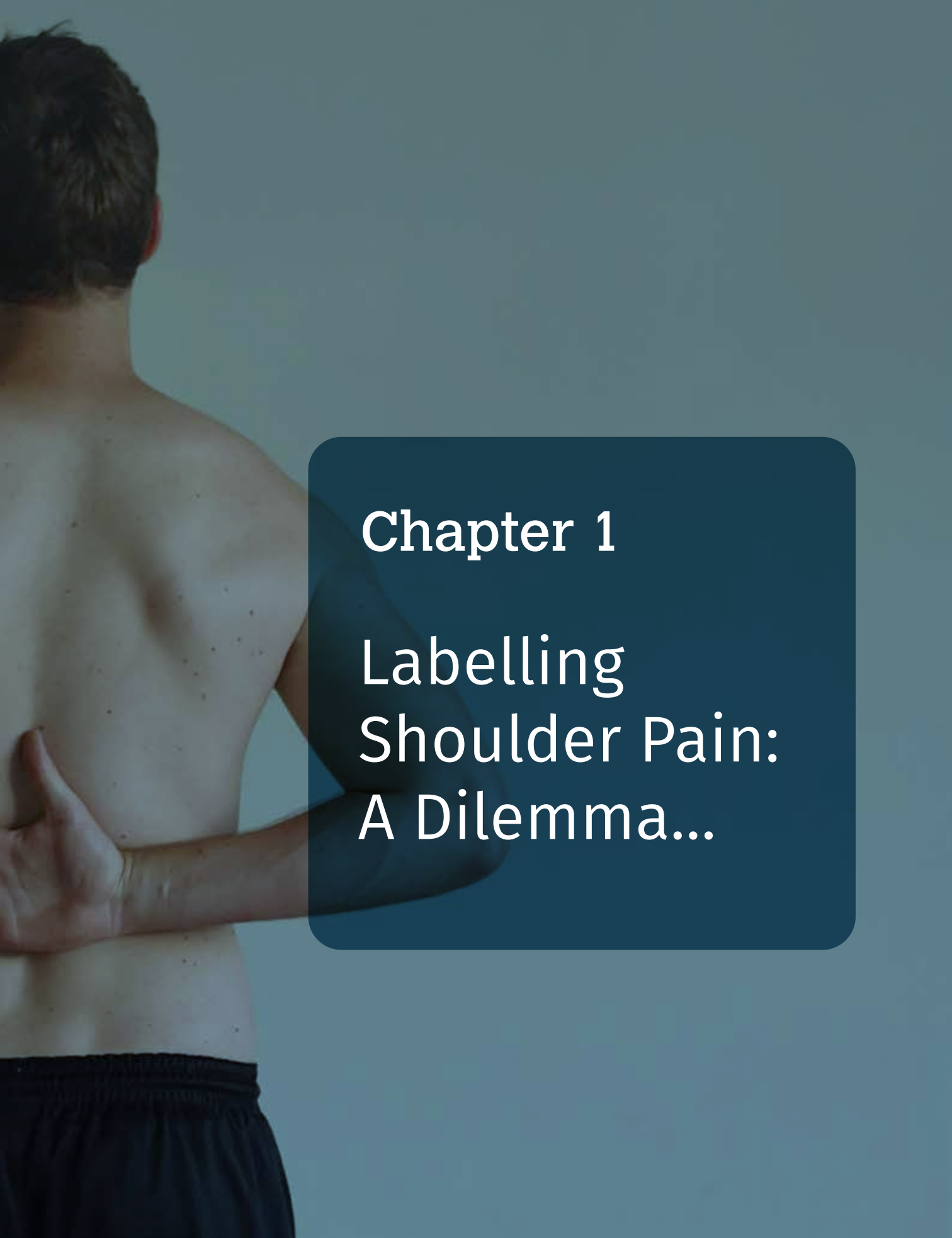

Chapter 1

Labelling Shoulder Pain: A Dilemma...

Shoulder injuries may be acute or chronic. Most acute injuries are based on a clearly identified trauma, and often lead to a clear structural diagnosis. The devil is in the definition or labelling of more chronic, non-traumatic shoulder pain, in which the inciting event is often insidious or even absent. Until recently, 'impingement' was the most commonly and frequently described clinical entity referring to chronic non-traumatic shoulder pain, in the general practice as well as in sports physiotherapy. Up to 65% of the shoulder complaints are categorized under the umbrella of impingement in the general physician's practice.[20] From a clinical perspective, a wide variety of clinical symptoms related to this diagnosis may be apparent, from inability to use the arm overhead during daily activities, up to specific complaints during shoulder loading phases of throwing-related activities. Nevertheless, the condition often results decreased functional abilities during daily life and sports and decreased participation in the professional or sports-related commitments.

The labelling of non-traumatic shoulder pain related to the structures of the subacromial space has been debated for many years. Historically, labelling of shoulder diagnoses and exploring theories about the underlying causes of shoulder pain are interrelated. Many authors use the phrase 'syndrome' describing a combination of findings, often occurring together, with an unknown or heterogeneous underlying pathogenesis. As such, Dr. Charles Neer introduced the diagnostic label of 'subacromial impingement syndrome' (SIS) of the shoulder in 1972.[16] This label was based on the mechanism of structural impingement of the structures in the subacromial space. This concept has been the dominant theory of injury to the rotator cuff tendons and other structures in the subacromial space and has served as the rationale for clinical tests, surgical procedures, and rehabilitation protocols for many years. Neer described a continuum of SIS starting with a reversible tendinitis of the rotator cuff (stage I) over an intermediate stage (stage II) to an irreversible rotator cuff tear (stage III), assuming a natural progression of a tendinitis at younger age into a tear at older age. Impingement signs and tests were described (painful arc, Neer-test and – sign – see Chapter 2) and the acromioplasty procedure was developed, with the purpose to 'raise the roof'.

However, the label of SIS is now controversial, as recent evidence suggests that this concept does not fully explain the mechanism.[11, 18] Until a few years ago, SIS was a widely accepted 'umbrella' term for a number of possible underlying structural or biomechanical causes. Throughout the years the description progressed from SIS to 'impingement related shoulder pain', or 'subacromial pain syndrome (SAPS)', with the growing opinion that 'impingement' represents a cluster of symptoms and a possible mechanism for the pain, rather than a pathoanatomic diagnosis itself. The recent tendency to expel SIS as a diagnostic label on one hand allows us to critically review our perspectives on shoulder

pain but on the other hand leads to uncertainties about the 'umbrella' we used to classify patients.[9, 10] Given this evolution and taking into account that the main goal of rehabilitation is to treat the cause of the problem and not the symptom that is the result of it, the continuous challenge is to identify the underlying cause of so-called impingement symptoms, including pain-related issues and psychosocial factors influencing the patient's symptoms and expectations. Researchers as well as clinicians have done many efforts to seek for functional, biomechanical, bio-psycho-social or structural causes of impingement symptoms, through biomechanical, cadaver or clinical studies.

To start with, impingement as such should be revisited: does it exist? If yes, is it only occurring in the subacromial space? Which structures may be impinged?

The original description of shoulder impingement refers to the mechanical encroachment of the soft tissue (mainly supraspinatus tendon and subacromial bursa) between the tuberculum majus of the humeral head and the acromion and was referred to as subacromial impingement (Figure 1.1). This encroachment was believed to occur mainly in the mid-range of motion during elevation, because by nature the size of the subacromial space decreases once 90° of elevation in any plane is achieved.[16] In more recent papers, this kind of impingement was also called 'external impingement', because the conflict occurs between one aspect of the glenohumeral joint (the humeral head) and a bony component outside the joint (the acromion).[3, 4] Similarly subcoracoid impingement may be described as an encroachment of soft tissue (mainly subscapularis) between the tuberculum minus and the coracoid (external)[6], mainly during forced internal rotation (IR) and horizontal adduction.

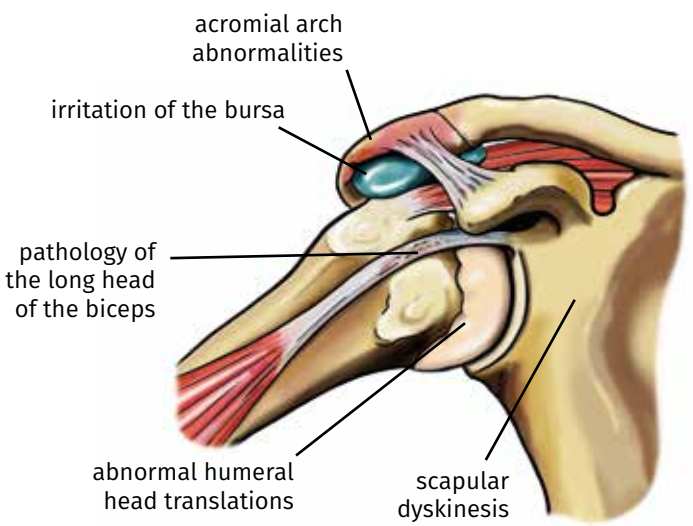

Figure 1.1

External subacromial conflict[2]

In 1991[21], 'internal impingement' was for the first time described as a conflict between the 2 components of the glenohumeral joint (humeral head on one side, labrum on the other side) with encroachment of the soft tissue (mainly rotator cuff tendons) in between these 2 bony/labral components, mainly occurring in overhead athletes. More specifically, postero-superior glenoid impingement refers to impingement of the supra- and infraspinatus tendon between the tuberculum majus and the postero-superior rim of the glenoid, occurring during the late cocking position of throwing (Figure 1.2). At the anterior aspect of the shoulder, impingement of the anterior soft tissue between the minor tubercle and the antero-superior rim of the glenoid, mainly occurring during the follow-through phase of throwing and referred to as antero-superior glenoid impingement. For the purpose of this chapter, in view of their higher prevalence, only subacromial and postero-superior impingement will be further discussed.

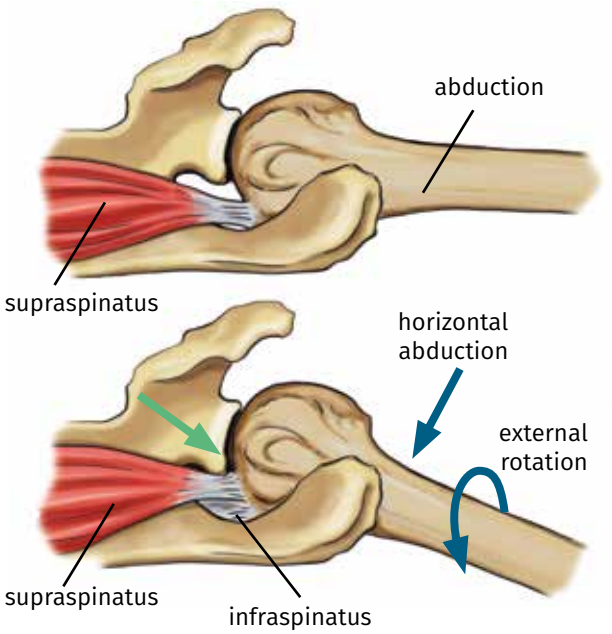

FIGURE 1.2

INTERNAL POSTERO-SUPERIOR GLENOID CONFLICT[2]

Subacromial pain may be categorized, based on the cause of the conflict, into primary and secondary conflict. Primary impingement or structural impingement is the result of a real narrowing of the available subacromial space due to structural changes in the subacromial area. These structural changes may decrease the available space, for instance as result of bony abnormalities at the level of the acromion or AC joint or increase the volume of the soft tissue in the subacromial space. Bony changes have been described as a type II or III acromion[17], spurs or osteophytes at the AC-joint. Increased or abnormal volume of the soft-tissue in the subacromial space may reflect swelling of the bursa or the Rotator Cuff (RC) tendons or calcifications in the supraspinatus.

However, it is generally accepted that there is no direct linear relationship between these structural abnormalities and symptoms of shoulder pain. On the contrary, recent literature suggests only a minimal association between the structural changes and the presence of symptoms.[13, 14] Many people with calcifications or increased thickness of the RC tendons do not have any symptoms of shoulder pain. As an example, calcifications have been found in a substantial proportion of a population of women over the age of 50 years old, with only a minority showing shoulder pain and discomfort.[15] Similarly, many patients with non-specific shoulder pain have no structural bony, ligamentous, muscular or bursal abnormalities on imaging. Therefore, the clinician should be aware that abnormalities on imaging do not always reflect the cause of the symptoms and should look for functional impairments rather than structural deficits.

Secondary impingement or functional impingement refers to the fact that the conflict between the bony components (humeral head and acromial arch) with in-between the encroachment of the soft tissue, is not structure- but function-based. From a structural perspective, the available space in the subacromial area may be sufficient to avoid impingement symptoms. However, during specific positions, movements or loads in daily life or sports activities, a temporary kinematic conflict may occur as a result of abnormal or excessive humeral head translation into superior, anterior or posterior directions, or inappropriate positioning or movements of the scapula, insufficiently elevating the acromial arch away from the humeral head. These functional deficits may be related to rotator cuff dysfunction, shoulder instability, biceps disorders, glenohumeral stiffness or scapular dyskinesis. We refer to each of the chapters describing the treatment strategy for these disorders for further analysis of the biomechanical conflict (Part 2).

The mechanism of internal impingement was originally described as occurring during the late cocking position of throwing.[21] The throwing movement consists of several phases: wind-up, cocking, late-cocking, acceleration, deceleration and follow through phase (see Part 3, Chapter 13). In the late-cocking position, the shoulder is put into a position of (1) maximal external rotation (ER), (2) maximal horizontal abduction, and (3) depending on the sport, more or less elevation/abduction. For instance, a baseball pitcher stays in the range of +/- 90° of abduction, whereas a tennis player performs the serve movement at +/- 110° of glenohumeral abduction and a volleyball player reaches up to 140° glenohumeral abduction during the smashing motion.[19] This position may, under poor biomechanical circumstances (such as minor shoulder instability, selective capsular stiffness of the posterior capsule or poor scapular alignment), lead to an instantaneous encroachment of the posterior cuff tendons against the postero-superior rim of the glenoid, thus causing temporary pain in the shoulder during throwing. Often, these athletes complain of pain at the posterior aspect

of the shoulder, in the area of the infraspinatus tendon which is often sensitive to pressure on palpation. Additionally, the external rotators may be weak, suggesting a conflicting disorder in the external rotators, being weak as well as tensed. From a biomechanical perspective, it is thought that during the late cocking position, an imbalance could occur in the tensile forces of the joint capsule, for instance as a result of anterior instability (looseness of the anterior restraint) and posterior shoulder stiffness (tightness of the posterior restraint). This may lead to increased humeral head translations, resulting in a temporary functional impingement of the posterior cuff muscles in-between the greater tubercle of the humeral head and the glenoid, (in which the tendons are compressed as a fold against the glenoid). Internal impingement is considered to be secondary/functional since it only occurs during specific shoulder positions. A phenomenon often described related to internal impingement is 'hyperangulation'[22] (Figure 1.3). Hyperangulation refers to an angle between the plane of the humerus and the plane of the scapula in the late cocking position of throwing. Under optimal circumstances, the scapula and the humerus should be aligned in the same plane, providing optimal congruence and stability during a variety of shoulder movements. However, if the humerus moves further posteriorly with respect to the plane of the scapula or if the scapula is not sufficiently moving into retraction to follow the backward movement of the arm, hyperangulation occurs, with possible intra-articular conflicts and compression of the posterior rotator cuff during the throwing position. Possible underlying impairments for this situation are glenohumeral anterior instability, posterior shoulder stiffness (both possibly changing humeral head translations with respect to the glenoid) or scapular retractor muscle weakness and/or anterior scapular muscle stiffness avoiding the scapula to sufficiently move into retraction.

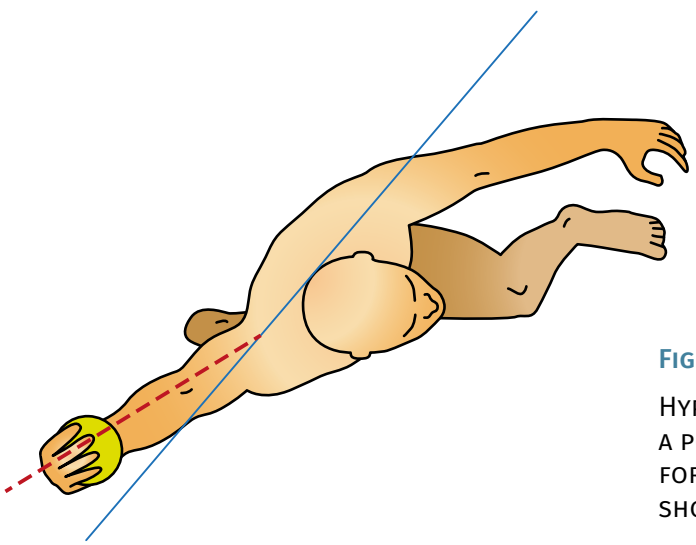

Figure 1.3

Hyperangulation as a possible mechanism for throwing-related shoulder pain