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Clinical implications of scapular dyskinesis in shoulder injury: the 2013 consensus statement from the 'scapular summit'

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ABSTRACT

The second international consensus conference on the scapula was held in Lexington Kentucky. The purpose of the conference was to update, present and discuss the accumulated knowledge regarding scapular involvement in various shoulder injuries and highlight the clinical implications for the evaluation and treatment of shoulder injuries. The areas covered included the scapula and shoulder injury, the scapula and sports participation, clinical evaluation and interventions and known outcomes. Major conclusions were (1) scapular dyskinesis is present in a high percentage of most shoulder injuries; (2) the exact role of the dyskinesis in creating or exacerbating shoulder dysfunction is not clearly defined; (3) shoulder impingement symptoms are particularly affected by scapular dyskinesis; (4) scapular dyskinesis is most aptly viewed as a potential impairment to shoulder function; (5) treatment strategies for shoulder injury can be more effectively implemented by evaluation of the dyskinesis; (6) a reliable observational clinical evaluation method for dyskinesis is available and (7) rehabilitation programmes to restore scapular position and motion can be effective within a more comprehensive shoulder rehabilitation programme.

INTRODUCTION

Before detailing the key elements of the consensus, we first review the gross anatomy and basic movement patterns in the shoulder, define scapular dyskinesis and explain why it is a superior term to 'dyskinesia'. We discuss the clinical assessment and argue that treatment is likely to be primarily in the rehabilitation domain.

Gross anatomy

Effective shoulder position, motion, stability, muscle performance and motor control are heavily dependent on the scapular performance. Anatomically, the scapula is part of both the glenohumeral (GH) joint and the acromioclavicular (AC) joint, and is the interposed bony linkage between the humerus and the clavicle/axial skeleton. Physiologically, it is the stable base of origin for muscles that contribute to the dynamic GH stability and produce arm motion, and scapular stability is needed for force production from muscles arising from the scapula. Mechanically, the coordinated coupled motion between the scapula and humerus, the so-called scapulohumeral rhythm (SHR), is needed for efficient arm movement and allows for GH alignment to maximise joint stability. *BJSM* readers may find Kibler *et al*¹ useful for its detailed discussion of shoulder anatomy.

Shoulder movement including scapular dyskinesis

Knowledge regarding the role of scapula in shoulder function has been gradually accumulating. It has been difficult to track scapular motion because of the relatively deep position of the scapula, the overlying muscles and the potentially complex nature of the position and motion of the scapula during planar arm motions and functional tasks. Early studies of scapular motion in healthy individuals defined two-dimensional SHR^{2,3} and the muscle force couples involved.^{3,4} More clinically relevant three-dimensional motion was derived from early motion analysis studies using surface markers and indwelling bone pins.⁵⁻⁹ These studies established the three-dimensional motion and translation patterns and the magnitudes of motions about the established axes, and derived the normative data for scapular motions. This information could be used to clarify the altered motions and became the basis for clinical tools to assess normal and altered scapular position and motion. Recent information is being developed by the use of biplanar fluoroscopy, which provides more precise measurements without invasive methods.¹⁰

Altered scapular motion and position have been termed scapular dyskinesis. The definition of dyskinesis is the alteration of normal scapular kinematics.¹¹ 'Dys' (alteration of) 'kinesis' (motion) is a general term that reflects the loss of normal control of scapular motion. An alternative term that is often used interchangeably is 'dyskinesia'. Dyskinesia is usually applied to abnormally active (voluntary) movements mediated by neurologically controlled factors such as tardive dyskinesia. Since there are many other factors that can cause the altered position and motion, such as clavicle fractures, AC joint separations and muscle detachments, the more inclusive term dyskinesis is preferred.¹¹ Dyskinesis by itself is not an injury or a musculoskeletal diagnosis.¹² Dyskinesis has been hypothesised to relate to changes in GH angulation, AC joint strain, subacromial space dimension, shoulder muscle activation and humeral position and motion.

Causes of dyskinesis

Multiple factors may cause dyskinesis. Bony causes include thoracic kyphosis or clavicle fracture non-union or shortened mal-union. Joint causes include high grade AC instability, AC arthrosis and instability and GH joint internal derangement. Neurological causes include cervical radiculopathy,

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long thoracic or spinal accessory nerve palsy. Soft tissue mechanisms for scapular dyskinesis involve inflexibility (tightness) or intrinsic muscle problems. Inflexibility and stiffness of the pectoralis minor and biceps short head can create anterior tilt and protraction due to their pull on the coracoid.¹³ Soft tissue posterior shoulder inflexibility can lead to GH internal rotation deficit (GIRD), which creates a 'wind-up' of the scapula on the thorax with reduced humeral internal rotation and horizontal abduction. Alterations in periscapular muscle activation are related to scapular dyskinesis. Serratus anterior activation and strength is decreased in patients with impingement and shoulder pain, contributing to the loss of posterior tilt and upward rotation causing dyskinesis.¹⁴ In addition, the upper trapezius/lower trapezius force couple may be altered, with delayed onset of activation in the lower trapezius, which alters scapular upward rotation and posterior tilt. Altered scapular motion or position both decrease linear measures of the subacromial space,^{15–17} increase impingement symptoms,^{5 18} decrease rotator cuff strength,^{19–21} increase strain on the anterior GH ligaments²² and increase the risk of internal impingement.²³

Clinical assessment: does this patient have scapular dyskinesis?

Clinical assessment methods for scapular dyskinesis have been developed, involving some type of single planar measuring device²⁴ (lateral scapular slide, inclinometer) or some type of criterion-based visual observation,^{25–27} and sometimes including manual modification^{20 21 24} (scapular assistance (SAT) and scapular retraction/reposition tests (SRT)). The current recommendation for clinical assessment based on a prior consensus meeting¹¹ is the use of dynamic scapular dyskinesis tests (SDTs). Specifically, clinical observation of the medial and inferior scapular borders for winging or medial border prominence, lack of a smooth coordinated movement as exemplified by early scapular elevation or shrugging during ascending arm forward flexion, and rapid downward rotation during arm lowering from full flexion. The motion is then characterised as dyskinesis as a 'yes' (presence of deviation or dysrhythmia/asymmetry bilaterally) or "no (no presence). This method has been shown to be reliable among observers^{26 27} and has acceptable clinical utility.²⁷

Scapular dyskinesis may be found in association with many types of shoulder pathologies,^{28 29} although the exact relationship between dyskinesis and clinical pathology is not clear. Scapular dyskinesis may be the cause or the result of a shoulder injury, exacerbate shoulder symptoms or adversely affect treatment or outcomes. In cases of nerve injury, fracture, AC separation or muscle detachment, the injury creates the dyskinesis which affects shoulder function.²⁹ In other cases, such as rotator cuff disease, labral injury and multidirectional instability (MDI), it may be that dyskinesis is the causative, creating pathomechanics that predispose the arm to injury, or it may be response to the injury, creating pathomechanics that increase the dysfunction.²⁹

Treatment of scapular dyskinesis

There are numerous specific treatment strategies to improve shoulder pain and functional loss. To date, the large majority is related to rehabilitation since scapular dyskinesis is likely due in large part to alterations in muscle activity, flexibility and/or balance.^{11 30–32} However, surgical treatments, either relating directly to injuries around the scapula (scapular muscle detachment, A-C separation) or indirectly to G-H joint internal derangements may be appropriate. Much more work is needed

to be carried out to adequately understand the content and application of the various treatment options.

We now detail the consensus statement relating to the following four themes—scapula and shoulder injury, scapula and sports participation, clinical evaluation of the scapula and interventions and resultant outcomes as well as summarising the clinical implications and applicability of specific examination techniques and interventions for scapular dysfunction.

SCAPULA AND SHOULDER INJURY

What is known and what is not known

There is substantial evidence of scapular kinematic abnormalities in persons with shoulder pain,^{28 29 33 34} across a variety of shoulder pathologies, and this body of literature continues to grow.³⁵ Three-dimensional scapular kinematic patterns during normal arm elevation are well described to include upward rotation, posterior tilting and varying internal/external rotation dependent on the plane and angle of elevation,^{9 36} while acknowledging substantial individual subject variability and variations in measurement approaches. Increasingly, literature is providing greater knowledge on scapular kinematics in specific populations.^{37–40} This is casting doubt on current theories regarding external and internal impingement.

Impingement

Greater knowledge is available regarding the three-dimensional proximity of the rotator cuff to the acromion during arm elevation.^{10 41} It is increasingly apparent that two-dimensional or three-dimensional representations of minimal acromiohumeral distance are not fully characterising the complex relationships between shoulder kinematics and rotator cuff mechanical impingement risk,^{10 41 42} as these minimal distances are not consistent with rotator cuff tendon proximity to potential impinging structures. The acromiohumeral distances are typically minimised at 90° humerothoracic elevation, while the supraspinatus humeral insertion has cleared the undersurface of the anterolateral acromion earlier in the range of humerothoracic elevation.^{10 41} The rotator cuff is 'available' for impingement under the acromion below approximately 70° of arm elevation,¹⁰ thus impingement of the tendon may occur, but no direct imaging has confirmed this. Further mechanistic studies are needed to determine what structures and pathomechanics are responsible for the symptoms in patients with rotator cuff disease. It may be that dynamic alteration of the positions of the humeral head, rotator cuff and acromion, rather than a static alteration like a bone spur, may create increased pressure and/or alter the geometry of the subacromial space.⁴³ Bursal compression rather than rotator cuff compression may be the source of pain during positive impingement tests performed at angles of humeral elevation above 90° of elevation. Also, internal impingement on the glenoid may be occurring during a positive Neer test.⁴⁴

There is increasing recognition of internal impingement beyond the throwing population, and beyond the position of abduction external rotation in which it was originally identified.^{44 45} Mechanical internal impingement risk as defined by GH contact pressure and impingement area has recently been demonstrated to be affected by scapular position in a cadaver model.⁴² In this model, a scapular position of less upward rotation and increased internal rotation (protraction), commonly seen in scapular dyskinesis, increased the area of contact of the humerus with the posterior superior glenoid and increased scapular internal rotation also increased the GH contact pressure.

In patient populations diagnosed with 'shoulder impingement', however, scapular kinematic deviations that have been identified have typically been small in magnitude and inconsistent in direction.³⁴ For example, several studies have demonstrated decreased scapular upward rotation in 'shoulder impingement' populations as compared with controls,^{28 32} while others report the increased scapular upward rotation.^{38 46} These disparate findings are believed to be at least in part due to the fact that mechanical impingement is probably a physical condition rather than a clearly identifiable diagnostic entity. Anatomical and biomechanical investigations of clinical impingement tests characterise inconsistent risk from the coracoacromial arch or glenoid-labral complex relative to specific rotator cuff structures.^{44 47} Additionally, this mechanical risk during clinical impingement testing may be no greater than proximity risk during certain ranges of active arm elevation motions.⁴⁸ It is not clear what the source of the pain is during positive clinical impingement testing. A diagnostic label of 'shoulder impingement' is very broad, incorporating a variety of potential pathologies that range from local problems such as rotator cuff/long-head biceps disease, bursitis and labral pathology to distant problems such as scapular dyskinesis. It is increasingly advocated that this diagnosis is no more specific than a diagnosis of anterior or posterior shoulder pain, and no more effective in directing treatment.^{48–50} The diagnosis of 'shoulder impingement' may be one exclusion (ruling out cervical referred pain, adhesive capsulitis, GH instability, etc), while still including a complex set of clinical conditions with multifactorial aetiology.

Activation sequencing patterns and muscle performance of the muscles that stabilise the scapula are altered in patients with impingement and scapular dyskinesis. Increased upper trapezius activity, imbalance of upper trapezius/lower trapezius activation so that the lower trapezius activates later than normal and decreased serratus anterior activation have been reported in patients with impingement.^{19 28 32} Increased upper trapezius activity may be clinically observed as shrugging, a clinical sign of dyskinesis. This activation may alter scapular kinematics and produce symptoms consistent with impingement due to the lack of acromial elevation. Frequently, lower trapezius activation is inhibited or is delayed, which may clinically be observed as scapular winging, with production of impingement symptoms due to loss of acromial elevation and posterior tilt. Serratus anterior activation has been shown to be decreased in patients with impingement symptoms, potentially reducing scapular external rotation and upward rotation with arm elevation.⁵¹

A shortened pectoralis minor has been suggested to be a contributing cause of symptoms in patients with impingement,⁵² but has been shown to alter three-dimensional scapular kinematics in healthy subjects only.¹³ This tight muscle creates a position of scapular protraction at rest and may limit scapular posterior tilt or external rotation upon arm motion, potentially predisposing patients to impingement symptoms.¹³

Impingement is truly a syndrome, not a precise diagnosis, with multiple possible causative factors. Alteration of scapular kinematics can be a key component in the production of the symptoms associated with impingement. This implies a need for a comprehensive physical examination to evaluate all local causative factors (subacromial/AC pathology, shoulder rotation deficits and intra-articular pathology) and distant causative factors (kinetic chain deficits and scapular dyskinesis) that alter scapular motion and change the dimensions and pressures in the subacromial space and alter SHR.⁴³

Rotator cuff tears

Studies of patients with demonstrated rotator cuff tears have shown increased scapular upward rotation of some magnitude.^{53 54} Also, in a large prospective study of patients with MRI proven full thickness rotator cuff tears, scapular dyskinesis was identified as a major factor associated with lower functional scores.⁵⁵ It is not clear whether the observed dyskinesis is a cause, an effect or a compensation for rotator cuff pathology. If it is a cause, it could be that the increased upward rotation and posterior tilt alter the size of the subacromial space and change rotator cuff clearance under the coracoacromial arch, producing mechanical abrasion and wear; that increased anterior tilt and internal rotation will create glenoid antetilt during arm motion, predisposing the rotator cuff to internal impingement or that increased strain within the rotator cuff tendon due to decreased scapular muscle activation may increase the observed apoptotic changes within the tendon cells.^{8 28 56–59} It is known that dyskinesis causes a weakness in demonstrated rotator cuff strength due to decreased activation and that the strength deficit can be improved by correction of the dyskinesis.^{20 21} If dyskinesis is an effect, it is probably due to the inhibitory effect of pain on individual muscle activation and the disruption of normal muscle activation patterns, and on the effect of pain avoidance upon kinematic patterns.⁶⁰ If the dyskinesis is an effect of the rotator cuff disease process, the altered mechanics could be considered a negative decompensation and could be expected to exacerbate the dysfunction of the entire shoulder complex. It appears that one effect, the increased upward rotation in patients with rotator cuff tears, may be a compensation in an attempt to increase or maximise arm elevation or positioning in the face of weakened or absent rotator cuff activation. In this situation, it could be considered a positive compensation. Whatever the relationship, scapular dyskinesis has been identified in those with rotator cuff disease; therefore, it should be identified and considered in treatment.

The clinical examination can be helpful in treatment planning. A positive SAT (see Clinical Evaluation section) will demonstrate that altered scapular motion in upward rotation and anterior/posterior tilt is part of the reason for impingement symptoms. Treatment considerations in these patients should include increased flexibility in pectoralis minor and short head of the biceps, and strengthening of serratus anterior as a scapular stabiliser in retraction and lower trapezius as a retractor, if these respective impairments are present. A scapular stability series of exercises may be effective to achieve these goals.^{61 62} A positive SRT (see Clinical Evaluation section) involves repositioning the scapula in external rotation and posterior tilt²¹ or primarily retraction.²⁰ A positive examination can indicate a lack of scapular stability during loaded arm elevation, and thus involvement of muscle weakness or control. Treatment is indicated to improve scapular stability, rather than to the rotator cuff, as the first step in the rehabilitation process.

Superior labral injuries

A high incidence of association of scapular dyskinesis with labral injuries has been described.^{63 64} The altered position and motion of internal rotation and anterior tilt is believed to change GH alignment, placing increased tensile strain on the anterior ligaments,²² increases 'peel-back' of the biceps/labral complex on the glenoid,⁶⁵ potentially creates pathological internal impingement^{42 66} and weakens the rotator cuff cocontraction strength. These effects are magnified in the presence of GIRD, which creates increased protraction due to 'windup' of

the tight posterior structures in follow through. Evaluation of dyskinesia in patients with suspected labral injury will be a key component for rehabilitation. Correction of the symptoms of pain found in the modified dynamic labral shear test⁶⁷ can be frequently demonstrated by the SRT. This indicates the presence of dyskinesia as part of the pathophysiology and the need for scapular rehabilitation to improve scapular stabilisation, including mobilisation of tight anterior muscles and institution of the scapular stability series of strengthening exercises. In addition, identification of scapular dyskinesia may be an important part of the prevention process for labral injury.^{28 65}

AC separations

The AC joint, through both the coracoclavicular (CC) and AC ligaments, represents the critical link in coordinated scapuloclavicular kinematics. A-C joint disruption or injury may disrupt the synchronicity of motion in multiple planes by altering the screw axis of the coordinated motion.⁶⁸ This alteration may manifest as scapular dyskinesia and the spectrum of subsequent shoulder symptoms.

The current system of classification has led to some controversy regarding the treatment of these injuries since it does not accurately depict the pathoanatomy, does not consistently guide treatment and is not predictive of outcomes. Traditionally, conservative care has been advocated yet several recent authors have noted the shortcomings of this approach. Schlegel *et al*⁶⁹ reported on 20 athletes managed non-operatively and followed up at 1 year. There were no significant differences in the range of motion or rotational strength. Interestingly, and likely due to loss of a stable scapular base, bench press strength was decreased 17% on average and 20% were unsatisfied with their result. In another study, 34 patients with type 3 injuries were followed and evaluated by defined measures at a mean 28 months by Gumina *et al*.⁶⁸ In total, 70.6% of patients exhibited scapular dyskinesia, and of these 58.3% met criteria for SICK scapular syndrome. Patients with dyskinesia achieved significantly lower Constant and Simple Shoulder Scores.

The examination and demonstration of scapular dyskinesia can aid in the development of treatment strategies. Patients with A-C injuries can be evaluated with the role of the A-C joint in coordinated scapuloclavicular motion as the foremost concern. Careful clinical physical examination can allow discrimination among those patients who demonstrate scapular dyskinesia and those which do not. The clinical demonstration of scapular dyskinesia shows the altered scapular position and motion which are believed to be key contributors to shoulder dysfunction after this injury. These patients may be counselled that surgical treatment can help in restoring the biomechanics and improve the function. Surgical treatment should be directed towards restoring normal A-C mechanics and SHR by repair/reconstruction of both A-C and C-C ligaments. In clinical practice, it is assumed that patients who demonstrate normal scapular mechanics can usually be treated with non-operative rehabilitation.

Multidirectional instability

GH stability is based on the relative positions of the scapula and humerus and stabilising muscle activity providing functional stability rather than static anatomic stability based on joint constraints. Scapular position and motion are integral to the functional stability.

Scapular dyskinesia is frequently seen in microtraumatic or non-traumatic types of instability such as MDI. One of the salient features of MDI is that symptoms and instability occur in the mid ranges of G-H motion, where concavity/compression,

GH bony alignment and muscle activation play the most important roles, rather than at end ranges of motion, where capsuloligamentous restraints are most important. Patients with MDI often have increased scapular protraction, in some cases decreased upward rotation and simultaneous humeral head migration away from the centre of the joint as the arm moves.^{28 70 71} This position allows the humeral head to translate inferiorly out of the glenoid socket creating the instability. Altered scapular muscle activation patterns are believed to create the abnormal scapular kinematics which produces scapular protraction. Inhibition of the subscapularis, lower trapezius and serratus anterior, coupled with increased activation of pectoralis minor and latissimus dorsi, have been demonstrated to place the scapula in a protracted position.⁷¹⁻⁷³ Increased rotator cuff activation and biceps activation occur potentially as a compensation for the altered scapulo-humeral rhythm which tends to allow the humeral head to migrate away from the joint centre.⁷¹ The combination of capsular laxity, altered scapular kinematics and muscle activity during elevation is believed to cause the glenoid to be positioned on a downward angle allowing the humeral head to be predisposed to escaping inferiorly.

Evaluation for the presence or absence of scapular dyskinesia should be included as part of a comprehensive examination of the unstable shoulder. Careful observation of the resting scapular position and dynamic motion of the scapula with arm motion will demonstrate protraction in many cases of MDI. This will be especially noted in the arm positions associated with instability symptoms. The SRT, by stabilising the scapula in retraction, alters the glenoid position and decreases latissimus dorsi activation, and may decrease or eliminate the instability symptoms with arm motion. This positive test directs treatment towards the strengthening of the lower trapezius and serratus anterior and increasing flexibility in pectoralis minor and latissimus dorsi.

Scapular muscle detachment

This clinical problem is not well known or well categorised and only preliminary results have been reported.⁷⁴ The pathoanatomy appears to be an anatomical or physiological detachment of the lower trapezius and rhomboids from the spine and medial border of the scapula. The large majority of cases present after an acute traumatic tensile load, half involving seat belt restrained motor vehicle accidents, but there are multiple other causes such as throwing, catching or lifting a heavy object with the arm at full extension, pulling against a heavy object, hanging on the rim after dunking a basketball and electrical shock such as electrocution or cardioversion. The presenting symptom cluster is very uniform with early post-traumatic onset of localised pain along the medial scapular border. The pain increases in intensity as the condition evolves and averages 6.7/10 numeric pain rating at rest and 8.3/10 upon use. There are major limitations of arm use away from the body in forward flexion or overhead positions. Increased upper trapezius activity and spasm, resulting from the lack of lower trapezius activity, may create migraine-like headaches. Neck and shoulder joint symptoms may be present due to dyskinesia and will often become the focus of treatment, including surgery with infrequent positive results.

The physical examination also exhibits a consistent cluster of findings including the localised tenderness, often a noticeable and palpable soft tissue defect, either due to the detachment or the muscle atrophy, altered scapular resting position as well as dynamic dyskinesia including snapping scapula, shoulder impingement and weakness in forward flexion and clinical decrease or relief of symptoms with manual scapular corrective

manoeuvres. MRI and CT scan have not been beneficial in making the diagnosis. Consistency of the history and physical examination findings allows for a reliable clinical diagnosis. Most of these patients have had workups to rule out neurological or bony causation, and have had varying types of treatment, including local or distant surgery and various rehabilitation protocols. If they have failed an appropriate scapular rehabilitation programme and do not demonstrate other anatomic defects, surgical reattachment is indicated. This is accomplished by direct reattachment through pairs of drill holes in the medial scapular border and scapular spine.⁷⁴ The detached and scarred rhomboids are mobilised and reattached onto the dorsal aspect of the scapula about 1 cm from the medial edge. The lower trapezius is mobilised and reattached along the proximal scapular spine. Preliminary results from a 2-year follow-up of a small cohort of patients (n=72) show that pain scores following the procedure average 2.5/10, and ASES scores improve from 39/100 to 63/100. These results are durable at 2-year follow-up.

In summary, there is limited understanding of the relationships between scapular dyskinesis (position or motion) and risk of injury to the rotator cuff, glenoid labrum, subacromial bursa, coracoacromial ligament or biceps long head. This area deserves additional investigation, particularly since scapular dyskinesis has been associated with reduced shoulder function.⁷⁵ It is also unclear which (if any) of the anatomical structures noted above are the source of the pain complaints. This issue of an unknown pain generator in impingement supports the argument for a diagnostic label of shoulder pain of unknown aetiology/origin. There is a limited understanding of how specific tissue pathology relates to shoulder function, as evidenced by asymptomatic rotator cuff tears.^{76 77}

Despite the large number of investigations identifying abnormal scapular kinematics in patients with shoulder pathology, the cross-sectional nature of these investigations does not allow the determination of cause versus effect. Interestingly, recent studies that have induced subacromial pain⁷⁸ or created rotator cuff dysfunction through experimental nerve block⁷⁹ have both demonstrated increased scapular upward rotation in response to these 'perturbations'. This suggests that the presence of increased scapular upward rotation in patients may be compensatory, while decreased scapular upward rotation may be contributory to shoulder dysfunction. This premise is further supported by the descriptive 'normalization' of scapular upward rotation after rotator cuff repair.⁸⁰ Greater investigation is necessary with regard to this question of causative versus compensatory scapular alterations.

While kinematic alterations are frequently investigated, a gap in the literature is the elucidation of anatomic alterations and their relationship to shoulder pain. Since the ability to effectively model three-dimensional effects of alterations on rotator cuff tendon proximity to potential impinging structures is lacking, the specific influences of shoulder anatomical alterations have not been investigated. Anatomical alterations alone, or in combination with scapular kinematic changes, may have effects on mechanical impingement risk. Finally, there is a lack of knowledge regarding whether deficits in the complex three-dimensional motion of the scapula are most important in affecting shoulder dysfunction. Current clinically based evaluation protocols are not sensitive enough to discriminate precisely.

Future directions

While understanding that the pain generator(s) is of scientific interest, it is uncertain that this pursuit will be the most effective

in the near term in optimising treatment intervention effectiveness for patients with shoulder pain. Characterising the relationships between kinematic abnormalities and shoulder function/dysfunction may be more effective in improving intervention outcomes. This pursuit may also assist the diagnostic challenges surrounding shoulder pain of unknown aetiology. It is increasingly apparent that the diagnostic label of 'shoulder impingement syndrome' is too broad to effectively guide treatment planning. Subgrouping shoulder pain patients by specific tissue pathologies may assist in surgical treatment planning⁵⁰; however, subgrouping based on movement-based diagnostic categories⁴³ may be more effective for rehabilitative interventions. For example, subgroups of patients with posterior shoulder tightness⁸¹ would have a differentially targeted conservative treatment approach than those with GH microinstability or scapular dyskinesis.⁸² Clinical providers should recognise their 'training bias', for example, surgeons are trained to look for pathoanatomical contributors to pain and dysfunction, while rehabilitation professions look for movement-related contributors. Yet, these different professions use the same diagnostic label ('shoulder impingement') with different underlying understanding of the problems. In order to allow preventive and effective rehabilitative interventions, greater understanding of the mechanisms of rotator cuff/long-head biceps disease, labral pathology, bursitis and anterior or posterior shoulder pain is needed. Mechanical impingement is likely a physical phenomenon internally or under the coracoacromial arch; however, better methods are needed to clearly identify the relatively small group of patients who truly have mechanical compression as the primary source of their impingement symptoms. The complex relationships between kinematic and anatomical mechanisms of soft tissue stress in the GH joint should be scientifically investigated using the most current technologies available through high resolution imaging and three-dimensional musculoskeletal modelling.

SCAPULA AND SPORTS PARTICIPATION

What is known and what is not known

The scapula plays a major role in sports participation performance as a central segment in the kinetic chain.^{65 66 83-87} Overhead tasks are performed through the utilisation and integration of multiple body segments and muscles. Sequential activation of specific muscle groups resulting in the performance of a specific dynamic action is known as kinetic chain function.⁸⁸ During throwing and serving tasks, the scapula is the pivotal link between the larger centralised body segments that produce stability and generate force and the smaller localised segments of the arm that produce mobility and apply force to the ball or racquet. It is the link within the kinetic chain which allows the transfer of energy from the pelvic and trunk muscles to the overhead moving arm.

Proper utilisation of the kinetic chain allows the multiple body segments to optimally contribute to the performance or execution of the specific task. In the tennis serve, a specific set of sequential actions have been described which begin distally at the lower extremity and cease with the segments of the upper extremity.^{83 89} The most effective serve motion creates adequate knee flexion, trunk rotation and core stability which allows the scapula to fully retract for increased energy storage and transference.^{83 88} Similar use of the kinetic chain is necessary for overhead throwing in baseball.⁸³ The scapula is positioned between the trunk and the arm; to maximise its potential while minimising injury risk requires the kinetic chain links preceding the arm to be utilised appropriately. To achieve optimal scapular control,

an overhead thrower must control the trunk over the back leg, have the forearm pronated during cocking, the front leg and hips directed at the target, and hip/trunk move synchronously in rotation towards the target.⁸³ This will allow maximal scapular retraction to occur resulting in the ability to fully horizontally abduct and externally rotate the shoulder, increasing the ability to develop maximal velocity.⁸³ In both scenarios, the larger muscles and segments serve as the initiators and regulators of function. However, the alteration of a particular segment in the kinetic chain can result in either altered performance or injury to a more distal segment.^{90 91}

Clinical implications

Sports participation results in slight differences in side-to-side motion and in scapular resting position in overhead athletes.^{66 84–86} The differences are increased or decreased upward rotation, increased internal rotation and/or variable changes in anterior/posterior tilt. Recent evidence has confirmed that some groups of throwing athletes have specific compensations in position, but display the same direction of motions during arm motion.⁸⁷ These findings require that side-to-side evaluation be done to check for abnormal asymmetries, and that the observed alterations be treated only if they are found in association with injury. However, if alterations are found with injury, they should be addressed since the altered scapular positions have been hypothesised to have implications for decreases in muscle function and in injury.^{19–21 28 30 32 51 66 92–94} Muscle activation is coordinated in task-specific balanced force patterns to for stabilisation and control of dynamic coupled motion. The key scapular muscles for scapular stability and mobility are the upper and lower trapezius muscles and serratus anterior.

Swimming

Scapular muscle weakness or imbalance in overhead athletes has been shown to negatively affect the muscle's performance^{95 96} as well as neuromuscular control.^{93 97} In swimmers who have no shoulder pain, the prevalence of scapular dyskinesis increased during one single training session to 82%.⁹⁶ Weakness of the scapular muscles leads to excessive protraction of the scapula, presenting as medial border prominence and forward rounded shoulder posture, which can alter rotator cuff muscle output during arm elevation.^{19–21} This deleterious position also is presumed to reduce the subacromial space leading to increased symptoms of impingement. The medial border prominence appears to be the result of abnormal muscle activations, either due to muscle involvement of shoulder rotation tightness, pectoralis minor inflexibility, weakness, fatigue or nerve injury, and is usually treated by rehabilitation.¹¹ In addition to the altered scapular position, abnormal scapular motion can occur when muscle function is suboptimal.

Throwing athletes

Scapular dyskinesis is important as a component of the disabled throwing shoulder.⁶⁵ It is associated with labral tears,^{65 98} internal impingement (the combination of partial rotator cuff injury and labral tears)^{42 99} and elbow injuries.¹⁰⁰ It is considered as a part of the shoulder at risk, and should be checked as a part of the routine preparticipation evaluation. Common causative factors for shoulder pain in sports participation are the GIRD or total range of motion. These range of motion alterations can result from capsular, muscular and possible osseous alterations.¹⁰¹ They create scapular dyskinesis in the form of scapular protraction due to a wind up effect as the arm, while continuing into forward flexion, internal rotation and

horizontal adduction in follow through, pulls the scapula into internal rotation and anterior tilt. Since optimised scapular function is a key factor in optimal sports participation, recognition of dyskinesis and restoration of scapular retraction capability should be a standard part of injury prevention strategies. Also, scapular dyskinesis resulting from fatigue was shown to be an important factor in producing errors of arm proprioception.¹⁰² However, the exact relationship between scapular position and/or motion and injury is unclear. Abnormal scapular motion or scapular dyskinesis has been described as a non-specific response to a painful condition in the shoulder rather than a specific response to or a definite cause of specific GH pathology.³¹ Various shoulder soft tissue pathologies including impingement (internal and external),²³ anterior capsular laxity,²² labral injury⁶⁵ and rotator cuff weakness⁸³ have been found in association with scapular dyskinesis in overhead athletes complaining of shoulder pain.^{65 103} However, the confounding issue is that scapular asymmetries have been noted in overhead athletes that are asymptomatic as well as those injured. At this time, it is unknown if scapular dysfunction is a cause and/or an effect of shoulder injury in overhead athletes.

Future directions

More information is required regarding the precise role of the scapula in each sport. This could help in determining specific evaluation and treatment strategies.

CLINICAL EVALUATION

What is known and what is not known

Acknowledgement: Portions of the 'Examination' section of this paper were taken and modified with permission from McClure P, Greenberg E, Kareha S. Evaluation and management of scapular dysfunction. *Sports Med Arthrosc* 2012;20:39–48.

The goal of scapular assessment is to identify abnormal scapular motion (dyskinesis), determine any relationship between altered motion and symptoms and identify the underlying causative factors of the movement dysfunction.^{11 31 104} Clinical assessment of scapular dyskinesis is inherently challenging due to the three-dimensional nature of scapular movement and soft tissue surrounding the scapula obscuring direct measurement of bony positioning. Several methods of identifying scapular dyskinesis have been described; although many of these tests have been shown to possess adequate levels of reliability, the validity of most tests remains questionable due to a lack of direct correlation with symptoms.^{11 12} Clinical evaluation of scapular dysfunction in patients with shoulder pain should include three basic elements: (1) visual observation to determine the presence or absence of scapular dyskinesis, (2) the effect of manual correction of the scapular dysfunction on symptoms and (3) evaluation of surrounding anatomic structures that may be responsible for the observable dyskinesis.¹¹

The Lateral Scapular Slide Test (LST) is a static measurement of the side-to-side difference of the distance from the inferior angle of the scapula to the adjacent spinous process.²⁴ The validity of this test has been questioned due to the findings that both symptomatic and asymptomatic individuals will demonstrate asymmetry when measured in this manner.^{105 106} Additionally, it is possible to have symmetrical pathological dyskinesis so validity is questionable when comparison is made only to the contralateral side. A lack of validity was also found in a systematic review,¹² which found that the LST was unable to differentiate between those with and without shoulder pain.^{107 108} The static and two-dimensional nature of this test fails to fully assess the dynamic three-dimensional motion found

to occur with scapular movement.^{26 105 109} This inadequacy of measurement along with questionable validity of results requires the use of other methods of scapular assessment during clinical examination.

Visual dynamic assessment schemes of classifying the presence of scapular dyskinesis during shoulder motion have been developed in an attempt to resolve the issues with linear or static measures.^{25–27} These methods are considered more functional and more inclusive with the ability to judge scapular movement in three-dimensional patterns. Kibler *et al*²⁵ were the first to describe a visually based system for rating scapular dysfunction that defined three different types of motion abnormality and one normal type. Reliability values for this system were too low to support clinical use and the test was subsequently refined in two later studies using a simplified method of classification.^{26 27}

The SDT²⁶ is a visually based test for scapular dyskinesis that involves a patient performing weighted shoulder flexion and abduction movements while scapular motion is visually observed. This test consists of characterising scapular dyskinesis as absent or present and each side is rated separately. Dyskinesis is defined as the presence of either winging (prominence of any portion of the medial border or inferior angle away from the thorax) or dysrhythmia (premature, or excessive, or stuttering motion during elevation and lowering). Good inter-rater reliability of this test (75–82% agreement; weighted $\kappa=0.48–0.61$) was achieved after brief standardised online training <http://www.arcadia.edu/academic/default.aspx?id=15080>. Concurrent validity was demonstrated in a large group of overhead athletes, finding those judged as demonstrating abnormal motion using this system also demonstrated decreased scapular upward rotation, less clavicular elevation and less clavicular retraction when measured with three-dimensional motion tracking.¹⁰⁹ These results support the assertion that shoulders visually judged as having dyskinesis utilising this system demonstrate distinct alterations in three-dimensional scapular motion, particularly during flexion. However, while visually observed dyskinesis resulted in altered three-dimensional motion, those with dyskinesis were no more likely to report symptoms.¹⁰⁹

Another dynamic test developed by Uhl *et al*²⁷ used essentially the same criteria as the SDT (winging or dysrhythmia) to classify an abnormality in scapular motion into the ‘yes’ classification and normal movement was classified as ‘no’. They studied both symptomatic patients with various soft tissue pathologies as well as an asymptomatic group. The ‘yes/no’ test was found to have superior inter-rater reliability (79% agreement; $\kappa=0.41$) and demonstrated better specificity and sensitivity values when using asymmetry found with three-dimensional testing as a gold standard.²⁷ An important finding in this study was a higher frequency of multiple-plane dyskinesis during shoulder flexion in patients (54%) compared with asymptomatic subjects (14%), while no differences between groups were detected during scapular plane elevation. It appears that the optimum position for evaluating scapular dyskinesis dynamically is in forward flexion. Another interesting finding was that the prevalence of overall scapular dyskinesis was essentially equal between those with and without shoulder pain, respectively, 76% and 77% in scaption and 71% and 71% in flexion.

The presence of scapular dyskinesis or abnormal scapular position is not able to diagnose the presence or absence of shoulder pain, as reported in a recent systematic review.¹² In this systematic review, the diagnostic accuracy values indicated that some tests had moderate-to-high specificity or sensitivity, but no test provided value in shifting the pretest probability when the test was used. This is likely due in large part to the common finding

of scapular dyskinesis in those with and without shoulder pain. Moreover, scapular asymmetry (side-to-side differences) is a common finding in healthy individuals,^{27 86 110} further complicating the ability to identify when scapular motion or position is abnormal. Based on evidence to date, scapular dyskinesis (dynamic) and position (static) tests are not helpful tests to completely diagnose shoulder pain. These tests should be considered as impairment assessment tools.

Since scapular dyskinesis is a common finding, a basic problem in evaluation is deciding if the presence of scapular dyskinesis is an important abnormality-perpetuating symptom. The possibility exists that alterations of scapular motion could be compensatory strategies to avoid stress on pain-sensitive tissue. Symptom alteration tests have been developed as a way to infer scapular mal-position is driving symptoms by manually correcting scapular movement during provocation testing. If altering scapular position causes an immediate decrease in symptoms, this provides direct evidence that scapular dyskinesis is a contributing factor to shoulder symptoms. The two main symptom alteration tests are the SAT test^{24 111} and the SRT.^{20 21}

The SAT involves manually assisting scapular upward rotation during shoulder elevation and determining the effect on pain.⁶³ This test was later modified by Rabin incorporating scapular posterior tilting as well.¹¹¹ A positive test is when pain with elevation is either decreased or abolished during the assisted manoeuvre. This test has demonstrated acceptable levels of reliability.¹¹¹ It is unknown if the SAT can identify those who have scapular dyskinesis or mal-position that is perpetuating their symptoms.

The SRT involves manually positioning and stabilising the medial border of the scapula with simultaneous posterior tilting in a slightly retracted position on the thorax.^{20 21} This test was developed in order to help in identifying patients in whom strength loss in shoulder elevation may be due to a loss of proximal stability of the scapula or that the scapular mal-position may be promoting pain. The test is considered positive when the patient demonstrates a reduction of pain or an increase in shoulder elevation strength when the scapula is stabilised during isometric arm elevation in the scapular plane at 90°.^{31 63} Kibler *et al*²⁰ studied this test in symptomatic and asymptomatic subjects and found no change in pain, and all subjects had improved strength output regardless of the symptoms. The SRT was also studied in overhead athletes, where roughly half of those with pain (46/98) during impingement testing had reduced pain and 26% had a substantial increase in isometric elevation strength.²¹ It is unclear if the SRT is helpful based on these equivocal findings,^{20 21} and future studies are needed to confirm if this test can identify a subset of patients with shoulder pathology that may benefit from interventions designed to improve scapular muscle function.

Examination of the surrounding tissue should be performed in order to identify those impairment factors that may be responsible for causing the altered scapular motion. Implicated as possible contributors to the development of scapular dyskinesis are the deficits in strength or motor control of scapular-stabilising muscles,^{11 21 24 112} postural abnormalities^{113–115} and impaired flexibility.^{13 98} A comprehensive examination of these components is necessary.

Muscle strength of key scapular stabilisers can be assessed utilising standard positions and procedures described by Kendall *et al*.¹¹⁶ The key muscles to test are the serratus anterior, middle trapezius and lower trapezius, as these are muscles that have been identified with key roles for scapular stabilisation and movement.^{14 31 51 93 112 117} An important concept in testing

these muscles is that even though resistance is applied through the arm, weakness is identified by early 'breaking' of the scapula rather than the arm. In patients with rotator cuff or deltoid weakness, the arm may need to be supported and resistance applied directly to the scapula¹¹⁸ to accurately determine scapular muscle weakness.

Many authors have suggested that forward head posture and increased thoracic kyphosis may contribute to scapular protraction and lead to adaptive shortening of postural muscles or muscular strength imbalances.^{113 115 119 120} A protracted scapular position may be associated with a narrowed subacromial space,^{121 122} upright posture with increased subacromial space¹²³ and a flexed thoracic spine and forward shoulder position alters scapular motion and results in diminished force output with elevation.¹¹³ Adaptive shortening of the pectoralis minor muscle has been identified as a contributor to abnormal scapular kinematics and implicated as a factor that may contribute to shoulder impingement syndrome.^{13 124} Sahrman has described an assessment method for pectoralis minor length that involves taking a linear measurement with the patient supine from the treatment table to the posterior aspect of the acromion, with any measurement greater than 2.54 cm suggesting tightness. Although highly reliable, the validity of this method is questioned as it failed to discriminate those with shoulder pain.¹²⁵ Another assessment method that has been described involves using a tape measure or caliper to record the linear distance between the anatomic origin and insertion of the pectoralis minor muscle.¹²⁶ This measure was found to have satisfactory intrarater reliability (intraclass coefficient=0.82–0.87) and good concurrent validity, but practicality for routine clinical use is questionable. This linear measure requires careful palpation and must be normalised to the size of the individual, but a threshold for 'tightness' has not been established.

Posterior shoulder tightness (capsular or rotator cuff) has been associated with excessive scapular protraction¹²⁷ and may contribute to scapular dyskinesis.⁹⁸ Three methods of assessing posterior shoulder tightness are (1) internal rotation at 90° abduction,^{128 129} (2) spinal level reached with reaching behind the back¹³⁰ and (3) horizontal adduction with the arm at 90° flexion and the scapula blocked from moving into abduction.¹²⁷ These methods have demonstrated acceptable levels reliability for clinical use. Gerber *et al*¹³¹ showed that different parts of the posterior capsule restrict internal rotation with the arm by the side versus 90°. Therefore, authors have recommended that clinicians utilise multiple assessment methods in order to allow for a more comprehensive assessment of posterior shoulder tightness.¹³² Measurements of shoulder internal rotation are affected by humeral and glenoid version and therefore make it difficult to distinguish between soft-tissue tightness and bony alterations causing diminished internal rotation.

Future directions

Much more data need to be assembled to make the clinical evaluation more diagnostic and more specific for treatment. While these data are being assembled, the fairly comprehensive evaluation protocol outlined should be used to create a clinical picture of the variety of alterations that can be demonstrated to be part of scapular dyskinesis. This examination should be included as a routine part of the shoulder examination.

REHABILITATION

What is known and what is not known

Optimal rehabilitation of scapular dyskinesis requires addressing all of the causative factors that can create the dyskinesis and

then restoring the balance of muscle forces that allow scapular position and motion.³⁰ Causative factors can be grouped into: (1) neurological factors include long thoracic, spinal accessory and dorsal scapular nerve palsies, evaluated by appropriate muscle testing, typical scapular position and diagnostic electromyography studies¹³³; (2) joint derangement factors include labral injury, GH instability, biceps tendinitis and A-C separations³¹; (3) bone factors include clavicle and scapular fractures²⁹; (4) inflexibility factors include shoulder rotation tightness (GIRD and Total Range of Motion Deficit)¹⁰¹ and pectoralis minor inflexibility¹³; (5) muscular factors include lower trapezius and serratus anterior weakness, upper trapezius hyperactivity or scapular muscle detachment^{11 29} and (6) kinetic chain factors include hip/leg weakness and core weakness.⁸⁸ The bone and joint internal derangement factors may require surgical repair before rehabilitation may be maximally effective. They may have to be healed before restoration of muscle performance.

Restoration of the scapular muscle force couples requires core strength and facilitation by kinetic chain activation.^{61 134} This establishes the proximal stability to prevent postural perturbation and force generation and maximises activation sequencing for the scapular retraction muscles. Once the stable proximal base is established, scapular rehabilitation can proceed along specific guidelines. An algorithm guideline has been proposed that is based on restoration of soft tissue inflexibilities and maximising muscle performance.¹³⁵

Several principles guide the progression through the algorithm. Acquisition of flexibility in the muscles and joints is usually required first because the tight muscles and capsule can inhibit strength activation. Also, muscles should be trained in sport or activity specific patterns. Research has demonstrated maximal scapular muscle activation when muscles are activated in functional patterns (vs isolated), when the muscles are activated in specific diagonal patterns using kinetic chain sequencing.¹³⁶ Also, the activation is facilitated when the scapula is placed in a retracted position, thus increasing serratus anterior and lower trapezius activation as stabilisers in retraction.^{20 21} Exercises should also emphasise lower trapezius and serratus anterior activation and reduce upper trapezius activation.

Using these principles, many rehabilitation interventions can be considered.^{61 134–136} A reasonable programme could start with standing low-load/low-activation exercises with the arm below shoulder level, to meaningfully activate the scapular retractors (>20% maximal voluntary isometric contraction) without putting the arm in an impingement position.⁶² It could then progress to prone and side-lying exercises that increase the load, but still emphasise lower trapezius and serratus anterior activation over upper trapezius activation.¹⁴ Additional loads and activations can be stimulated by integrating ipsilateral and contralateral kinetic chain activation and adding distal resistance.¹⁴ Final optimisation of activation can occur through weight training emphasising proper retraction and stabilisation.¹³⁵

Although the guidelines, principles and protocols may appear to be straightforward, the actual rehabilitation process is frequently complicated and prolonged. Many patients come into rehabilitation with well-established flexibility deficits, muscle activation patterns and compensatory motions so that overcoming these obstacles requires a prolonged rehabilitation course. The lower trapezius is frequently inhibited in activation, and specific effort may be required to 'jump start' it. Tightness, spasm and hyperactivity in the upper trapezius, pectoralis minor and latissimus dorsi are frequently associated with lower trapezius inhibition, and specific therapy should address these muscles.

Finally, special attention should be paid to the GH joint because internal derangements can inhibit scapular muscle activation that may not correct until the internal derangement is corrected.

REHABILITATION OUTCOMES

What is known and what is not known

Multiple studies have identified methods to activate scapular muscles that control scapular motion and have identified effective body and scapular positions that allow optimal activation.^{14 61 62 135–139} Scapular muscle performance is improved and clinical symptoms are decreased with the use of these exercises, but equivocal results regarding a change in scapular motion, position or dyskinesia occur in patients with shoulder pain.^{33 140–142} Only two randomised clinical trials have examined the effects of a scapular focused programme by comparing it to a general shoulder rehabilitation, and the findings indicate the use of scapular exercises result in higher patient-rated outcomes.^{82 143}

Multiple clinical trials have incorporated scapular exercises within their rehabilitation programmes and have found positive patient-rated outcomes in patients with impingement syndrome.¹⁴⁴ Studies in other populations are also starting to indicate positive outcomes. A multicentre study of patients with chronic full-thickness rotator cuff tears showed that an exercise programme that included scapular exercises reduced symptoms and patients opted for no surgery in 80% of the patients.¹⁴⁵ Three studies documented that a rehabilitation programme that included scapular exercises improved symptoms and function and avoided surgery in up to 50% of patients with superior labral tears.^{146–148}

It appears that it is not only the scapular exercises but also the inclusion of the scapular exercises as part of a rehabilitation programme that may include the use of the kinetic chain is what achieves positive outcomes. When the scapular exercises are prescribed, multiple components must be emphasised, including activation sequencing, force couple activation, concentric/eccentric emphasis, strength, endurance and avoidance of unwanted patterns.

Future directions

The effects of scapular-focused exercise programmes have not been studied across all relevant shoulder diagnoses. The use of scapular exercises in the rehabilitation sequence varies in published protocols. Most place scapular emphasis at the beginning, but some do not advocate scapular exercise until later in the rehabilitation programme. Studies need to determine if there is an optimal placement, and to determine progression of exercise. It is not known why scapular dyskinesia is not consistently abolished in patients who improve scapular muscle performance. It is also not precisely known how improvements in strength, motor control or scapular motion (if altered) relate to change in symptoms and function. Finally, the exact 'dose' and load of the exercises is not known. It may be best to emphasise high-repetition/low-weight exercises,¹⁴⁹ but more information needs to be developed. More information is needed to guide treatment decision-making.

CONCLUSIONS

Biomechanical and clinical knowledge regarding the role of the scapula in shoulder function and dysfunction is growing, and the concepts regarding how to evaluate and treat scapular dyskinesia are evolving. There is enough information to emphasise the clinical implications for treating shoulder patients.

This consensus conference revealed that scapular involvement in almost all types of shoulder pathology may play an important, but as of now not a completely understood role in creating or exacerbating the shoulder dysfunction. Shoulder impingement symptoms in particular appear to be affected by scapular position and motion. Scapular dyskinesia is probably most aptly viewed as a potential impairment to optimum shoulder function and shoulder be evaluated and treated as part of the comprehensive treatment protocol.

Evaluation for scapular dyskinesia is primarily by clinical observation. A specific methodology has been identified with the tests showing to be reliable, but not specific. It can provide a good picture of the variety of alterations that can be associated with scapular dyskinesia.

Scapular rehabilitation protocols have been developed that have the potential to improve scapular muscle strength, alter scapular position and alter shoulder symptoms. They are best utilised within a comprehensive programme and should be implemented when all of the causative factors for scapular dyskinesia have been identified and addressed.

Correction notice This article has been corrected since it was published Online First. The list of collaborators was omitted in the original version.

Collaborators List of Scapular Summit meeting participants: Klaus Bak, David Ebaugh, W Ben Kibler, Paula Ludewig, Jed Kuhn, Phil McClure, Augustus Mazzocca, Lori Michener, Lane Bailey, Aaron D Sciascia, John Borstad, Ameer Seitz, Ann Cools, Tim Uhl, Mark Cote.

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