Guest Editorial

Special Edition: Shoulder Injuries in Sport

The shoulder was regarded as the "forgotten joint" in orthopaedic circles until recently. There has been, however, an "explosion" of interest in the shoulder over the past few years.

The biomechanics and pathology of shoulder conditions have been well researched recently and has resulted in a thorough understanding of effective treatment modalities. This is reflected in an excellent article on shoulder rehabilitation by Ms Gisela Lauterbach.

The advent of shoulder arthroscopy has made an invaluable contribution to our understanding of the normal and pathological anatomy.

Arthroscopic surgical techniques have advanced to a level where they do not only compete with the open surgical procedures, but have become the methods of choice, especially when caring for the shoulder of the young athlete.

It is generally accepted in 1994 that a knee surgeon would perform a larger proportion of procedures arthroscopically — the same is fast applying to the shoulder.

It is therefore appropriate that we wrote a short article on "The role of arthroscopy in shoulder problems of the athlete".

An article on "Acute shoulder injuries in the athlete" is a summary of the more commonly encountered injuries and the treatment thereof.

The most common shoulder problem (in athletes and the non-athletic population alike) is that of chronic rotator cuff pathology and a separate article deals with this.

We trust that this Shoulder Edition will contribute to effective care of the shoulders of our athletes.

JF de Beer MBChB, M Med (Orthop)

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Forthcoming Conferences

NATIONAL

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June 19-22. INTERNATIONAL RUGBY MEDICAL AND SPORTS TRAUMATOLOGICAL CONGRESS, SUN CITY. For further information contact the Venue Coordinator, Rugby World Cup, 1995. International Rugby Medical and Sports Traumatological Congress, PO Box 99, Newlands 7725. Tel: (021) 685-3038. Fax: (021) 685-6771.

July 20-23, 1995. ICPFAR SYMPOSIUM 1995, THE INTERNATIONAL COUNCIL FOR PHYSICAL FITNESS AND ACTIVITY RESEARCH. Itala Game Reserve, Northern Natal. Abstracts should be submitted before January 15, 1995. For further details contact Prof M F Coetsee, Department Human Movement Science, University of Zululand, Private Bag X1001, Kwa Dlangezwa 3886. Tel: (0351) 93911 ext 235. Fax: (0351) 93911.

INTERNATIONAL

April 5-7, 1995. CONFERENCE ON NUTRITION AND PHYSICAL ACTIVITY, TO OPTIMIZE PERFORMANCE AND WELL-BEING. The Ritz-Carlton, Buckhead, Atlanta, Georgia, USA. Contact: Ms Lili C Merritt, International Life Science Institute, 1126 Sixteenth Street, NW, Washington, DC 20036, USA. Tel: 202 659 0074. Fax: 202 659 3859.

May 21-25, 1995. FIRST WORLD FORUM ON PHYSICAL ACTIVITY AND SPORT, QUEBEC, CANADA. Contact: World Forum on Physical Activity and Sport, 2 Olace Quebec, Suite 510, Quebec City, Quebec, Canada GIR 2B5. Tel: (418) 648-6000. Fax: (418) 648-0404.

May 23-27, 1995. 10TH INTERNATIONAL SYMPOSIUM ON ADAPTED PHYSICAL ACTIVITY. Contact: 10th ISAPA Secretariat, The Norwegian University of Sport and Physical Education, Department of Information, Postboks 40, Kringsja, N-0807, Oslo, Norway.

May 27-30, 1995. THE ELEVENTH INTERNATIONAL JERUSALEM SYMPOSIUM ON SPORTS INJURIES, DAN PANORAMA, TEL AVIV, ISRAEL. Contact: Congress Secretariat, Dan Knassim Ltd., PO Box 57005, Tel Aviv 61570, Israel. Tel: (972) 3 562 6470. Fax: (972) 3 561 2303.

June 1997. INTERNATIONAL ASSOCIATION OF SPORTS INFORMATION CONGRESS, 10TH SCIENTIFIC CONGRESS, PARIS. Contact: Mr A Poncet, Comite d'Organisation, 11 Avenue du Tremblay, 75012 Paris, France. Tel: 33 1 43 74 11 21 Email FRA 7501 @ CRIUC. UNICAEN. FR.

August 25-26 1995. FISU/CESU CONFERENCE, FUKUOKA, JAPAN. Sport and man: Creating a New Vision. Contact: The Organizing Committee for the Universiade 1995, Fukuoka. Cesu Conference Planning Section, 6-1 Tenjin 2-chome, Chuo-ku, Fukuoka City 810, Japan. Tel: 81 92 733 5212. Fax: 81 92 733 5290.

September 14-17, 1995. THE XITH FINA WORLD SPORTS MEDICINE CONGRESS, ATHENS HILTON, GREECE. Contact: Public Relations Centre, Helen Halyvides, 102 Michalakopoulou Street, 115 28 Athens, Greece. Tel: (301) 775 6336 or 777 1056. Fax: (301) 771 1289.

LETTERS to the EDITOR

Readers' letters concerning articles in the Journal are invited, and will be forwarded to our Editors for consideration for publication.

Please post to: **Glenbarr Publishers C.C.**
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SIXTH SOUTH AFRICAN SPORTS MEDICINE ASSOCIATION CONGRESS

MARCH 22-24 1995 — ELANGENI HOTEL, DURBAN

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PRESS RELEASE

The Sixth South African Sports Medicine Association Congress will be held at the Elangeni Hotel, Durban between 22-24 March 1995.

The theme thoughout the congress of "Professional Sports Care Through Unity", will encompass discussions of Sports Medicine areas over the three days. These will include nutrition, sports medicine and injuries, exercise science, physiotherapy, psychology, biokinetics and sports podiatry.

Areas that compliment and have association will be dealt with in joint workshops. Some combinations are Sports Medicine/Physiotherapy, Exercise Science/ Biokinetics and Physiotherapy/Biokinetics.

Various symposia on the above subjects will be available each day in three different venues, with two sessions each morning and afternoon. Free Communication consisting of presented papers on relevant topics will be held immediately after morning and afternoon tea every day. Interested parties can contact the numbers below for an abstract form.

The visiting speakers will include:
Dr Louis Burke — Nutrition, the competitive edge.
Prof Claude Bouchard — Muscles, performance and genes.
Mr Craig Purdam — Rehabilitation of ankle injuries in sport.
Dr Ken Crighton — Tibial stress fractures: Update on diagnosis and management.

Titles cover a broad spectrum of topics such as muscle injury and disease in sport, difficult hamstring injuries in sport, exercise, infection and immunity, isokinetic dynanometry, drugs in sport.

Conference registration will commence on Tuesday March 21 at the Elangeni Hotel, with a Welcome Cocktail function that evening. On each of the congress mornings, a sporting activity will be organised during the three day period.

It is hoped that the delegate congress package cost will be kept to the 1993 price charged in Cape Town. Special rates for group airfares, accommodation and car hire will be available to congress delegates.

In conjunction with the congress, will be the largest and most exciting sports medicine exhibition to date. New products and techniques, plus a variety of exciting material will be on display. Stand space is still available to any companies who wish to exhibit to a highly selective target audience.

For further information contact Sue or Val — McKenzie Dickerson Int. Tel: (011) 402-3240. Fax: (011) 402-0164.

Acute Shoulder Injuries in the Athlete

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Introduction

The aim of this article is to discuss some of the more common acute injuries of the shoulder complex. The injuries in each area will be discussed with respect to the mechanism of injury, clinical diagnosis, special investigations and management. The rehabilitation of acute injuries as well as the management of overuse injuries will be discussed in another paper in this journal.

Sternoclavicular (SC) Joint Dislocation

Injuries to this joint are rare. The main acute injury to consider in the SC joint is dislocation. Acute dislocation of the SC joint can be either anterior or posterior, with the anterior type being the most common, and fortunately the most benign.

Anterior dislocation

The mechanism of anterior SC dislocation is either from forces transmitted from the hand or a direct blow to the shoulder.

The pathological feature that is of importance is tearing of the joint capsule and costo-clavicular ligament. This may be a problem as it can result in a mechanical block and obstruct attempts at reduction.

The clinical features are acute pain, swelling and deformity of the medial end of the clavicle. Overhead or rotational movement of the arm can cause grating, clicking and popping. This condition must be distinguished from a proximal clavicular fracture by X-Rays. Due to overlying shadows this is a difficult area to distinguish detail on X-rays and specialized views, tomograms and computerized tomography (CT) scans are often needed.

The condition is managed by achieving reduction as follows: The arm is pulled in abduction and the proximal clavicle is manipulated to achieve reduction. The reduction is often difficult to hold but surgery for acute anterior dislocation is seldom indicated as it carries a high risk due to the proximity of such vital structures as the large vessels. Moreover, the chronically dislocated SC joint is usually compatible with good painless function.

The long term complications are that the athlete may notice aching, swelling, clicking or popping in that area. Resection of the proximal portion of the clavicle may be required, with or without stabilization for the rare case. It must be stressed again that surgical intervention for acute or chronic cases should be the exception.

Posterior (retrosternal) dislocation

The mechanism of injury is similar to that described for anterior dislocation, except that the direction of the forces are different.

The clinical presentation of posterior dislocation is classical and requires immediate intervention. Important retrosternal structures such as the trachea and the great vessels are compressed and this may be life-threatening. Classical symptoms are dysphagia, snorting type of breathing and neurovascular symptoms in the upper extremity.

The management is immediate reduction (anaesthesia might be required) by the following method: The patient lies supine with a sandbag or equivalent between the two scapulae. The arm is abducted and extended while traction is applied. The clavicle may have to be grasped with a towel clip and then manoeuvered up and forward to achieve reduction.

Fractured Clavicle

The most common acute injury to the clavicle is a fracture. The mechanism of the injury is a fall on the outstretched arm.

The most common site is the middle third of the shaft. The distal fragment is pulled down by the weight of the shoulder girdle while the proximal fragment is held in place by the trapezium and sternomastoid muscle. There may be a central fragment.

The diagnosis is made clinically and confirmed on X-ray examination. The management is mostly conservative. It is not possible or essential to achieve good reduction but the position may be improved by pulling both shoulders backwards. A figure of eight bandage could be used to hold the position but it is only effective in children, not in adults. It is more important to support the limb with a sling for 3 weeks for pain. Mobilization of the fingers, wrist and elbow should begin early. Most fractures of the clavicle will unite, usually resulting in mal-union which is compatible with normal function. The patient is left with a lump in the area which may get smaller with time, and a slightly "shortened" shoulder. Fractures of the clavicle in children have excellent remodelling potential, but this does not apply to adults to nearly the same extent.

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In rare instances reduction and internal fixation of clavicle fractures are indicated. These are:

i) severely displaced fracture fragments (with the skin being endangered due to pressure from the underlying fragments)
ii) for severe pain
iii) when multiple injuries have been sustained
iv) if early function is critical
v) cosmetic considerations

Acromioclavicular (AC) Joint Sprain

Mechanism of injury

The most common mechanism is a fall or a direct blow to the top of the shoulder.[1] The force is directed downwards and medially. This results in damage to the supporting structures. The magnitude of the force will determine which structures are damaged and thus the severity of the injury. The weakest structures and the first to be damaged are the AC joint capsule and the AC ligament. With progressive increase in the magnitude of the force the coracoclavicular ligaments are damaged and rarely the coracoid process is fractured.

There are other rarer mechanisms of injury. These are:

— a direct lateral blow to the shoulder causing intra-articular damage but no ligament disruption
— a force that is directed posteriorly which damages capsular ligaments, muscles and the trapezoid ligament alone
— a fall on the outstretched arm which drives the humerus into the acromion damaging the capsular structures but not affecting the coracoclavicular ligaments

Classification

A classification of acute acromioclavicular injuries (Rockwood) is listed in Table 1. Types I, II and III are by far the most common injuries.

Clinical features

Clinically, athletes present with a history of acute trauma to the top of the shoulder followed by varying degrees of pain, deformity and loss of function (typically unable to abduct the arm).

In type I injuries there is pain and tenderness over the AC joint. This may be the only finding or mild swelling and loss of abduction may also be present.

In type II and III injuries there is more severe pain swelling and loss of function. However, associated deformity differentiates these from type I injuries. In type IV injuries (posterior displacement of the distal clavicle) there is more pain. Clinically, the displacement can be best appreciated by looking from above the shoulder.

Type V injury is an exacerbation of the Type III injury in which the upper extremity is grossly drooping lower than the normal side. Pain is also more severe than in type III.

Type VI injury is due to an abduction force to the upper extremity and the distal clavicle is displaced inferior to the acromion process. The superior aspect of the

shoulder therefore has a flat appearance. This is extremely rare.

Special investigations

The following X-Rays may be useful in the diagnosis of AC joint dislocation. The routine AP view often demonstrates the AC joint adequately. A better view is the Zanca AC joint view. This is an AP view which is tilted 15° upwards and centred on the AC joint. To distinguish between type II and III injuries, stress views can be done; weights are strapped to both wrists in the standing position and the AC joint views are taken of both shoulders. If the coracoclavicular distance is increased by more than 50% compared to the normal side it represents torn coracoclavicular ligaments. If a posterior dislocation is suspected the axillary view is useful as a diagnostic investigation.

Treatment

Type I injuries

The treatment of these injuries is symptomatic. A sling may be necessary for 7-10 days. Rehabilitation should commence as soon as possible and should emphasize strengthening of the trapezium and deltoid muscle groups. Return to sport can occur as soon as pain allows.

Type II injuries

The treatment of type II injuries is also symptomatic although a slightly more aggressive approach is advocated. The sling could be worn for 10-14 days for pain relief. A well designed rehabilitation programme is again important.

Type III injuries

There is controversy in the literature on the management of type III injuries. The two approaches are either conservative (closed) or surgical (open). Conservative management ("expert neglect") will lead to a chronically dislocated joint which can be compatible with normal painless function. In the young overhead athlete it is probably best to do early open surgery: reduce the acromioclavicular joint and repair the ligaments, including the torn periosteal tube and delto-trapezius fascia. If the injury was left untreated and resulted in a chronically painful instability a late reconstruction with excision of the distal end of the clavicle can be done successfully (Weaver-Dunn procedure).

Types IV, V and VI injuries

The treatment is usually surgical: open reduction and repair of soft tissue, including the delto-trapezius fascia, acromioclavicular ligament and coracoclavicular ligaments. The authors prefer immobilization of the repair using an absorbable sling of PDS tape from the clavicle around the coracoid.

Glenohumeral Joint Instability

Classification

Glenohumeral instability can be classified according to:[2,3]

— the degree of instability (subluxation, dislocation)
— the circumstances of the instability (acute, chronic, recurrent or voluntary)
— the aetiology (traumatic, non-traumatic)
— the direction of the instability (anterior, posterior, multidirectional)

In this article acute glenohumeral instability will be discussed.

Acute anterior glenohumeral dislocation

Anatomy

The shoulder provides more mobility than stability and is therefore prone to instability. The main anatomical structures that prevent anterior displacement of the humeral head are the glenoid labrum, the long head of the biceps tendon, subscapularis and the glenohumeral ligaments (the inferior ligament is the most important). The rotator cuff muscles also provide an important mechanism for stabilizing the humerus because they contract and pull the humeral head against the glenoid.

Mechanism of injury

Acute anterior dislocation can occur when one or more of the anterior stabilizers of the shoulder are stretched or disrupted. The forces that may cause this disruption may be direct (applied to the posterior aspect of the humeral head) or indirect (a movement associated with vigorous hyperextension, abduction and/or external rotation). The latter mechanism is common in sports participation.

The magnitude of the force that is applied will determine the extent of damage. The damage may be only an anterior capsular sprain or there may be subluxation or dislocation. The stability of the shoulder is challenged in any sport where the shoulder is active in extension, abduction or external rotation.

Pathology

Displacement of the humeral head from the glenoid cavity results in injury to the joint capsule, glenoid labrum, humerus, nerves, blood vessels and the rotator cuff.

The joint capsule may be stretched, torn (more likely in older people) or avulsed from its attachment to the glenoid (younger people) — the so-called Bankart lesion. Tears of the glenoid labrum, especially the anterior labrum can occur as part of the Bankart lesion. Superior labral tears, involving the biceps anchor can occur (SLAP lesions).

Fractures of the greater tuberosity can occur. The size of the fragment, displacement of the fragment and site of the fracture are important to consider. If healing takes place with a fragment in a displaced position impingement can result. Fragments displaced >5mm should be managed by open reduction and internal fixation. Large fragments (>25% of the head) should be managed by open reduction and internal fixation. Impaction of the humeral head against the anterior glenoid lip can cause fractures of the posterolateral humeral articular surface (Hill-Sacks lesion). This results in loss of joint congruity and can result in recurrent instability.

The brachial plexus is located antero-infero-medial to the glenohumeral joint. Damage may therefore occur to this structure. The most common nerve that is damaged is the axillary nerve (5-33% of cases). Both sensory (sergeant stripe area) and motor (deltoid) function must be assessed before and after reduction of the dislocated shoulder. Persistent neurological deficits must be assessed by EMG. The nerve injury is usually a neuropraxia that spontaneously improves in a six-month period.

The axillary artery lies in close proximity to the brachial plexus and may also be damaged in anterior dislocation. The pathology may be an intimal tear, laceration, occlusion, branch avulsion or a complete rupture.

The rotator cuff may also be torn in anterior dislocation. It may be difficult to assess the rotator cuff initially but two useful tests are isometric strength in abduction and external rotation. Subscapularis may be torn or avulsed; the important "lift-off test" will indicate this. Rotator cuff tears are more common in older people.

Classification of anterior dislocation

The following types of anterior glenohumeral dislocation have been described according to the site of the humeral head.

— Subcoracoid: This is the commonest type in athletes and is characterized by anterior displacement of the humeral head so that it lies inferior to the coracoid process.
— Subglenoid: This is the second most common type and here the humeral head lies anterior but inferior to the glenoid fossa.
— Subclavicular: This is rare and here the humerus lies medial to the coracoid process and inferior to the lower border of the clavicle.
— Luxatio erecta: In this condition the humeral head is dislocated anteriorly but the arm is fixed in a position of complete elevation.
— Intrathoracic: In this type the humeral head is driven between the ribs into the thoracic cavity.

Diagnosis

The athlete will present with pain and loss of function after an acute precipitating event. The classical history of the mechanism of injury can be obtained. On examination the shoulder is held in slight abduction and the

athlete is unable to adduct, abduct or rotate the shoulder. The lateral contour of the shoulder may be flattened and the humeral head may be palpated below the coracoid process.

The physical examination should also include an assessment of the possible neurological and vascular complications of anterior shoulder dislocation.

The diagnosis should be confirmed by X-Rays. Two views at right angles to each other should be taken. These can be an anteroposterior view, lateral view or an axillary view. Radiographs can indicate the type of dislocation as well as any associated fractures.

Management

The immediate management is to reduce the dislocated shoulder. Gentle reduction may be attempted on the field if the diagnosis is obvious. Early reduction has the following advantages: less muscle spasm to overcome, less risk of damaging neurovascular structures and minimizes damage to the humeral articular surface.

Reduction can be achieved by a number of methods. The principle that is to be followed is that of applying traction to the arm in the abducted and flexed positions. The methods of reduction are briefly as follows:

— Lying supine on the bed with traction applied in 30° abduction while countertraction is applied (Hippocratic method)
— Lying supine with the elbow flexed. Traction is applied, the arm is slightly externally rotated and then internally rotated while adducted (Kochers method)
— Lying prone on the bed with the injured arm hanging down over the side. Traction may be applied in a downward fashion.

Immediately after reduction the athlete should be reassessed clinically and with X-Rays. It is important to re-assess the neurovascular status as well as the integrity of the rotator cuff. With the development of arthroscopic shoulder surgery the treatment is more aggressive. The younger the athlete, the more important early stabilization becomes. The reason for this is the high rate of recurrent instability in young athletes after the first dislocation: in the 20-year old recurrence can be as high as 100% and for any athlete playing contact sport ± 85%.

In traumatic anterior dislocations the most important features of the post reduction management are immobilization and rehabilitation. Immobilization is done in the position of relaxed anterior structures (adduction, internal rotation) and is best achieved by a sling. The duration of immobilization is usually 3 weeks.

The rehabilitation will be discussed in another article in this journal but the principles are to strengthen the anterior supporting structures (subscapularis, inferior glenohumeral ligament) and the external rotators (infraspinatus).

Complications

The complications of acute anterior glenohumeral dislocation are:

— recurrent anterior instability is a very common complication
— neurovascular damage
— associated injuries to other structures (rotator cuff and biceps tendon)
— joint stiffness after dislocation (caused by prolonged immobilization or unreduced dislocation)

Acute posterior glenohumeral dislocation

Posterior glenohumeral dislocation is much less common in the athlete. The incidence ranges from 1 to 4% of all acute dislocations. The sports in which this injury has been described are: skiing, throwing, football, volleyball, gymnastics, racquet ball, wrestling and tennis.

Mechanism of the injury

The mechanisms of an acute traumatic injury can be:

— a severe blow to the front of the shoulder in internal rotation and adduction
— a fall on the outstretched hand with the elbow extended and the humerus in internal rotation

Other more common non-athletic mechanisms of injury are falls during seizures, electric shocks, motor vehicle accidents, industrial injuries and congenital abnormalities that predispose to posterior dislocation (glenoid dysplasia).

Clinical diagnosis

This is an injury that is often missed and would therefore only be diagnosed by carefully paying attention to detail. The mechanism of injury is important to establish and careful questioning is required. The athlete may also complain of severe pain (more than in an anterior dislocation).

The features on clinical examination are:

— locking of the shoulder in internal rotation and adduction (no external rotation is possible)
— abnormally prominent coracoid process
— flattening of the anterior deltoid
— prominent head of the humerus posteriorly
— posterior angulation of the long axis of the arm
— lack of either active or passive external rotation or abduction of the affected arm

A high index of suspicion is required to make the diagnosis clinically.

Special investigations

It is most important to obtain the correct X-Ray views if the diagnosis is to be made. The view that will confirm the diagnosis is an axillary view or trans-scapular view.

Management

The challenge in this condition is to make the diagnosis as early as possible. Once the diagnosis is made the treatment is early reduction. This is best done by an experienced orthopaedic surgeon. The method is to apply gentle traction to the arm in adduction, applying gentle anterior pressure to the humeral head and as pressure is maintained to slowly externally rotate the arm. General anaesthesia is usually required.

Maintenance of reduction is achieved by holding the arm in external rotation and slight abduction with a plaster jacket for 3 weeks. Active rehabilitative exercises are carried out after this until function is regained.

Acute Traumatic Subacromial Bursitis

This injury is caused by a sudden impact force which drives the humerus against the acromion. The usual position producing this injury is a fall on the outstretched

hand with the arm in slight abduction. The clinical presentation is severe pain and loss of function. Haemorrhage occurs in the bursa and blood can be aspirated from the bursa. Pain can be relieved by instilling local anaesthetic into the bursa.

The treatment is conservative consisting of rest followed by mobilization and rehabilitation. Recovery requires 6-8 weeks of treatment.[4] This condition is difficult to distinguish from acute rotator cuff rupture and will often require ultra sonography or arthrogram to exclude the latter.

Fractures of the Scapula

Fractures of the scapula are uncommon in athletes but have been described in football, ice hockey and riding. The mechanism of injury is usually a direct crushing force which fractures the body, neck, acromion or coracoid process. Rarely the injury may be associated with fractured ribs or clavicle, or dislocation of the sternoclavicular joint or AC joint.

On examination the athlete will complain of painful shoulder movements but generally the movements will still be possible. If there are associated respiratory symptoms a thoracic injury must be excluded. X-Rays are usually necessary to confirm the diagnosis. In certain scapular fractures there might be pseudo-paralysis of the rotator cuff muscles mimicking a rotator cuff tear.

The management is conservative. A sling can be worn for comfort and active exercises of the shoulder, elbow and hand should be encouraged from as early as possible. Fractures of the scapula may lead to compartment syndrome of the supra- or the infra-spinatus muscles: this complication should be recognized early and dealt with surgically.

More serious scapular fractures that require operative intervention are usually due to high impact injuries (road accidents) and are not in the scope of this paper.

Acute Soft Tissue Injuries of the Shoulder

Acute rotator cuff tear

Tears of the rotator cuff muscles are rare in younger individuals, but should always be considered in the older patient with a shoulder injury (40 years and older).

A tear of the supraspinatus is the most common and can result from a direct fall on to the shoulder or indirect forces on the arm. Larger tears can extend posteriorly to involve the infraspinatus and teres minor.

Tears of the subscapularis tendon are rare and should specifically be considered in the older sportsman where the arm was forced into abduction and external rotation.

Rotator cuff tears present clinically as follows:

— Severe pain not responding to conservative treatment modalities
— Weakness of elevation. Weak supraspinatus test ("Jobe's thumb down" test)
— Weak "lift-off" test (subscapularis tears).
— Weak external rotation (infraspinatus/teres minor tears)

Confirmation of the tears is by ultra-sound, arthrogram or MRI.

Traumatic tears of the rotator cuff should be repaired surgically as soon as possible. Partial thickness tears occur commonly and can be diagnosed most accurately with the arthroscope.

Rupture of the deltoid muscle

Rupture of the deltoid is infrequent but has been described in handball and volleyball players. It may occur as a result of an acute blow to the arm or as a result of overuse. It usually affects only a part of the muscle. The diagnosis is made on clinical signs and the treatment is conservative followed by active rehabilitation.

Rupture of the pectoralis major muscle

The pectoralis major can be injured (partial or complete rupture) if excessive loads are applied to the shoulder in internal rotation. Sports in which this has been described are weight lifting (bench press), wrestling, shot-put, discus and javelin.

The clinical signs are pain at the insertion of pectoralis on the humerus, swelling, bruising and loss of function. The management is conservative except in cases of total rupture where surgical repair is indicated. Active rehabilitation post injury is encouraged.

Rupture of the long head of biceps

Rupture of the long head of the biceps is seen in older athletes (40-50 years). This is usually secondary to some degenerative change in the tendon, and is associated with rotator cuff tears. The sports where this has been described are: gymnastics, tennis, badminton, wrestling, weight lifting, javelin and oarsmen.

The clinical features are pain over the anterior aspect of the shoulder, swelling (which is prominent on the anterior aspect of the humerus on contraction of the muscle) and loss of function (weakness).

Management is conservative in older athletes but surgical repair is indicated in the competitive or younger athlete (tenodesis of the tendon in its groove). Active rehabilitation post injury or surgery is indicated.

Rupture of the triceps muscle tendon

The tendon of triceps may rupture during sport by falls on the flexed arm or in throwing. The clinical features are pain on the posterior aspect of the elbow and sometimes a gap can be felt. Loss of function is characterized by pain on elbow extension. Treatment is conservative unless there is a total rupture in a young competitive athlete.

TABLE I
The modified classification is as follows:

TYPE 1
- Sprain of acromioclavicular ligament
- Acromioclavicular joint intact
- Coracoclavicular ligaments intact
- Deltoid and trapezius muscles intact

TYPE II
- Acromioclavicular joint is disrupted
- Acromioclavicular joint wider; may be a slight vertical separation when compared with the normal shoulder
- Sprain of the coracoclavicular ligaments
- Coracoclavicular interspace might be slightly increased
- Deltoid and trapezius muscles intact

TYPE III
- Acromioclavicular ligaments disrupted
- Acromioclavicular joint dislocated and the shoulder complex displaced inferiorly
- Coracoclavicular ligaments disrupted
- Coracoclavicular interspace greater than the normal shoulder (i.e. 25 to 100 per cent greater than in the normal shoulder)
- Deltoid and trapezius muscles usually detached from the distal end of the clavicle
- In children, a pseudodislocation of the acromioclavicular joint occurs. The coracoclavicular ligaments remain intact to the intact periosteal tube, and the clavicle is displaced out of the periosteal tube.

TYPE IV
- Acromioclavicular ligaments disrupted
- Acromioclavicular joint dislocated and clavicle anatomically displaced posteriorly into or through the trapezius muscle
- Coracoclavicular ligaments completely disrupted
- Coracoclavicular space may be displaced, but may appear to be same as the normal shoulder
- Deltoid and trapezius muscles detached from the distal clavicle

TYPE V
- Acromioclavicular ligaments disrupted
- Coracoclavicular ligaments disrupted
- Acromioclavicular joint dislocated and gross disparity between the clavicle and the scapula (i.e., 100 to 300 per cent greater than the normal shoulder)
- Deltoid and trapezius muscles detached from the distal half of the clavicle

TYPE VI
- Acromioclavicular ligaments disrupted
- Coracoclavicular ligaments disrupted
- Acromioclavicular joint dislocated and clavicle displaced inferior to the acromion or the coracoid process
- Coracoclavicular interspace reversed with the clavicle being inferior to the acromion or the coracoid
- Deltoid and trapezius muscles are detached from the distal clavicle

Fractures of the Proximal Humerus

Fractures of the proximal humerus can occur during sports participation. It is more common in the older athlete and is usually the result of a fall on the outstretched arm or direct trauma to the shoulder. It has been described in contact sports such as football, rugby, skiing and riding. The most common site of fracture is the neck of the humerus. Avulsion fractures of the greater tuberosity (supraspinatus insertion) often occurs with shoulder dislocation. The lesser tuberosity (subscapularis tendon insertion) can be avulsed in posterior dislocation.

The clinical presentation is pain, swelling and loss of function. Adequate and comprehensive X-Rays are most important to establish the extent and type of injury. The "Trauma Series" of X-Rays should include a true AP, scapular lateral and "trauma axillary" view.

In general the management is conservative (sling) with early mobilization being very important. Surgical treatment is indicated in severe angulation (> 40°), in epiphyseal plate fractures, and displaced fractures of the greater tuberosity (associated with tears in the rotator cuff). More complex fractures of the proximal humerus involving multiple parts of the humeral head usually require surgical reconstruction by a shoulder surgeon.

REFERENCES

1. *Wickiewicz TL: Acromioclavicular and sternoclavicular joint injuries. Clin Sports Med 1983; 2 (2): 429-438.*
2. *Matsen FA and Zuckerman JD: Anterior glenohumeral instability. Clin Sports Med 1983; 2 (2): 319-338.*
3. *Neer CS and Welsh RP: The shoulder in sports. Orthop Clin North Am 1977; 8: 583-591.*
4. *Warren RF: Subluxation of the shoulder in athletes. Clin Sports Med 1983: 2 (2): 339-354.*

FURTHER SUGGESTED READING:

Andrews JR and Wilk KE: The athlete's shoulder. Churchill Livingstone. New York 1994.

Apley AG and Solomon L: Apley's system of orthopaedics and fractures. Butterworth and Co, (Publishers) Ltd, 1982, London.

Davies GJ, Gould JA and Larson RL: Functional examination of the shoulder girdle. Phys and Sportsmed 1981; 9 (6): 82-104.

Jobe FW and Bradley JP: The diagnosis and nonoperative treatment of shoulder injuries in athletes. Clin Sports Med 1989; 8 (3): 419-438.

Kulund DN: The injured athlete. Lippincott, Philadelphia, Toronto, 2nd Ed, 1982.

Marom PJ: Shoulder injuries in Sports. Aspen Publishers, Rockville, 1992.

Peterson L and Renstrom P: Sports injuries: Their prevention and treatment. M Duncan and G Beaton (editors). Juta and Co Ltd, 1986, Johannesburg.

Rockwood and Matson: The shoulder. W Saunders, 1990.

Rosenorn M and Pederson EB: The significance of the coracoclavicular ligament in experimental dislocation of the acromioclavicular joint. Acta Orthop Scand 1974; 45: 346-.

Samilson RL and Prieto V: Posterior dislocation of the shoulder in athletes. Clin Sports Med 1983; 2 (2): 369-378.

Snyder SJ: Shoulder arthroscopy and related surgery. Orthop Clin North Am 1993; January.

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Rotator Cuff Impingement Syndrome in Athletes

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Introduction

The most common chronic shoulder injury in the athlete is the rotator cuff impingement syndrome. Pain presenting in the shoulder joint as a result of pathology in the rotator cuff represents a spectrum of clinical conditions which have been labelled as painful arc syndrome, rotator cuff tears, supraspinatus tendonitis and subacromial bursitis of the shoulder.[2] However, all these syndromes represent a spectrum of diseases and can all probably best be described clinically by the term rotator cuff impingement syndrome.

Mechanism of injury

There are two proposed mechanisms of injury to the rotator cuff in the rotator cuff impingement syndrome. These have been termed the mechanical model and the vascular model.

MECHANICAL MODEL

This model, which was proposed in the early 1970's, states that mechanical compression of the soft tissues in the subacromial area is the principle mechanism of injury.[3] The basis for this compression is the approximation of the greater tuberosity of the humerus to the coracoacromial arch specifically during elevation of the arm.

The exact site of the impingement has been identified. Initially it was thought that the impingement was on the lateral acromion but subsequent studies have shown that the predominant area of impingement was the anterior part of the acromion.[4]

The normal function of the rotator cuff involves the movement of soft tissues (rotator cuff tendons, capsule and biceps tendon) in the subacromial space (space between the humeral head and the coracoacromial ligament). The predisposing factors to mechanical impingement can therefore be related to either narrowing of the subacromial space, or abnormal movement of tissue through the subacromial space. The factors that play a role in each of these mechanisms will now be discussed.

Narrowing of the subacromial space

This is probably the most important cause for impingement. The size of the subacromial space can be decreased by any of the following mechanisms.

Type of shoulder movement:

The size of the subacromial space does not remain constant during shoulder movement. Elevation of the arm, particularly in abduction and forward flexion, causes narrowing of the subacromial space. This is aggravated if the arm is held in internal rotation during the movement because the greater tuberosity of the humerus then enters the canal. It has been shown that during forward flexion and internal rotation the distance between the coracoid and the humerus is reduced to 6.7mm. This space must accommodate the articular cartilage of the humeral head (2-3mm), the subscapularis muscle (2-4mm), the joint capsule (1-2mm) and still leave room for movement. Small variations in the anatomy may predispose to impingement.[5]

Weakness and / or fatigue of the shoulder girdle muscles:

During shoulder function the subacromial space may also decrease if there is excessive movement of the humeral head (upwards movement). The rotator cuff muscles are responsible for approximation, stabilization and centralization of the humeral head during shoulder movement. Any weakness or fatigue of these muscles can result in excessive movement of the humeral head and therefore obliteration of the subacromial space.[6]

Pathology in the subacromial bursa:

Hypertrophy and fibrosis of the subacromial bursa can result in obliteration of the subacromial space.[7]

Pathology of the acromioclavicular (AC) joint:

Degenerative lesions, thickening or separation of the acromioclavicular joint have been described as associated factors in impingement syndrome.[3,8] The mechanism by which AC joint pathology causes impingement is that it causes narrowing of the subacromial space.

Acromion process:

It has been postulated that variations in the shape, size, slope or thickness of the acromion process can

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predispose an individual to narrowing of the subacromial space and therefore the impingement syndrome.[4,9] In this regard the slope and thickness of the acromion appears to play an important role. In particular, if the shape of an acromion is one that angles down too far and forms a "beak" on the end it causes narrowing of the subacromial space.[10] Bigliani[11] has classified the acromion in three types: Type I (flat), Type II (curved) and Type III (hooked).

Abnormal movement

a. Excessively frequent movement:

It has been postulated that excessive repetitive movement of the shoulder in the elevated position can cause irritation and inflammation of the rotator cuff. The resultant oedema will cause swelling and therefore increase the volume of soft tissue in the subacromial space.[12] In more chronic cases the inflammation progresses to fibrosis and calcification. A compromised blood flow to the area can also aggravate this process (see vascular model).[13]

b. Abnormal type of movement:

There are specific movements that are related to rotator cuff impingement syndrome. These are all movements in which there is arm movement above the shoulder level. The sports in which this is a common movement are throwing (baseball, cricket), racquet sports and swimming. If overhead movements are associated with internal rotation of the shoulder the risk of impingement is greater.

c. Muscle weakness:

Impingement can occur due to relative weakness of the humeral head depressors. In swimmers and other overhead athletes there is often an imbalance between the internal and external rotators of the shoulder (the latter being weaker). Attention to these muscles (infraspinatus and teres minor) as a cause of secondary impingement is mandatory.

Poor function of the "scapular setting" muscles can be associated with secondary impingement. The periscapular muscles (trapezium, rhomboids, levator scapulae and serratus anterior) "position" the scapula to enable the rotator cuff to function properly. Should this function be inadequate, the rotator cuff is fatigued and this leads to impingement.

d. Shoulder instability:

Instability (even minor or "subtle" instability) is also an important cause of secondary impingement. During elevation of the arm the humeral head can sublux antero-superiorly, and cause impingement. The instability obviously needs to be assessed and treated (often arthroscopically) for the impingement to be cured.

VASCULAR MODEL

The vascular model for rotator cuff pathology is based on findings of the microvascular pattern of the rotator cuff tendons during movement. The classical description of the avascular zone of the supraspinatus and biceps tendon was reported in 1970.[14] The rotator cuff tendons have a poor blood supply which predispose them to poor healing following repeated microtrauma.

The blood supply to the supraspinatus tendon and the intracapsular portion of the biceps tendon is decreased when the arm is in the adducted position (arm hanging at the side) due to the pressure of the humeral head on the blood vessels. If the shoulder is abducted the vessels are filled.[9] The mechanical model and the vascular model may compliment each other to explain the mechanism of impingement (mechanical irritation and poor healing). However, it has been stated that the area of avascularity may have developed as a protective mechanism to prevent recurrent haemorrhage in an area which is susceptible to repeated microtrauma.[7]

Pathology of rotator cuff impingement syndrome

The pathology of chronic lesions of the rotator cuff can be regarded as a continuous process which ranges from irritation of the soft tissues and ends in rupture of the rotator cuff. Most authors therefore adopt a process of staging lesions of the rotator cuff along this continuum. The most widely accepted staging is that of Neer.[9,15,17] Clinical diagnosis and treatment of the impingement syndrome is based on Neer's stages. The pathology of these stages will now be discussed.

Neer's stage I (Oedema and haemorrhage)

This is the first stage of rotator cuff impingement and is characterized by oedema and haemorrhage in the rotator cuff.[3] The site of the injury can vary from being predominantly in the supraspinatus tendon (most common) or the biceps tendon (less common). It occurs typically in the young athlete (<25 years) and is reversible if managed correctly. This is the predominant lesion seen in the young active sportsperson.

Neer's stage II (Fibrosis)

This stage is characterized by fibrosis in the rotator cuff and therefore reflects persistent inflammation usually as a result of repeated irritation.[3] The site of fibrosis and thickening can be in the rotator cuff tendons (mainly supraspinatus), subacromial bursa or the long head of biceps can also become involved. It is more common in the older age group (25-40 years) and is not necessarily reversible.

Neer' stage III (Rotator cuff tears, biceps rupture)

This stage of the rotator cuff impingement syndrome is characterized by permanent changes in the tissues. These changes are ruptures of the rotator cuff (partial or full) and ruptures of the long head of the biceps.

Neer's stage IV (bony changes)

Specific examples of bony changes that can occur are traction spurs in the coracoacromial ligament and on the ventral surface of the acromion followed by erosion of the anterior acromion.[3]

Neer's stage V (cuff tear arthropathy)
Due to the chronic rotator cuff tear there is cartilaginous degeneration of the glenohumeral joint.

Typically the patients that present with rotator cuff impingement syndrome (types III, IV and V) are older (>40 years). The changes are also permanent unless treated surgically.

Although the same principles of staging are used generally, other authors have used a four stage classification of impingement syndrome.

JOBE'S CLASSIFICATION OF ROTATOR CUFF IMPINGEMENT[1,16]

Stage I
This stage is characterized by oedema and inflammation. There is temporary thickening of the bursa and rotator cuff but no permanent defect.

Stage II
This stage is characterized by disruption of fibres of the rotator cuff but no actual tear. If allowed to heal without correct treatment there is a risk of developing contractures. In addition there is permanent thickening of the bursa and rotator cuff with scar formation.

Stage III
This stage is characterized by permanent thickening and scar formation (as in stage II) but there is a possible partial tear of less than 1 cm. This tear refers to a tear greater than 1cm in the circumferential plane.

Stage IV
In this stage there is permanent thickening of the bursa and the capsule, associated scarring and a tear in the rotator cuff which is greater than 1cm in the circumferential plane.

In this classification the management is again based on the stage of the syndrome.[16]

In this paper the staging of Neer will be used unless indicated otherwise as this is the most widely accepted staging of rotator cuff impingement syndrome.

Diagnosis of rotator cuff syndrome

SYMPTOMS:

Although it is important to elicit the symptoms in order to establish the correct diagnosis it must be emphasized that the history should also include thorough questioning on the mechanisms and possible causes of the athletes' shoulder complaint. This should include history of the mechanism of injury, training program, and equipment used.

Stage I
The main presenting symptom in all stages is pain in the shoulder. However, the nature of the pain may differ. In stage I the pain is often a dull ache only after the activity. The pain may be described as "tooth ache" like pain.[15] This can then progress to pain experienced during activity which eventually interferes with the activity.[15] A hallmark of this pain is that it subsides on cessation or modification of the activity. The pain may also manifest as night pain.

There is no associated weakness of muscles in stage I except that related to pain. In the overhead athlete the arm movement must be analyzed and note should be taken where in the movement the pain occurs, as this can help with the diagnosis, eg. in the baseball-pitcher, if the pain occurs in the "cocked" position it is more likely due to instability. If the pain occurs when the arm is overhead it is more than likely due to impingement, and so on. This pattern can be applied for all the different overhead sports.

Stage II
The symptoms of stage II are an extension of those in stage I. The pain is often more frequently encountered during the day and may be experienced during non-sports activities such as reaching overhead. Night pain is also more prominent.

There may be complaints of a slight limitation of movement or associated muscle weakness in stage II. The hallmark of stage II is that the disease process is no longer reversible with activity modification or cessation.[3]

Stage III
The young competitive athlete rarely presents in this stage. There is usually a long history of chronic pain and loss of function. Athletic activity is often not possible because of the limitations of pain and weakness. The symptoms of stiffness (due to loss of range of movement) and muscle weakness (abduction and external rotation) are prominent. The periods of pain are prolonged and are particularly prominent at night. The hallmarks of this stage are the chronicity of the disease and the permanent pathological changes.

SIGNS

It is important to elicit the clinical signs that will conform the correct diagnosis as well as identify the stage of the disease. However, a thorough biomechanical examination of the shoulder must include evaluation of the range of movement, muscle strength and mechanism of injury.

Stage I
The most important clinical sign in the diagnosis of this condition is a positive Neer's impingement sign.[3] The diagnosis can be confirmed by a positive Neer's impingement test (injecting the subacromial space with 6-10ml local anaesthetic and repeating the impingement test). Other clinical signs are:[13]

- palpable tenderness over the greater tuberosity of the humerus at the supraspinatus insertion
- palpable tenderness along the anterior edge of the acromion
- painful abduction specifically between 60 and 120° (painful arc sign)
- muscle weakness associated with abduction which disappears after injection of local anaesthetic into the subacromial space
- tenderness over the biceps tendon (biceps involvement in stage I however, is rare and this finding is not common).[17]

Stage II
The clinical signs of stage I can all be demonstrated in stage II. In addition there is:
— limitation of movement
— the weakness is more pronounced (can however be normal following the injection of local anaesthetic)
— soft tissue crepitus
— a catching sensation during abduction at approximately 100° which is due to fibrosis and scarring.

Stage III
The findings on physical examination of stage III are:[13,15,18]
— limitation of shoulder movement (active more than passive)
— muscle atrophy (infraspinatus and supraspinatus)
— biceps tendon lesions ranging from tenderness to rupture
— acromioclavicular joint involvement (positive compression test)
— weakness of shoulder musculature which persists after injection of local anaesthetic into the subacromial space
— positive clinical signs for rupture of the rotator cuff muscles
— sings of shoulder instability may also be present

SPECIAL INVESTIGATIONS
X-Rays
Good quality X-Rays should be done including the following views:
— True AP
— Supraspinatus outlet view
— Axillary view
— AP 30° (caudal tilt) is optional
— AC joint view is essential

The X-Ray findings in stage I are normal. The X-Ray changes of impingement syndrome typically lag behind the clinical course.[15] However, causes of underlying predisposing factors may be identified on X-Rays (acromial shape and angle, AC joint pathology).

There may be features noted on X-Rays later in stage II. These are cystic changes in the greater tuberosity of the humerus as seen best on the 10° AC view, osteophytes on the undersurface of the acromion and, changes in the acromioclavicular joint.

X-Ray changes are most likely seen in stage III. The abnormalities that must be identified are:[9,15]
— narrowing of the subacromial space (< 5mm space indicates a rotator cuff tear)
— superior migration of the humeral head
— erosion of the acromion
— sclerosis and osteophyte formation on the acromion and greater tuberosity of the humerus

X-Rays must also be evaluated for the presence of underlying predisposing factors to impingement and causes of narrowed subacromial space should be identified.

Ultrasonography
Ultrasound is cost-effective and accurate to detect tears of the rotator cuff but is operator-dependant. It is also very helpful to show calcific deposits in the cuff, biceps tendon pathology, subscapularis tears and coracoid impingement.

Arthrography
Double contrast arthrography of the shoulder joint is a useful technique for the diagnosis of rotator cuff tears with an accuracy of 99%.[19] Communication of contrast medium between the glenohumeral joint and the subacromial bursa indicates a complete tear of the rotator cuff. It can be combined with CAT scan ("CT arthrography"). Arthrography only indicates the presence, not the size of a rotator cuff tear.

Magnetic resonance imaging (MRI)
MRI is a very useful investigation to delineate exact pathology in the soft tissues (a shoulder dedicated coil must be used). It is therefore accurate in the diagnosis of rotator cuff pathology. The major limitation is the time it takes and the expense involved, and should only be used in the exceptional case.

Arthroscopy of the shoulder joint
This technique is rapidly becoming the investigative procedure of choice in rotator cuff impingement syndrome. It is very accurate for the identification of rotator cuff pathology and has the added advantage that surgery may be performed at the same time.[15] Surgical procedures that can be done arthroscopically include bursectomy, acromioplasty, excision of the lateral end of the clavicle, rotator cuff debridement and stabilization procedures like anterior and posterior Bankart repair and SLAP lesion repair. (The diagnosis and treatment of the latter can in fact only be done arthroscopically).

Differential diagnosis
The following conditions must be distinguished from impingement syndrome.[13]
— acute traumatic bursitis
— shoulder instability syndromes (which may be associated with secondary impingement)
— primary acromioclavicular pathology
— cervical spine disease
— glenohumeral arthritides
— calcific tendonitis
— frozen shoulder

Management of Rotator Cuff Impingement Syndrome
Stage I
This stage of the disease process is reversible and should therefore be managed correctly to avoid progression. The management is conservative and the principles of conservative management are to diminish the inflammation, maximize shoulder function and to correct or modify the activity.[18] The conservative management and rehabilitation will be addressed in a separate article.

General principles
It is important to note that rotator cuff impingement in the young athlete is often secondary to instability

— the latter should then be addressed first. Weakness of the humeral head depressors (rotators) is also a common causative problem and specifically strengthening of the external rotators (infra-spinatus and teres minor) should be addressed. Posterior capsular tightness is often an associated causative factor and stretching of the posterior capsule is mandatory.

Non steroidal anti-inflammatory drugs
A short course of these drugs can be prescribed to decrease inflammation in this stage of the disease.[13,15,20]

Subacromial cortisone injection
Although this is a common form of therapy, frequent injections without caution increases the risk of tendon rupture.[21] In general no more than two or three cortisone injections are recommended. The cortisone can be mixed into the solution when doing the impingement (injection) test with local anaesthetic.

Surgical treatment (Stage I)
Rarely athletes with stage I lesions are not successfully rehabilitated on conservative treatment. Although these athletes are a small minority, surgery may have to be considered if conservative treatment has failed (at least 6 months of adequate conservative treatment must be allowed).[13] The athlete must also be aware that athletic competition after surgery may be compromised (important in the throwing athlete). Surgical management should include a thorough arthroscopic evaluation of the gleno-humeral joint to exclude instability with secondary impingement and other pathology, followed by evaluation of the sub-acromial space to confirm impingement. If bursectomy, excision of the coraco-acromial ligament and acromioplasty is decided upon it should preferably be done arthroscopically in the young athlete.

Stage II
Conservative treatment
The initial treatment for stage II of rotator cuff impingement syndrome is conservative. The type of treatment is essentially the same as stage I. Differences in the conservative approach to stage I and II are:
— activity in stage II must be restricted more than in stage I (ie. only kicking in swimming, complete rest from throwing and limiting tennis shots to the forehand hitting against a wall)
— medication use in stage II may be more prolonged
— greater emphasis is placed on a range of motion exercises in stage II to prevent adhesive capsulitis
— resistance exercises tend to be less intense in stage II to prevent overloading of the rotator cuff

Surgical management of stage II
Surgical management is more common in stage II than in stage I. The main indication for surgery is failed conservative treatment (duration of at least 6-12 months). Other indications for surgery are i) permanent narrowing of the subacromial space for example if there is an abnormal angle of the acromion and ii) associated instability of the shoulder which results in impingement. Under these circumstances conservative treatment is likely to fail unless the underlying defect is corrected. Surgical management should again include arthroscopic evaluation and arthroscopic treatment as far as possible.

Stage III
The treatment of stage III of the rotator cuff impingement syndrome is surgical.[10,13,15,22] Surgery involves decompression, repair of the rotator cuff and anterior acromioplasty. The outer part of the clavicle and the acromioclavicular joint may also have to be resected as part of the decompression. The tendency is to perform arthroscopy of the shoulder earlier and this is both as a diagnostic procedure as well as performing the surgery arthroscopically.

Prognosis
There are no published controlled clinical trials on the conservative treatment of rotator cuff impingement syndrome.[15] In one report on the outcome following surgical treatment for rotator cuff tears in athletic population, the results indicate pain relief post operatively in 76% of subjects, but only 32% of the professional pitchers were able to return to the same level of competition after surgery.[22]

REFERENCES

1. *Jobe FW and Jobe CM: Painful athletic injuries of the shoulder. Clin Orthop and Rel Res 1983; 173: 117-124.*
2. *Brunet ME, Haddad RJ and Porche EB: Rotator cuff impingement syndrome in sports. Phys Sportsmed 1982; 10 (12): 86-94.*
3. *Neer CS and Welsh RP: The shoulder in sports. Orthop Clin North Am 1977; 8: 583-591.*
4. *Neer CS. Impingement lesions. Clin Orthop 1983; 173: 70-77.*
5. *Gerber C, Terrier F and Ganz R: The role of the coracoid process in the chronic impingement syndrome. J Bone Joint Surg (Br) 1985; 67: 703-708.*
6. *Pappas A, Zwacke R and McCarthy C: Rehabilitation of the pitching shoulder. Am J Sports Med 1985; 13: 223-235.*
7. *Ciullo JV and Stevens GG: The prevention and treatment of injuries to the shoulder in swimming. Sports Med 1989; 7: 182-204.*
8. *Kessel L and Watson M: The painful arc syndrome. J Bone Joint Surg (Br) 1977; 59: 166-172.*
9. *Hawkins RJ and Hobelka PE: Impingement syndrome in the athletic shoulder. Clin Sports Med 1983; 2 (2); 391-404.*
10. *Nash HL: Rotator cuff damage: Reexamining the causes and treatments. Phys and Sportsmed 1988; 16 (8): 12135.*
11. *Bigliani LU: Arthroscopic Resection of the Distal Clavicle. Orthopaedic Clinics of North America 1993; 24: 133-141.*
12. *Penny JN and Welsh RP: Shoulder impingement syndromes in athletes and their surgical management. Am J Sports Med 1981; 9 (1): 11-15.*
13. *Hawkins RJ and Kenndy JC: Impingement syndrome in athletes. Am J Sports Med 1980; 8 (3): 151-158.*
14. *Rathbun J and Macnab I: The microvascular pattern of the rotator cuff. J Bone Joint Surg (Br) 1970; 52: 540-553.*
15. *Thein LA: Impingement syndrome and its conservative management. JOSPT 1989; 11 (5); 183-191.*
16. *Jobe FW and Moynes DR: Delineation of diagnostic criteria and a rehabilitation program for rotator cuff injuries. Am J Sports Med 1982; 10 (6): 336-339.*
17. *Ciullo JV: Swimmer's shoulder. Clin Sports Med 1986; 5 (1): 115-136.*
18. *Simon ER and Hill JA: Rotator cuff injuries: An update. JOSPT April 1989; 394-398.*
19. *Newberg AH: The radiographic evaluation of shoulder and elbow pain in the athlete. Clin Sports Med 1987; 6: 785-809.*
20. *Richardson A: Overuse syndromes in baseball, tennis, gymnastics and swimming. Clin Sports Med 1983; 2: 379-389.*
21. *Kennedy JC, Hawkins R and Krissoff WB: Orthopaedic manifestations of swimming. Am J Sports Med 1978; 6 (6): 309-322.*
22. *Tibone JE, Elrod B, Jobe FW, Kerlan RK et al: Surgical treatment of tears of the rotator cuff in athletes. J Bone and Joint Surg 1986; 68A (6): 887-891.* □

The Role of Arthroscopy in Shoulder Problems of the Athlete

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Introduction

Arthroscopy has revolutionized our knowledge of the anatomy and pathology of shoulder problems. Arthroscopy has also vastly improved the accuracy of diagnoses, and instituted more specific treatment with much better outcome. A surgeon can not adequately deal with shoulder problems without a thorough working knowledge of shoulder arthroscopy.

Impingement

The majority of impingement syndromes in young athletes are secondary to instability and other lesions like labral tears (including SLAP lesions). These problems can best be diagnosed and treated arthroscopically. In primary (structural) impingement in the athlete, resistant to conservative treatment, an arthroscopic acromioplasty is the treatment of choice. The advantages of arthroscopic acromioplasty (rather than open acromioplasty) are:

— The gleno-humoral joint can be inspected to rule out other causes of impingement (this is mandatory in the "overhead athlete" like throwers, swimmers, etc).
— The sub-acromial space can be inspected to confirm impingement.
— There is no damage to the deltoid muscle as in open acromioplasty.
— There is early return to full function.
— There is usually minimal post operative pain.
— Short hospital stay (can be done on an "out-patient" basis).
— Cosmetic advantage: no incisions with an unacceptable appearance.

The results of arthroscopic acromioplasty have become so good that it will probably be regarded as "unacceptable" to do open acromioplasty in young athletes.

Posterior Impingement

Posterior impingement is caused by abduction and external rotation of the shoulder, mostly in throwers. In this position the posteriosuperior glenoid rim acts as a "chisel" stripping the insertion of the rotator cuff from the greater tuberosity. This diagnosis can only be made with dynamic arthroscopy.

SLAP lesions (Superior Labrum Anterior to Posterior)

These are tears of the superior glenoid labrum and insertion of the tendon of the long head of the biceps. Lesions can only be diagnosed, differentiated and treated arthroscopically.

Shoulder Instabilities

Arthroscopy has increased our understanding of shoulder instability and labral tears. The direction, degree and pathology of instability can be assessed arthroscopically. Although open surgical repair of Bankart lesions had been regarded as the "gold standard" arthroscopic stabilization has become a successful procedure in the hands of experienced shoulder arthroscopists. The following procedures can be adequately performed:

— Anterior Bankart repair
— Posterior Bankart repair
— Capsular plication (multi-directional instability)
— Labral repair (like SLAP lesion repair).

Rotator cuff tears

Full-thickness tears: can be accurately detected if there was doubt about the diagnosis. Smaller tears can be repaired arthroscopically.

Partial-thickness tears: occur mostly on the joint side of the cuff and can only be diagnosed arthroscopically. At the same time they can be debrided using the arthroscope.

Full-thickness tears of the rotator crescent, with an intact rotator cable (= "functional" tears as described by Burkhart) can be treated arthroscopically with debridement and arthroscopic acromioplasty. This would usually apply to the older sportsperson in less demanding sports like golf, bowls, etc.

Acromioclavicular joint pain

Painful acromioclavicular joint is common in sports people. In those where conservative treatment has failed, excision of the lateral end of the clavicle becomes indicated ("Mumford" procedure). This procedure can be adequately performed arthroscopically, in fact the open procedure is less acceptable in the "overhead athlete" as it can lead to damage to the delto-trapezius fascia and weakening of the shoulder. The arthroscopic procedure also avoids damage to the acromioclavicular ligament with less likelihood of joint instability.

Chondral and Osteochondral lesions

These lesions of the joint surfaces can only be diagnosed arthroscopically and can often be treated at the same time. "GLAD" lesions (gleno-labral articular disruption) is a good example of this.

(Continued on page 23)

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Rehabilitation of the Athlete's Shoulder

Gisela Lauterbach

Abstract

A holistic approach to the rehabilitation of athlete's shoulder is presented. Factors that should be included in the assessment are posture, spinal and neuromeningeal mobility, physiological and accessory movements of all joints of the shoulder complex, stability tests, resisted movements, and synchrony of movement during active movements. Decrease of pain and control of inflammation are important initial goals of rehabilitation. Normal range of movement of all joints of the shoulder girdle should be achieved before starting with a strengthening programme and mobilizations should be based on the normal arthrokinematics. Dynamic proximal control must be established before adding distal mobility. Early strengthening exercises for the rotator cuff therefore are of paramount importance. The humeroscapular rhythm must be restored during elevation before strengthening specific muscles. Closed kinetic chain exercises enhance static control. Both concentric and eccentric contractions should be included and techniques should be adapted to replicate the biomechanics of the specific sporting activity. Progression of exercises is determined by the control of movement, presence of pain or related symptoms, and never by time. Trunk and lower extremity strength and endurance work should be included into the programme. The programme should employ correct movement patterns to prevent return of symptoms and for improved performance.

INTRODUCTION

Dysfunction of the athlete's shoulder is a commonly seen condition in a sports injuries clinic. Symptoms may range from pain during or after activity, weakness, catching sensations, or loss of performance. Varied approaches to treatment have been described. The Cyriax (1980) approach advocates treating localized lesions by deep transverse frictions; Travell and Simons (1983) identified specific pain distributions of trigger points of each muscle which are locally treated. Results of treatment are often disappointing and frustrating for the athlete. Too often, treatment is directed to a specific component of the pain only, neglecting the multi-structural approach to the shoulder complex.

Various factors should be considered as possible origin of the shoulder dysfunction. In the overhead athlete utilizing repetitive movement of the arm at high forces and acceleration, secondary impingement is a common syndrome. Contributing factors are minor instabilities, loss of functional stabilization by the scapular muscles, fatigue and/or loss of depressor action of the rotator cuff muscle (Kamkar 1993). The aim of rehabilitation is to return the athlete to the previous level of performance in the shortest, yet safest possible time. Further, predisposing factors to injury must be identified and corrected as more emphasis is placed on injury prevention. Detailed reviews of rehabilitation programmes are available from recent literature (Jobe and Pink 1993, Litchfield et al 1993, Magarey and Jones 1992, Wilk and Arrigo 1993). The aim of this paper is to present a holistic approach to the rehabilitation of athlete's shoulder. As a detailed assessment of the athlete forms the basis of the rehabilitation programme, components that should be examined are presented.

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Assessment

Maitland (1991) presents a systematic approach to the examination of the shoulder joint. A detailed history and training programme of the athlete is taken. The posture of the athlete must be analyzed as increased thoracic kyphosis and an anteriorly displaced head position have been identified as possible factors leading to shoulder dysfunction (Ayub 1992). Further, range of movement of the cervical and thoracic spine is assessed and considered as predisposing factors to dysfunction. Physiological and accessory movements of all joints of the shoulder complex should be tested for range and reaction of pain and muscle spasm. Posterior capsular tightness has been identified as a factor leading to secondary impingement (Kamkar 1993). This is assessed by the amount of anterior displacement of the humeral head during passive medial rotation of the ±45° abducted arm.

Stability tests are performed, including apprehension test, sulcus sign, anterior and posterior drawer signs (Malone 1994). It is important to determine the range of movement of these, the end-feel and the response of pain during these tests. Testing positions should be adapted to the athlete's specific biomechanics. For example, with the throwing athlete, the anterior drawer sign, usually performed in 90° flexion in the scapular plane, may be repeated in the position of full flexion, lateral rotation (Fig. 1). With the swimmer it may be performed in the position of mid-recovery of the crawl stroke i.e. in the quadrant position.

Resisted movements are used to determine a soft tissue component, for example lesions of the rotator cuff. However, if these are used as isolated findings, the full functional biomechanics of the shoulder complex is not appreciated. Control of movement in functional direc-

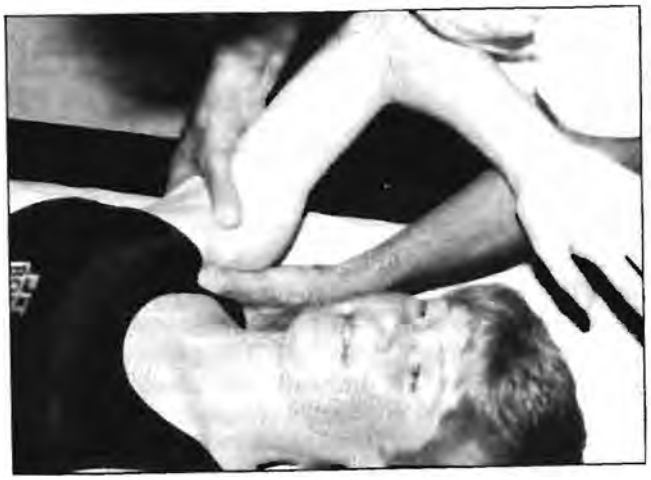

Fig. 1. *Anterior drawer sign tested in a functional position. The relative movement between the fixed coracoid and the humeral head is noted.*

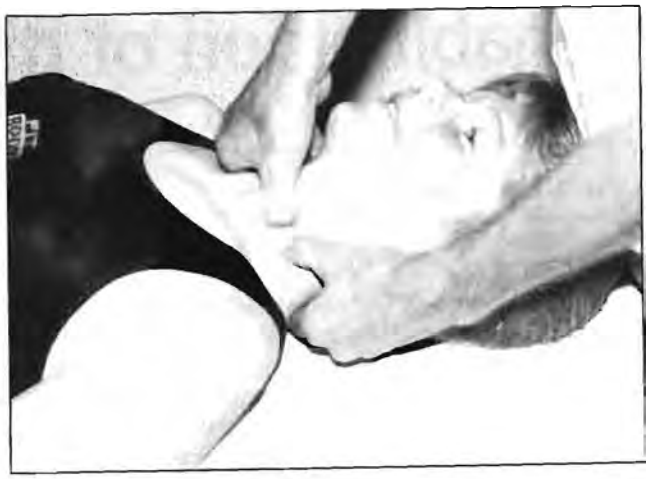

Fig. 2. *Anterior-posterior mobilizations of the cervical spine.*

tions is considered more important in the evaluation of shoulder dysfunction than pure reproduction of pain during a specific resisted test. For example, scapula control and the function of the rotator cuff in various positions must be tested and compared to the uninvolved shoulder adapting the tests to the athlete's specific sports activity. Isokinetic equipment may be used to assess muscle power. However, these tests are often in non-functional isolated directions. In the shoulder joint control and co-ordination of complex movement are more important than muscle power per se in the shoulder joint.

Neuromeningeal structures have been identified as possible components of shoulder dysfunction (Butler 1991) in both overuse and traumatic injuries. The upper limb tension tests are used to assess firstly, the mobility and integrity of the neural structures of the brachial plexus and sympathetic nervous system and, secondly, response of presenting symptoms to these movements.

Planning of the rehabilitation programme needs to incorporate every component found to be comparable to the athlete's symptoms during examination. It should be designed according to the athlete's specific needs and recipe-type programmes should not be used. Rather, signs and symptoms must be re-assessed constantly and the programme be progressed accordingly.

Principles of Rehabilitation

1. Decrease of pain

Decrease of pain and control of inflammation are important initial goals of rehabilitation. Anti-inflammatory modalities, ice and electrotherapy, may be applied. To treat pain, Grade I and II passive accessory mobilizations of the involved joints are used (Maitland 1991). Anterior-posterior mobilizations of the cervical spine are effective if neural tension techniques were comparable to the patient's pain during the examination (Fig. 2).

2. Increase of range of movement

Before starting strengthening programmes it is absolutely necessary to achieve normal glenohumeral and scapulothoracic motion. Without normal flexibility abnormal movement patterns may be enhanced (Pappas et al 1985). Mobilization of the shoulder joint should be based on the normal arthrokinematics. The accessory movements of inferior and posterior glide occur during the first 90° of flexion, followed by an inferior and anterior glide. Specific techniques can be used at the limitation of the physiological range of movement as Grade IV mobilizations to stretch the capsule and restore normal motion (Fig. 3).

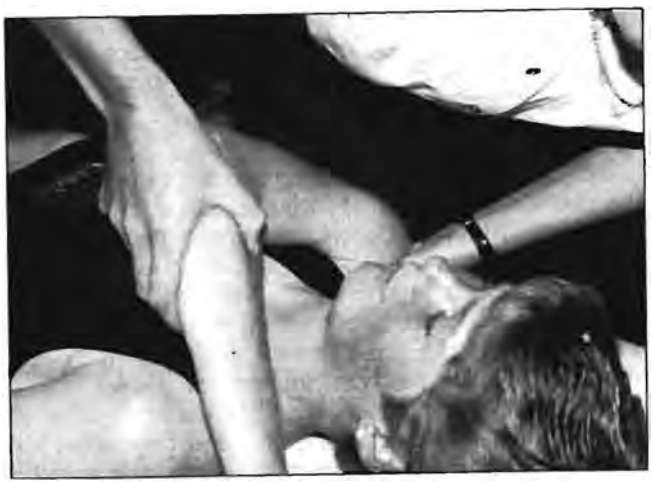

Fig. 3. *Mobilization of the posterior capsule. Compression through the long axis of the humerus is added oscillatory on horizontal adduction.*

Athletes often need an increased range of movement for efficient performance and mobilization techniques should be adapted towards these. Lateral rotation of 170° in full elevation has been documented in baseball pitchers (Dillman et al 1993). Hypermobility of the anterior shoulder is common in swimmers and is neces-

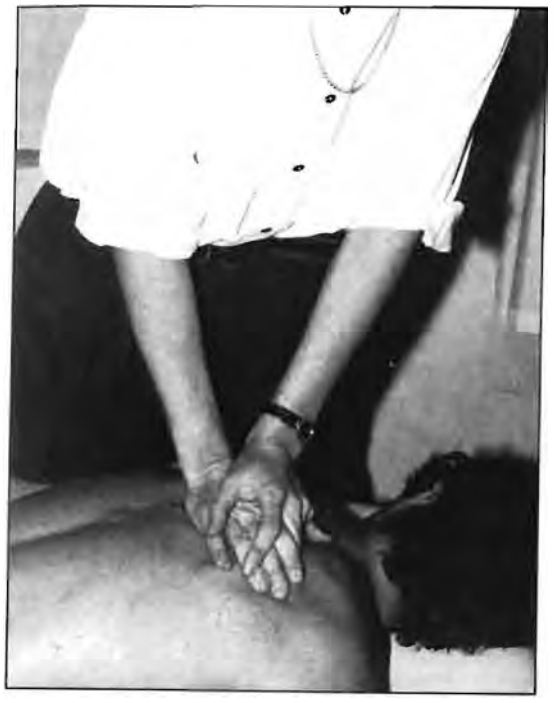

Fig. 4. *Mobilizations of the thoracic spine.*

sary during the recovery phase of the crawl phase. The quadrant position (Maitland 1991) best replicates these movements and should be "cleared" before discharging the athlete from rehabilitation. Further, mobilization of the cervical and thoracic spine may be necessary if these joints were found to be stiff or painful on examination (Maitland 1987) (Fig 4).

3. Improvement of the rotator cuff function

The rotator cuff and biceps muscles function as depressors during elevation and have a strong eccentric contraction during follow-through of the throw (Dillman et al 1993). If this function is reduced by early fatigue or weakness, impingement of the cuff occurs against the anterior coracoacromial arch (Kamkar 1993). Early strengthening exercises, maintaining control of the scapular position, are therefore of paramount importance. Isometric contractions using rhythmical stabilization techniques (Knott 1968) in neutral position are progressed to various positions of flexion and abduction into the direction needed in the particular sporting activity (Fig. 5). Once painfree control is achieved in various positions, dynamic through-range exercises are added. Various methods of resistance can be employed, for example manual resistance by the therapist, self resistance, elastic bands and light weights. Scapular control must be emphasized throughout to prevent re-injury. As endurance is an important factor to be considered, it is advisable to use low weights with high repetition.

4. Re-education of movement

The correct muscle firing pattern is an important prerequisite for optimum performance and injury prevention. In the shoulder dynamic proximal control must be established before adding distal mobility. The humeroscapular rhythm must therefore be restored during elevation before strengthening specific muscles. As painfree range of movement is regained, control of the scapula can be achieved in various starting positions, for example using proprioceptive neuromuscular facilitation (PNF) techniques (Knott 1968) of the scapula in side lying. This can be followed by improvement of scapula control in progressive ranges of flexion and elevation in the scapular plane (Fig. 6). Free active exercises in prone are added, emphasizing rhomboid and lower and middle trapezius muscle strengthening (Fig 7). Exercises are progressed by adding light weights or using elastic bands.

Stability exercises include the closed kinetic chain exercises, for example push-ups. These facilitate joint compression and stimulate neuromuscular proprioceptors to enhance static control (Dickoff-Hoffman 1994). The serratus anterior muscle plays an important role in proximal stabilization of the shoulder girdle, and is strengthened during press-ups "with a plus", i.e. adding full protraction of the scapula. These are initially per-

Fig. 5. *Rhythmical stabilization techniques for the rotator cuff are used in the early phases of rehabilitation. Scapular position is monitored constantly.*

Fig. 6. *Re-education of the muscle firing pattern. Scapular stabilization is monitored while adding flexion or abduction of the glenohumeral joint.*

Fig. 7. *Free active exercises emphasizing interscapular muscle contraction. The arms are horizontally extended while controlling the scapular position.*

Fig. 8. *Press-up exercises are progressed by using an exercise ball in the final stages of rehabilitation.*

formed against the wall, and progressed to the floor. Performing them on a balance board or exercise ball (Fig. 8) requires maximum proximal stabilization and should be used in the last phases of rehabilitation.

5. Strength training

Straight arm exercises in flexion and abduction should never be employed before these movements cannot be controlled sufficiently by the proximal stabilizers, viz. scapular and rotator cuff muscles. PNF patterns, especially the diagonal flexion/abduction/external rotation, can be used in preparation of these, utilizing manual resistance. This has the advantage of constantly monitoring control of the movement and resistance can be accommodated accordingly. Both concentric and eccentric contractions should be included. Using these techniques in the seated or standing starting position replicates the activity needed during sports. Weight training on apparatus can be commenced, monitoring scapular control constantly. Isokinetic apparatus should be regarded only as an adjunct to functional rehabilitation as movements are isolated to specific directions.

Progression of exercises is determined by the control of movement, presence of pain or related symptoms, and never by time. Further, pelvic control is important for optimal timing during any biomechanical sequence. Pelvic position whilst performing any exercises should therefore be monitored and corrected. Trunk strengthening, including abdominal setting exercises, and lower extremity strength and endurance work should be included into the programme.

6. Functional rehabilitation

As the aim of rehabilitation is to return the athlete to previous performance levels, exercises involving the biomechanics of the specific activity must be incorporated into the programme. Gymnasts will need weight-

bearing exercises in various positions. Swimmers can work against resistance in the prone position. Plyometrics must be introduced gradually for throwers and racquet sports (Fig. 9). A complete programme has been recently described elsewhere (Wilk et al 1993).

Fig. 9. *Throwing is gradually introduced. Emphasis should be placed on muscle firing pattern and scapular stabilization.*

Rehabilitation of the shoulder may appear very complex, but if the functional biomechanics are followed and restored, it is very logical and poses a great challenge. Throughout the programme, the normal restraints of healing must be considered, and signs and symptoms must be re-assessed constantly. The programme should employ synchrony of muscle firing and correct movement patterns to prevent return of symptoms and for improved performance. Therapists need to be aware of all possible causes of dysfunction and if patients do not respond to treatment within expectations, they must be referred for further investigations as soon as possible. For the earliest possible return to optimal performance levels, communication between all members of the medical team remains of paramount importance.

REFERENCES

Ayub E (1992) Posture and the upper quadrant. In: Donatelli R (Ed) Physical therapy of the shoulder. Churchill Livingstone.

Butler DS (1991) Mobilization of the nervous system. Churchill Livingstone.

Cyriax J (1980) Textbook of orthopaedic medicine Volume 2. Bailliere Tindall.

Dickoff-Hoffman (1994) Neuromuscular control exercises for shoulder instability. In Andrews JR, Wilk KE (Ed) The athlete's shoulder. Churchill Livingstone.

Dillman CJ, Fleisig GS, Andrews JR (1993) Biomechanics of pitching with emphasis upon shoulder kinematics. JOSPT 18: 402-408.

Jobe FW, Pink M (1993) Classification and treatment of shoulder dysfunction in the overhead athlete. JOSPT 18: 427-432.

Kamkar A, Irrgang JJ, Whitney SL (1993) Nonoperative management of secondary shoulder impingement syndrome. JOSPT 17: 221-224.

Knott M, Voss DE (1968) Proprioceptive neuromuscular facilitation. Harper & Row.

Litchfield R et al (1993) Rehabilitation for the overhead athlete. JOSPT 18: 433-441.

Magarey M, Jones M (1992) Clinical diagnosis and management of minor shoulder instability. Austr J Physio 38: 269-280.

Maitland GD (1991) Peripheral Manipulation. Butterworth Heineman.

Maitland GD (1987) Vertebral Manipulation. Butterworth Heineman.

Malone TR (1994) Elements of a standardized shoulder examination. In Andrews JR, Wilk KE (Ed) The athlete's shoulder. Churchill Livingstone.

Pappas AM et al (1985) Rehabilitation of the pitching shoulder. Am J Sports Med 13: 223-235.

Travell JG, Simons DG (1983) Myofascial Pain and Dysfunction. The trigger point manual Volume 1. Williams & Wilkins.

Wilk KE, Arrigo C (1993) Current concept in the rehabilitation of the athletic shoulder. JOSPT 18: 365-378.

Wilk KE et al (1993) Stretch-shortening drills for the upper extremities: theory and clinical application. JOSPT 17: 225-239. □

(From page 18)

Other Lesions

There are other problems which may not be particular to athletes but can trouble them:

Calcific tendinitis

In resistant cases arthroscopic removal of calcific deposits from the rotator cuff is the method of choice.

Frozen shoulder (adhesive capsulitis)

Arthroscopic debridement and capsulotomy can be very effective.

Conclusion

From the above it is evident that the arthroscope has become an effective tool when dealing with shoulder problems. It has become essential for a surgeon dealing with the athlete's shoulder to master arthroscopic techniques.

SUGGESTED READING

Burkhart SS: Reconciling the Paradox of Rotator Cuff Repair versus Debridement. Arthroscopy 1994; 10: 4-19.

Bigliani LU: Arthroscopic Resection of the Distal Clavicle. Orthopaedic Clinics of North America 1993; 24: 133-141.

Kay SP: Arthroscopic Distal Clavicle Excision. Clin Orthop Rel Res 1994; 301: 181-184.

Re LP: Management of Rotator Cuff Calcifications. Orthopaedic Clinics of North America 1993; 24: 125-131.

Snyder SJ: SLAP lesions of the Shoulder. Arthroscopy 1990; 6: 274. □

A survey to determine types and dosages of Anabolic Androgenic Steroids used by competitive bodybuilders in South Africa

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Abstract

A questionnaire was sent to all bodybuilders affiliated to two Provincial Bodybuilding Associations (n = 249). Out of the 80 completed returned questionnaires, 30 bodybuilders admitted to using anabolic androgenic steroids (AAS). From this sample the AAS which were most frequently reported used were nandrolone decanoate (Deca-durabolin®) (83%), oxymetholone (Anapolon-50®) (63%), testosterone cypionate (Depo-testosterone®) (57%), ethylestrenol (Orabolin®) (47%), methenolone enanthate (Primobolan®) (30%) and testosterone propionate (Sustanon-250®) (40%). The maximum reported dosages used by the bodybuilders exceeded the maximum recommended clinical dosages by up to 18 times. Ninety-seven percent of the users of AAS reported significant increases in body weight and strength while on a course of AAS. Most users obtained the AAS from peers in the gymnasium (63%). Seventy-three percent used more than one drug simultaneously ("stacking") and increased and then decreased their dosages ("pyramiding"). Forty percent of the users were in favour of AAS production being limited to prevent anyone having an unfair advantage in body-building competitions. This suggests there is pressure for bodybuilders to use these drugs. This study confirms the anecdotal evidence that AAS are used by bodybuilders in doses which greatly exceed the recommended clinical doses.

Introduction

Anabolic androgenic steroids (AAS) were reportedly first used to enhance athletic performance in the mid 1950's.[1] Since that time the use of AAS has increased, being used by athletes in a wide variety of sports,[2], particularly bodybuilders and powerlifters.[3] Although it has been reported that powerlifters in South Africa self-administer anabolic steroids in suprapharmacological doses,[4] not much is known about the use of AAS by bodybuilders in South Africa.

Secrecy surrounds the use of AAS in bodybuilders not only because of the possibility of large fines and possible prison sentences (South African Parliament, Act 101 of 1965), but also because of the controversy surrounding the use of AAS.[1] Negative publicity has also made bodybuilders secretive about the types and dosages of AAS used (Underground Steroid Handbook). Therefore, little is known about the types and dosages of AAS used, although it is apparent that bodybuilders in the USA and Europe use AAS in dosages which exceed dosages for clinical use[5,6,7,8,9,10] Furthermore, it has been shown in these studies there is no consistency in either the type or dosage of AAS used by bodybuilders. This is possibly because bodybuilders obtain their information on AAS from either non-scientific sources (Underground Steroid Handbook) or from peers in the gymnasium.[3]

In South Africa, there are no data available about the different types and dosages of AAS used by bodybuilders. Therefore, the aim of this survey was to determine the types and dosages of AAS used among bodybuilders affiliated to two Provincial Bodybuilding Associations in South Africa.

Methods

The population surveyed in this study were all the members affiliated to either the Western Province (n = 80) or Natal Bodybuilding Associations (n = 169).

A questionnaire developed in English and Afrikaans was validated in a pilot study. The questionnaire was then sent to all members in the target population. A covering letter explaining the reason for the survey and ensuring anonymity and confidentiality was also included.

The questionnaire was divided into a general section

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and a section to be completed only by those bodybuilders that claimed to have, or were using, AAS. Four weeks after the questionnaire was sent, a follow up letter was sent to all members of the two associations to encourage those who had not returned their questionnaires to do so.

A question about the respondent's body mass and maximal strength (bench press and squat) before and after a course of AAS was included with the questionnaire.

Values are expressed as the mean ± standard deviation. A paired t-test was used to detect significant differences in the reported body weight pre- and post-AAS use, and differences in the reported maximum bench press and squat pre- and post-AAS use. Values were accepted as being different when $p < 0.05$.

Results

Of the 169 questionnaires sent to the members of the Natal Bodybuilders Association, 12 (7%) were returned uncompleted because of unknown addresses, and 49 (29%) were returned completed. Of the 80 questionnaires sent to the members of the Western Province Bodybuilding Association, 6 (8%) were returned uncompleted because of unknown addresses and 31 (39%) were returned completed. It was decided that the two regions could not be separated or compared because of the low percentage response. Therefore, all the respondents (n = 80) were grouped and analysed together (35% of the total).

Ninety-eight percent of the respondents stated they had competed in a bodybuilding contest, and 50% had achieved a 1st place in their weight category. Fifty percent of the respondents had competed at provincial level, 15% had competed at national level, and 6% had competed internationally. Seventeen percent of the total sample were female (n = 14) and 83% (n =66) were male. The average number of body-building training sessions per week was 5 ± 1 (mean ± SD).

Of the 80 completed questionnaires, 30 respondents (28 men and 2 women) admitted to having used AAS (38% of responders).

The average cost per month of AAS for one user was R500 (range R70 to R2 000) (n = 28).

The average age that AAS were first used by the subjects in this study was 27 ± 9 years (range 18 to 53 years).

The reasons for the respondents using AAS are shown in Table I. Most bodybuilders (83%) used AAS to increase their physical size (Table I). A number of respondents indicated more than one reason for using AAS.

The type and dosages of AAS used by the respondents in this study are shown in Table IIa and IIb with the percent respondents reported using these drugs. In this table the dosages used by bodybuilders are compared to the maximum recommended doses for clinical use. Nandrolone decanoate (Deca-durabolin®) was the most commonly used AAS (83%) followed by oxymetholone (Anapolon-50®) (63%) and testosterone cypionate (Depo-testosterone®) (57%). All the users of nandrolone decanoate (Deca-durabolin®) methenolone enanthate (Primobolan®) and testosterone propionate (Sustanon-250®) exceeded the maximum recommended dose for clinical use. Other AAS reportedly used in this study were: methandrostenolone (Dianabol®), oxandrolone (Anavar®) and stanozolol (Winstrol®), but these were not reported in Table II because dosages were not given. These drugs are not available in South Africa so it can only be assumed that they were brought into the country.

TABLE I
Reasons for bodybuilders in this sample using AAS (n = 30)

REASON	% of users
Increased physical size	83
To remain competitive	70
Increased strength	43
Improved endurance	30
Increased resistance to injury	23
Improved resistance to fatigue	17

Nine of the respondents reported using the veterinary anabolic steroids nandrolone decanoate (Tribolin®) and boldenone undecylenate (Vebenyl®). These have not been included with Table II because the dosages were not reported.

Drugs were also used by the respondents to prevent the feminising effects during or after AAS use. Tamoxifen (Nolvadex® and Neophedan®) was used by 34% of the respondents and human chorionic gonadotropin (APL® and Pregnyl®) was used by 30% of the respondents.

The reported subjective changes and negative side effects experienced by the bodybuilders using AAS in this study are shown in Table III. Nearly all users (97%) reported an increase in body size and muscular strength. Both the female respondents reported deepening of their voices and increased clitoral size.

Seventy-three percent of the respondents stated they "stacked" the drugs and "pyramided" the dosages. Seventy % of the respondents used oral and injectable forms of AAS concurrently.

Bodybuilders use AAS in cycles which last between 8-10 weeks, although this may vary (Underground Steroid Handbook). The bodybuilders using AAS in this survey had each taken an average of 5 ± 4 cycles (mean ± SD) (n = 30).

The average reported body weight of the respondents before a cycle of AAS was 79 ± 13 kg. Subjects reported that their weight after a cycle of AAS increased to 88 ± 13 kg ($p < 0.05$, n = 28).

There was also a reported increase in strength following a cycle of AAS. Average maximum bench press reported before a cycle of AAS was 104 ± 28 kg which increased to 133 ± 32kg after a cycle ($p < 0.001$; n = 28). The reported average maximum squat before a cycle of AAS was 135 ± 42 kg, which increased to 177 ± 54 kg after a cycle ($p < 0.005$; n = 27).

Table IV depicts the source where the AAS users in this study obtained their drugs. Most users of AAS obtain their drugs from peers in the gymnasium (63%). More than one source was often used to obtain the AAS (Table IV).

TABLE IIa
Types, % users, and the range of doses of oral AAS used in the sample of bodybuilders (n = 30). The drugs are classified into anabolic or androgenic according to MIMS Desk Reference. Doses in this sample of bodybuilders are compared to the maximum recommended clinical dose (Mims Desk Reference) and the percent of responders exceeding this dose are listed.

NAME OF AAS	% users	Reported dose range (mg/day) Min	Max	Max recommended clinical dose (mg/day)	% body-builders exceeding clinical doses
ORAL ANABOLICS (per day)					
Anapolon-50® (50mg/tab) (oxymetholone)	63	50	350	400* (for 80 kg person)	0
Orabolin® (2mg/tab) (ethylestrenol)	47	2	20	4	79
Primobolan® (5 and 25 mg/tab) (methenolone acetate)	30	10	125	20	79
ORAL ANDROGENIC (per day)					
Proviron® (25mg/tab) (mesterolone)	33	25	100	75	11
Androxon® (40mg/tab) (testosterone undecanoate)	20	40	200	160	17
Halotestin® (5mg/tab) (fluoxymesterone)	7	-	10	10	0

TABLE IIb
Types, % users, and the range of doses of injectable AAS used in the sample of bodybuilders (n = 30). The drugs are classified into anabolic or androgenic according to MIMS Desk Reference. Doses in this sample of bodybuilders are compared to the maximum recommended clinical dose (Mims Desk Reference) and the percent of responders exceeding this dose are listed.

NAME OF AAS	% users	Reported dose range (mg/week) Min	Max	Max recommended clinical dose (mg/week)	% body-builders exceeding clinical doses
INJECTABLE ANABOLICS					
Deca-durabolin® (25 and 50mg/ml) (nandrolone decanoate)	83	25	300	17	100
Primobolan® (100mg/ml) (methenolone enanthate)	30	100	200	50	100
Durabolin® (25mg/ml) (nandrolone phenylpropionate)	23	50	300	50	71
INJECTABLE ANDROGENIC					
Depo-testosterone® (100mg/ml) (testosterone cypionate)	57	100	400	100	65
Sustanon-250® (250mg/ml) (testosterone propionate)	40	250	750	83	100

TABLE III
Subjective effects experienced by the users of AAS in this sample (n = 30).

SUBJECTIVE EFFECTS	% of users
Increased strength	97
Improved size and appearance	97
Improved resistance to fatigue	73
Increased endurance	70
Increased appetite	70
Increased sex drive	60
Increased water retention	60
Disturbance of normal sleep pattern	40
Less prone to injury	37
Increased body hair	37
Decreased sex drive	33
Testicular atrophy	33
Greater number of headaches	27
Increased acne	23
Deepening of voice	20
Gynaecomastia	17
Pins and needles	17
Nosebleeds	7
Stopped growing	3
Increased clitoral size (n = 2)	100

In 1992, AAS were re-classified to schedule 5 drugs. Penalties for an infringement of drugs in this class may be up to a R40 000 fine or 10 years in prison (South African Parliament, Act 101 of 1965). Of the respondents using AAS in this study, 37% stated this change in the law had affected their decision to take AAS and 60% stated AAS were now more difficult to get. Forty percent of the users felt that AAS production should be stopped completely.

TABLE IV
The sources used by bodybuilders in this study (n = 30) to obtain AAS.

SOURCE	% of users
Gymnasium friends	63
Doctor (other than family doctor)	27
Pharmacist	23
Family doctor	17
Gymnasium owner/instructor	3

Discussion

The most important finding from this study was that except for oxymetholone (Anapolon-50®) and fluoxymesterone (Halotestin®), bodybuilders in South Africa use doses of AAS which exceed the recommended dose for clinical use. This is comparable to bodybuilders in the USA who used up to 4 times the recommended clinical doses.[10]

Furthermore, this study confirmed that most bodybuilders reported to "pyramid" the dosage and "stack" types of steroids which agrees with previously reported information (Underground Steroid Handbook,[5, 10]).

The use of tamoxifen (Nolavadex® and Neophedan®) by 34% respondents and human chorionic gonadotropin (APL® and Pregnyl®) by 30% of the respondents indicates that bodybuilders try to avoid side-effects such as gynaecomastia and testicular atrophy during and after AAS.[11] Mesterolone (Proviron®) was also used by 33% of the respondents to reduce the feminising effects of AAS (Underground Steroid Handbook).

In reported clinical trials there is no consensus on the effect of AAS on body mass and strength changes.[1,2] Despite this controversy in the scientific press, bodybuilders believe that AAS improve strength and body mass (Underground Steroid Handbook). In this study both the reported body mass and strength increased significantly after a cycle of AAS. Although these are subjective data, it does indicate that bodybuilders do believe AAS cause an increase in body mass and strength.

According to the literature[5] some most commonly used AAS are veterinary preparations. In this survey veterinary preparations (Tribolin® and Vebenyl®) were used by 30% of the bodybuilders. These drugs are marketed for use in horses.

The reason for bodybuilders using them is that the preparation Tribolin® (veterinary preparation) for example, contains nandrolone decanoate, which is also in Deca-durabolin® (human preparation). Furthermore, the veterinary drugs are cheaper. However, veterinary preparations are considered to be highly toxic and dangerous to humans (Underground Steroid Handbook,[5]) because of the impurities which may occur in these drugs resulting from the less stringent quality control in their manufacture.

All the results of this study were based on the respondent's (35%) answers to the questionnaire. The anonymous nature of the questionnaire made it impossible to characterize the non-responders in the study. It can therefore not be assumed that the bodybuilders in this study are necessarily representative of bodybuilders in South Africa.

In summary this study documents the reported use of anabolic androgenic steroids in a selected sample of bodybuilders in South Africa, and describes the types and dosages of drugs used. This study agrees with reports from the USA that doses of AAS used by bodybuilders exceed recommended maximum doses for clinical use. Clearly users of AAS believe the drugs are offering them some advantage at these high doses. Although the adverse side-effects of AAS in these high doses have been well characterized,[1,4,5,6,11,12] this does not seem to deter the users. This can possibly be explained by the fact that although the bodybuilders are well informed about the side-effects of AAS, they are pressurized into using the drugs to remain competitive. This is confirmed by the respondents in this study (40%) who were in favour of AAS production being limited to prevent a possible unfair advantage by AAS users in bodybuilding competitions. Although the new legislation on AAS has had some effect on their avail-

ability, tighter regulation in the production and distribution of use of AAS is needed. The South African Bodybuilding Association also needs to take a firmer stance against the use of AAS by bodybuilders affiliated to their organization.

REFERENCES

1. *Haupt HA and Rovere GD. Anabolic Steroids: A review of the literature. Am J Sports Med 1984; 12: 469-484.*
2. *Elashoff JD, Jacknow AD, Shain SG and Braunstein GD. Effects of anabolic-androgenic steroids on muscular strength. Ann Int Med 1991; 115: 387-393.*
3. *Goldman B, Bush P and Klatz R. Death in the locker room. Steroids and Sport. Icarus Press Inc, South Bend, Indiana, USA, 1984.*
4. *Cohen JC, Noakes T and Benade AJS. Hypercholesterolemia in male power lifters using anabolic-androgenic steroids. Phys Sportsmed 1988; 16: 49-55.*
5. *Wilson JD. Androgen abuse by athletes. Endocrine Rev 1988; 9: 181-199.*
6. *Wilson JD and Griffin JE. The use and misuse of androgens. Metabolism 1980; 29: 1278-1294.*
7. *Alen M and Hakkinen K. Physical health and fitness of an elite bodybuilder during 1 year of self-administration of testosterone and anabolic steroids. A case study. Int J Sports Med 1985; 6: 24-29.*
8. *Forbes GB. The effect of anabolic steroids on lean body mass: The dose response curve. Metabolism 1985; 34: 571-573.*
9. *Alen M, Hakkinen K and Komi PV. Changes in neuromuscular performance and muscular fibre characteristics of elite power athletes self administering androgenic and anabolic steroids. Acta Physiol Scand 1984; 122: 534-544.*
10. *Burkett LN and Falduto MT. Steroid use by athletes in a metropolitan area. Phys Sportsmed 1984; 12: 69-74.*
11. *Friedl K and Yesalis CD. Self treatment of gynaecomastia in bodybuilders who use anabolic steroids. Phys Sportsmed 1989; 17: 67-79.*
12. *Hickson RC, Ball KL and Falduto MT. Adverse effects of anabolic steroids. Med. Toxicol. Adverse Drug Exp. 1989; 4: 254-271.* □

A profile of Biokinetic Services in South Africa from 1988 to 1991

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Abstract

A survey was undertaken to establish the nature and extent of Biokinetics services in the Republic of South Africa for the years 1988-1991. Questionnaires were sent to all Biokinetic centres and practices. The results show that Biokineticists willing to enter full time practice (as oposed to those working part-time) increased from 45% in 1988 to 73% in 1991. A growing trend in patient numbers was also experienced i.e. from 5 736 (286/centre) in 1988 to 11 241 (750/centre) in 1991. Of the patients seen by Biokineticists, 41% were referred by other medical disciplines. The highest proportion of referrals came from Orthopaedic Surgeons (34,9% in 1991) followed by General Practitioners (18,2% in 1991) and Physiotherapists (13,0% in 1991). These results illustrate the growth of Biokinetics as a member of the multi-disciplinary health team.

Introduction

On 9 September 1983, the profession Biokinetics was formally announced in the Government Gazette.[1] It can therefore be regarded as a relatively young member of the health profession. Biokinetics is defined as "The profession that deals with scientifically founded and controlled physical activities with regard to the following: 1. Promoting physical performance; 2. Prevention of ailments; 3. Final phase rehabilitation; and 4. Physical selection."[2] Biokinetics thus aims to promote quality of life by means of physical assessment and prescription of rehabilitative and preventative exercise. The benefits of physical exercise has been well researched and documented notably, on 19 February 1992 the American Heart Association issued an official position statement recognising inactivity as a risk factor for coronary artery disease.[3]

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The aim of this research project launched, in 1988, is to monitor the growth of Biokinetics and the support it has gained from other members of the medical profession. The results of the last four years are now available and this article attempts to summarize these findings.

Methods and procedures

In January of each year, from 1989 to 1992, questionnaires were sent to all Biokinetic centres and practices. Respondents were requested to fill out the questionnaires using patient/subject data pertaining to the previous year. Over the four years of the study the number of responses were as follows. 1988 = 20; 1989 = 8; 1990 = 16; and 1991 = 15. Because some centres and practices employed more than one person the actual number of Biokineticists and Assistant Biokineticists that were involved were: 1988 = 33; 1989 = 19; 1990 = 37; and 1991 = 37. The reason for the drop in responses in 1989 may be attributed to a change in the questionnaire i.e. in addition to the existing questionnaire respondents were asked to categorise orthopaedic cases according to the site of injury and information on referral of patients. It seems as if the respondents had difficulty in completing the more comprehensive questionnaire the first year it was introduced. Since 1989 the questionnaire remained unchanged and the number of responses increased possibly due to better record keeping during the previous year.

The questionnaire involved four categories i.e. 1. To determine the ratio of full time and part time practices; 2. To determine the patient numbers seen by Biokineticists; 3. To determine the extent and nature of referrals to Biokineticists from members of the medical profession; and 4. To determine the nature of services rendered by Biokineticists.

Results and discussion

Ratio of full-time to part-time practices

The ratio of part time vs full-time practices changed appreciably over the four years since 1988. The 1988 survey showed that 45% of all practices were full-time

practices and 55% part time practices. After a drop to 38% in 1989 (possibly due to the low response rate as discussed in the previous section) the full time practices increased to 56% in 1990 and 73% in 1991. This points to a growing confidence in the viability of Biokinetics as a full time profession despite the high costs of setting up a practice.

Patient numbers seen by Biokineticists

The total number of patients seen at the respective Biokinetic centres that responded over the 4 years was as follows: 1988 = 5 736; 1989 = 1 122; 1990 = 9 582; and 1991 = 11 241. The average number of patients seen per centre was calculated as follows: 1988 = 286; 1989 = 140; 1990 = 599; and 1991 = 750. Apart from 1989 (possibly the results are affected by the low response) these results point to a growing demand for Biokinetic services.

Referrals from other members of the medical profession

Being part of a multi-disciplinary team, Biokineticists place a high emphasis on cooperation with other medical and para-medical professions. The survey for 1988 showed that 31,1% of the total patients seen by Biokineticists were referred by other medical professions. The figures for the following years were: 1989 = 39,8%; 1990 = 49,6%; and 1991 = 43,1%. This indicates positive cooperation between Biokineticists and other medical professionals. In response to the questionnaire, views regarding the cooperation between Biokinetics and other disciplines varied from excellent to poor, suggesting that although many members of the medical profession cooperate with Biokineticists, a lack of awareness still exists. It is hoped that this article might help to promote greater cooperation in order to benefit the patient.

Knowledge regarding the type of patient referrals Bio-

TABLE II

Profile of patient cases seen by Biokineticists during 1988-1991.

	1988 (%)	*1989 (%)*	*1990 (%)*	*1991 (%)*
ORTHOPAEDIC	35,6	20,7	30,6	37,5
Foot/ankle		3,2	4,4	6,5
Knee		7,1	12,8	13,4
Hip		0,6	1,5	1,8
Back		7,1	7,8	9,3
Shoulder		1,7	2,6	4,2
Hand/arm		0,6	0,5	1,2
Neck		0,4	1,0	1,1
CARDIAC CONDITIONS	9,3	14,3	12,4	5,8
Coronary risk		10,8	7,2	2,2
Documented coronary condition		3,5	5,2	3,6
PRE-PARTICIPATION EVALUATION	23,8	21,5	18,6	18,9
HYPOKINETIC CONDITIONS	19,9	25,3	23,1	17,1
Hypertension	0,6	13,5	4,8	5,8
Cholesterol	0,4	1,9	6,2	3,7
Obesity	10,9	9,1	7,8	4,4
Posture	8,0	0,8	4,3	3,2
OTHER	0,8	4,7	5,0	6,6
Asthma	0,3	1,1	0,9	0,6
Diabetes	0	1,0	0,9	0,7
Arthritis	0,5	2,6	3,2	2,2
Osteoporosis	0	0	0	0,2
Emphysema	0	0	0	0,1
Psychological	0	0	0	1,2
Neurological	0	0	0	0,6
Gynaecological	0	0	0	1,0
SPORT EVALUATIONS	10,6	13,5	10,3	14,1

kineticists receive, is very important for upgrading the type of Biokinetic training that will be offered in the future. Table I gives a profile of referrals to Biokinetic practices from 1989 to 1991. The highest number of referrals came from Orthopaedic surgeons followed by General Practitioners and Physiotherapists.

TABLE I
Profile of referrals for Biokinetics from 1989 to 1991

	1989 (%)	1990 (%)	1991 (%)
Orthopaedic surgeons	13,4	26,3	34,9
General practitioners	41,0	25,3	18,2
Physiotherapists	23,9	15,8	13,0
Coaches	6,7	14,1	8,6
Cardiologists	3,5	6,6	7,1
Dieticians	1,2	2,0	6,3
Specialist physicians	1,2	5,6	4,6
Gynaecologists	1,8	0,3	3,2
Psychologists/Psychiatrists	0	0,1	2,1
Podiatrists	0	0	0,6
Occupational therapists	0	0	0,6
Nursing sisters	7,3	0,1	0,5
Chiropracters	0	0,8	0,2
Neuro-surgeons	0	3,0	0,1

Interesting trends were revealed regarding the type of work most prevalent in a Biokinetics practice (Table II). Biokineticists are predominantly engaged in orthopaedic rehabilitation, particularly rehabilitation of the knee.

Another major area is the physical evaluation of individuals, prior to their engagement in any activity, in order to assess the safety of their participation. The third area Biokineticists are actively involved in, is the prescription of exercise as a mode of rehabilitation for individuals exhibiting certain hypokinetic problems such as hypertension, high cholestrol, obesity and bad posture. The last, but progressively more demanding area of involvement, is the assessment of sportsmen in an attempt to identify possible shortcomings, optimize training, monitor progress and in so doing improve performance.

Conclusion

The research data collected thus far, clearly demonstrated that Biokinetics has gained acceptance as a member of the multi-disciplinary health team in both rehabilitative and preventative medicine. It has been established that a variety of other medical disciplines cooperate with Biokineticists. Biokinetics, being a fairly young discipline, is showing an escalating growth rate in the number of patients making use of its services.

REFERENCES

1. *Strydom GL. Biokinetika: As Professionele Dissipline — Historiese Oorsig en Ontwikkeling (uitgawe 2) Potchefstroom PU vir CHO, 1989.*
2. *Biokinetic activities as approved by the Professional Board for Medical Scientists in 1993 and submitted for publication in the Government Gazzette.*
3. *Fletcher GF et al. 1992. AHA Medical / Scientific Statements, Statement on exercise. Circulation Vol 86, No 1.* □

ERRATUM

TABLE I
Basic Skeletal muscle fibre type classifications

Skeletal Muscle Fibre Types			
	Nomenclature		
	Type I slow oxidative SO	IIA fast oxidative-glycolytic FOG	IIB fast glycolytic FG
Characteristics:			
Colour	red	red	white
Twitch	slow	fast	fast
ATPase activity	low	high	high
lost at pH	9.4-10.4	4.4-4.6	4.4
Glycogen content	low	high	high
Fatigue resistance	high	high	low
Oxidative capacity	high	high	low

An error inadvertently occured in Table 1 in the review article by K.H. Myburgh, "Muscle proteins and the contractile properties of muscle fibres". (SA Journal of Sports Medicine Vol 1 No 1: 13, 1994). The corrected table appears below.

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