reduce training and use icepack after training. The coach should look for technical stroke analysis and correction. Exercise directed toward specific dysfunction. The best documentation of scapular stabilizing exercises is for the low rows, lawn mower, robbery, shrugs and push-ups [57,58].

When the pain is daily and not related to swimming practice (phase 2), the strategy is to rest [11]. Swimming should not be allowed for 1 o 2 weeks. A short course of nonesteroidal anti-inflammatory drugs for 5 to 7 days may be prescribed. Injection of corticosteroid in the bursa is not advisable; this practice is at least controversial. Once the pain is tolerable, direct exercise can continue.

If the pain persist despite of the rest and treatment for more than 3 months [11]. Imaging and a complete study should be done and other strategies should be addressed. Surgical strategies should be considered.

An approach to the specific impairments associated with the symptoms should look for: impaired posture, tight posterior capsule, scapular stabilization and altered scapulohumeral rhythm, impaired rotator cuff strength and glenohumeral hypermobility or instability.

5.5. Impaired posture

Impaired posture are managed through joint and soft tissue mobilization, improve flexibility and strengthening of scapular retractors and deep cervical flexors. Lynch [59] demonstrated that an 8 week exercise program to correct posture and strength muscles result in a decrease in pain and dysfunction in elite swimmers.

The propose muscles that should be stretched are the pectoralis major [Figure 5 and 6], the pectoralis minor [Figure 7], the scalene muscles [Figure 8] and elevator scapula [Figure 9]. The scapular retractor muscles that should be strengthened are the middle/lower trapezius [Figure 10] and the rhomboid muscle [Figure 11 and 12].

Care must be taken to avoid overstretching the anterior capsule [Figure 13], because this could lead anterosuperior migration of the humeral head in swimmers with shoulder laxity.
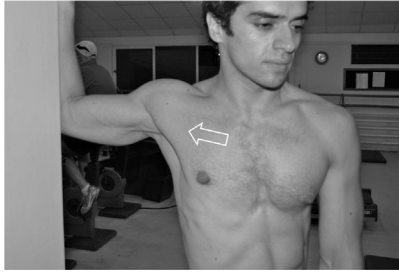
Figure 5. The shoulder is at 90° abduction and 90° external rotation and some extension, in swimmers with excessive shoulder laxity caution has to be taken to not overstretch the anterior capsule.
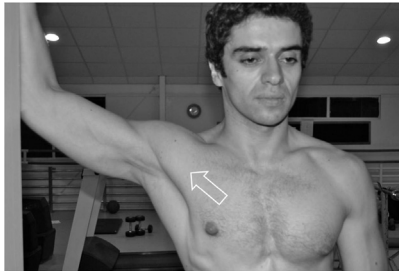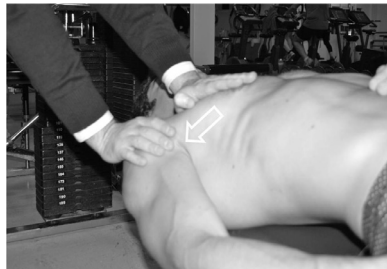

Figure 6. The shoulder is at 120° abduction and 90° external rotation and some extension, in swimmers with excessive shoulder laxity caution has to be taken to not overstretch the anterior capsule.

Figure 7. The pectoral minor is stretched in supine position when the scapula (coracoids) is mobilized superior and downwards.

Figure 8. Self stretching of the scalene muscles.

Figure 9. Self stretching of elevator scapula muscle.

Figure 10. Recruitment of the middle/lower trapezius.

Figure 11. Strengthening of the rhomboid muscles.

Figure 12. Strengthening of middle/lower trapezius and rhomboid muscles.
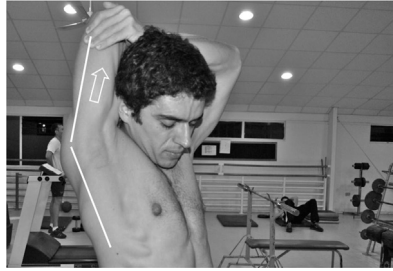
Figure 13. Care must be taken to stretch muscles without compromising the anterior capsule.

5.6. Tight posterior capsule

Posterior capsule tightness is associated with anterior shoulder laxity. Another clinical finding that is common seen is an external rotation and internal rotation deficit. Posterior capsule mobilizations can be performed [Figure 14] or manual self stretching also can be done [Figure 15].
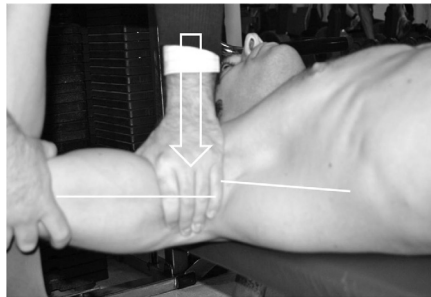
Figure 14. Downward forces are applied to the humerus in order to stretch posterior capsule.
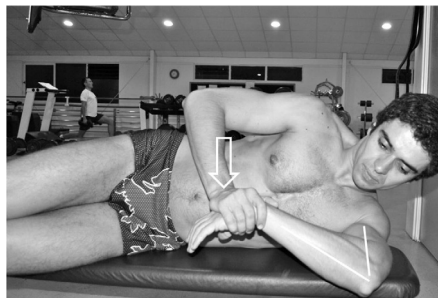
Figure 15. Self stretching posterior capsule.

5.7. Scapular stabilization

Scapular stability and scapulohumeral rhythm is essential in prevention and rehabilitation. The scapular position determines the strength of the rotator cuff and its ability to center the

humeral head. The essential muscles to scapular stability are middle and lower trapezius, serratus anterior and rhomboids. In order to improve scapular movement, a soft tissue release and neuromuscular control must be achieved [Figure 16 and 17].
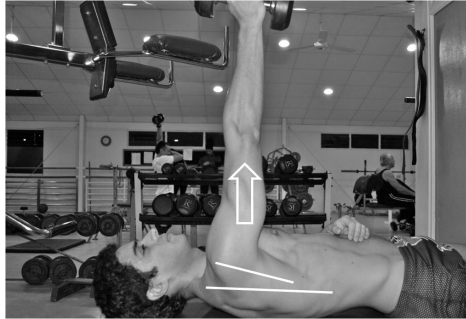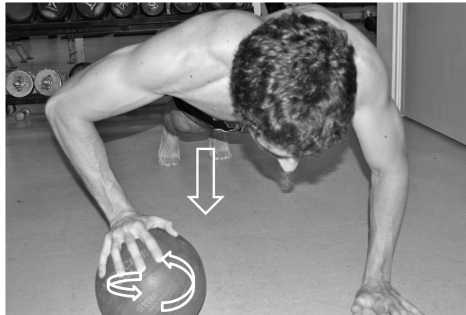

Figure 16. Serratus anterior exercises.
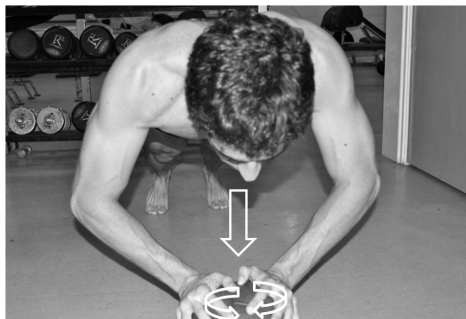

Figure 17. Push up one hand in a ball

Figure 18. Push up both hands in a ball.

The clinician should instruct the patient to retract the scapula prior to and during the humeral motion. Scapular protraction and stabilization in the protracted position are trained through a series of exercises, an example is provided in figure 16, 17 and 18.

5.8. Rotator cuff strength

The range of rotator cuff strengthening exercises may include isometric, concentric, eccentric, and plyometric. Infraspinatus and teres minor are strengthen to counter force the translator forces of anterior muscles [Figure 19 and 20].
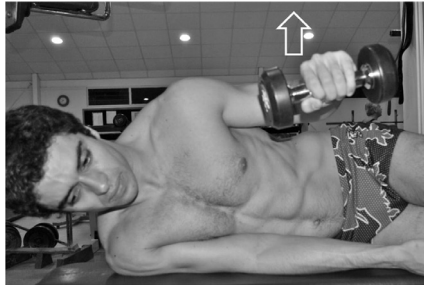

Figure 19. Isolated external rotator exercise.

Figure 20. Scapular retraction should be addressed prior external rotation.

5.9. Hyperlaxity

This special condition is very common in swimmers. Hyperlaxity is often multidirectional in swimmers. The superior migration of the humeral head can cause impingement syndrome [32]. Only certain athletes who crossed the physiological laxity to instability are prone developing symptoms and should be treated. It is documented that heavy-resistance overhead weight training has on causing shoulder pain in swimmers [60]. This is presumably due to forced subluxation of the lax joint during the activity. There are protocols to ameliorate the rotator cuff strength ratio shifts that occur in swimmers and found that this can be helpful for symptomatic athletes but may not be universally effective [61].

Swimmers with a diagnose shoulder laxity and pain; a global training program must be emphasize. This includes strengthening external rotators and scapula stabilizers as lower trapezius.

Clinically, for swimmers with demonstrated pathologic shoulder laxity and pain, the exercise protocol, which emphasizes strengthening of the abductors, external rotators, and

lower trapezius muscle, seems to be helpful. The efficacy of such a program may be multifactorial, including reducing fatigue in the external rotators and scapula stabilizers, and better centering the humeral head in the lax joint, thereby reducing subluxation potential.

Given the shoulder joint laxity of this population, caution should be exercised when prescribing certain training activities that may have a laxity-potentiating effect. Activities that need to be critically examined include passive stretching of the shoulder, especially when forced or causing discomfort, the use of hand paddles, and heavy-resistance overhead weight training. Any activity that promotes increased joint laxity and has the potential to move the situation from physiologic to pathologic laxity must be examined.

For the swimmer with painful shoulders, certain technical adaptations may be helpful. These include increasing body roll, maintaining a high elbow, and avoiding excessive elbow extension before beginning the hand insweep.

5.10. Surgical treatment

A failed conservative treatment is considered after 3 to 6 month, and when a pathological condition is found after a proper clinical and imaging study.

Return to competition after a surgical intervention is not very promising. In a series of anterior acromioplasty for impingement syndrome described by Tibone [15] only a 44% percent return to sport. Another study by Brushøj described returning rates of 56% at a mean of 4 month with labral debridement and subacromial bursectomy [16]. McMaster [63] described a bucket-handle labral tear like a meniscal pathology and labral fraying in an arthroscopic series of swimmers with shoulder problems.

In the other hand when a swimmer presents with pain, global shoulder laxity and failed conservative treatment the surgical results are very promising. Montgomery [64] demonstrated an 80% percent of return to sport when an arthroscopic capsular plication is done.

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