

The Mechanics of Golf Swing Related Injury: A Literature Review, Synthesis and Analysis

Kiran D. Kanwar

LPGA T&CP

Master Thesis Proposal

Abstract

Introduction. This article is a three-part literature review about the mechanical basis of golf injury. **Methods.** A comprehensive search was made of PubMed, CINAHL Complete, ProQuest Nursing & Allied Health Database, Scopus, SPORTDiscus, Physical Education Index and Google Scholar databases and of the Proceedings of the World Scientific Congresses of Golf 1990, 1994 and 1998, using relevant search terms. **Results.** Part 1 lists all the tissues commonly injured at each joint. Part 2 describes tissue-based etiology of injury at each joint, as conjectured by golf swing or other sports researchers. Part 3 reports an exemplar collection of relevant biomechanical data, to elucidate whether the implicated injury-causing positions, movements and loads actually do exist in the golf swing. **Discussion.** Typical causative mechanisms of injury at each joint/body segment are summarized. Then, based on suggestions sourced from golf injury literature, a joint-by-joint solution is described. Finally, all suggestions are compiled into recommendations for the set-up, the backswing and the downswing, to form what might be considered a “pain reducing golf swing”. Golfers of all skill levels can reduce the pain-causing movements and loads at many joints, and thus delay the onset of, or attenuate, the potential for mechanical injury.

Keywords: epidemiology, etiology, injury mechanisms, biomechanics

TABLE OF CONTENTS

	Page
Title Page	1
Abstract	2
Table of Contents	3
Chapter	
I Introduction	4
Purpose of the literature review	6
II Method	7
III Results	9
Part 1	9
Part 2	25
Part 3	47
IV Discussion	92
V Conclusion	99
VI References	101

The Mechanics of Golf Swing Related Injury: A Literature Review, Synthesis and Analysis

For any sports movement, effectiveness, efficiency and safety are three important goals. Effectiveness refers to the “ability to bring about some end result with maximum certainty”, while efficiency typically involves a “minimum outlay of energy, or of time and energy” (Wulf & Lewthwaite, 2010, p. 75). Safety, in sport, means the prevention of injury (Timpka, Finch, Goulet, Noakes, & Yammine, 2008). These three constructs should be considered simultaneously to produce any appropriate and beneficial sports movement.

In the game of golf, the terms “effectiveness” and “efficiency” are often used interchangeably: “Efficiency refers to a full swing that is able to consistently and predictably hit the ball in the desired direction for the proper distance” (Lindsay, Mantrop, & Vandervoort, 2008, p.188). Injury, meanwhile, is considered to take place when forces applied to body tissue exceeds its ability to resist them. While sports injury can be classified from many perspectives, the type of injury that is most meaningful for a golfer or golf instructor to understand the likely causation of, is one which is intrinsic - that is, a result of stresses developed within an individual’s body. Intrinsic injury is typically overuse injury, and can be acute, resulting from onetime overuse or can be chronic, and related to overuse over a longer time period (Williams, 1980). “Overuse” in golf has been defined in many ways. In simple terms it can be thought of as “excessive use” (Gosheger, G., Liem, D., Ludwig, K., Greshake, O., & Winkelmann, 2003) or “too much practice” (McCarroll, 2001), or, more definitively, can be considered to be the act of playing golf an average of eight times per month, compared to, for instance, 3.2 times per month (Batt, 1992). In golf research literature, “poor mechanics” is often considered to have a different causative mechanism of injury than intrinsic overuse, but the terms are not distinguished in typical sports injury etiology classifications. In fact, it has been said that “Although many golf

injury studies have attributed injury risk to poor swing mechanics, this hypothesis is yet to be formally tested” (Sherman & Finch, 2000).

Ultimately, therefore, it is important for a golfer or golf instructor to incorporate or teach, respectively, swing positions and movements that can produce the best results from a holistic perspective which includes both effectiveness of movement for better ball-striking, as well as for enhanced safety. Such safety should come about through reduced loads on the joints, which might help to avoid, or delay, overuse injury. Such an undertaking should ideally be guided by the extensive research that has been conducted on the golf swing.

One review of all aspects of golf research that had been made by the beginning of the 21st century (Farrally et al., 2003), found that most golfer-related research had been with respect to swing biomechanics, followed by far fewer studies in sports medicine. In other words, golf swing research to date has typically studied the golf swing from a kinematics (description of movement) or kinetics (cause of movement) perspective using motion capture technology and force plates, or by using electromyography (EMG) to understand muscle force production capability. The most commonly used outcome measure to indicate swing effectiveness has been club speed. In addition, research has typically focused on a few specific body segments, not on assessing movement at all the major joints simultaneously. Finally, in the words of Farrally et al. (2003, p. 756), “Although these studies provide information relative to a portion of the swing, they do not study the swing from a multidisciplinary perspective”, referring to disciplines such as biomechanics, physiotherapy, sports psychology and motor control.

While much is known about swing movements that are well correlated with effectiveness, especially with those that produce better club speed, less is known about the movements that may cause or exacerbate injury. Of the studies that have looked at golf injury, many have used an

epidemiological approach, listing the body segments injured and the commonly seen types of injury, and then speculated upon the causative factors such as poor biomechanics, age, overtraining, equipment, environment, fitness, hydration and nutrition, and health status (Sherman & Finch, 2000).

Few studies have attempted to formally, as opposed to anecdotally, correlate the commonly seen intrinsic, non-contact, musculoskeletal golf injuries at all body segments, with causative or exacerbating swing mechanics or technique. Sherman & Finch (1999), while referring to prior researchers who had claimed a connection between swing mechanics and injury, recommended that there was a need for a formal study to investigate the relationship between injury risk and swing mechanics.

Such an endeavor should look at golfers in general, as well as at female and senior golfers, because these latter two groups together comprise a substantial percent of the USA's total golfing population. According to the National Golf Foundation (2017), an industry-leader in collating golf demographic information, 24% of the 23.8 million people who played golf in the USA in 2016 were females. Additionally, 8.7% were either mature golfers (aged 50-64 years) or seniors (aged 65 years and above).

The purposes of this study were threefold. Firstly, to ascertain, through a review of literature, epidemiologically researched intrinsic golf swing-related injuries of all the major body segments and related joints, for all adults, as well as for females and seniors. Secondly, to describe the probable causative mechanisms of such injuries as established by physical examinations of injured golfers, or inferred from golf or other sports' research. Thirdly, to precisely determine, through a review of biomechanically researched golf swing positions,

motions and loads, which risk factors actually exist in the golf swing and are likely to cause or exacerbate commonly seen overuse golf injuries. The overall objective of this study was to develop a comprehensive resource for golf practitioners, both players and coaches, on the types and laterality of common golf injuries as well as their causative mechanisms, and to serve as a template for similar reviews in other sports.

Method

The review of literature involved a three-part process. In Part 1, the most commonly seen intrinsic golf injuries were sourced from the literature, and listed according to anatomical sites. The body segments comprised the spine; the upper limb including the shoulder, elbow and wrist; the lower limb including the hip, knee and ankle; and the ribs. Comparative details of professionals versus amateurs or females versus males are only reported if germane to a particular issue. In Part 2, a search was made for the causative factors for those types of injuries. As the etiology of golf-specific injury has not been considered for all body segments, joints and tissue-types, the information for this part of the study was obtained through golf and non-golf sports' literature. A synthesis of relevant information has been made after each section within Part 2.

Finally, for Part 3, a search was made for information from golf swing research involving kinematics and kinetics, to assess whether the injury-causing factors, as implicated in the literature, actually do exist in the golf swing. This part of the search included information from research that used motion capture, force-plate data and/or EMG research. From these golf studies, correlations were made to indicate mechanics that might cause or exacerbate injury among golfers. Each section within Part 3 is accompanied by a synthesis and analysis of the information contained in it.

This review article did not seek to distinguish between skilled and unskilled golfers, as no specific definition exists for those terms. In fact, both groups can have “poor technique” or overuse injuries because, as stated in one study (Lindsay, Versteegh, & Vandervoort, 2009, p.131), “Differences in technique even among elite predispose to injury.” It was beyond the scope of the present study to assess the quality of biomechanical evidence sourced for Part 3, as data were not extracted or analyzed for the specific purposes that the original authors had intended. Moreover, all raw data reported here have been presented exactly as published in the original journal articles, without standardizing units of measurement or the number of decimal points.

An electronic search was conducted to access all literature relevant to the purpose of this review. The databases that were searched for peer reviewed articles were PubMed, CINAHL Complete, ProQuest Nursing & Allied Health Database, Scopus, SPORTDiscus, Physical Education Index and Google Scholar. Common key words, search terms/phrases and filters used were golf, along with human, English, all adult. No restrictions were placed on publication dates. For Part 1, “epidemiology”, “incidence”, “prevalence”, “wounds and injuries”, or “range of motion” were the terms used. Further detail was added by including the name of a particular joint or body segment, from one of: cervical spine, thoracic spine, lumbar spine/low back, lumbosacral region, shoulder, elbow, wrist/hand, hip, knee and ankle. Some common injuries for the shoulder and elbow were also entered by name. For the shoulder joint, terms added were “impingement syndrome”, “rotator cuff tear” / “rotator cuff arthropathy” or “bursitis”. For the elbow joint, the specific injury terms were: “tennis elbow”, “elbow tendinopathy” or “cubital tunnel syndrome”. For Part 2, terms used were “etiology” or “cause” or “mechanism”, and the search was repeated for each joint/body segment. For Part 3, either “biomechanics” or

“biomechanical phenomena” was incorporated along with the body part-specific terms. Finally, the Proceedings of the World Scientific Congresses of Golf of 1990, 1994 and 1998 were searched, and hand-searches were performed using Google Scholar to source cross-disciplinary research.

Inclusion criteria for this review were studies with participants from healthy, adult, populations. Exclusion criteria were studies involving fitness/training programs or mathematically developed simulation models of the golf swing.

Results

A total of 869 articles were sourced from the database search. After removing duplicates, as well as articles which did not match the inclusion and exclusion criteria of the present study, 119 peer-reviewed journal articles were used to provide the evidence and data for parts 1, 2 and 3 of this study.

Part 1. Epidemiology

This section lists, by anatomical joints, epidemiological information related to intrinsic overuse golf injuries discussed in the literature. Overall there is a paucity of detail regarding laterality (lead or trail side, i.e. left or right respectively in a right-handed golfer) and tissue type. This is especially important because the golf swing is known to be an asymmetric movement (Lee et al., 2015). In the words of the authors of a systematic review on the knee joint, “Details surrounding the laterality, mechanisms and type of knee injuries that players experience are scarce” (Baker et al., 2017, p. 2636), and this is true for most joints. Only nine of the epidemiology-centric articles referenced in this study reported the laterality of injuries. Information also varies based on the populations studied. For instance, one study (McHardy, Pollard & Lou, 2007) stated that in their population of golfers, with an average age of 55 years,

there was no difference in lower back injury based on age, gender or skill level. Other studies have shown differences between golfers based on those same variables (McCarroll & Gioe 1982; McNicholas, Nielson & Knill-Jones, 1998).

One group of researchers discovered that golf-related injury low back pain (LBP) typically has a laterality, for the right-handed golfer, of right or “trail” side, (further away from target). Conversely, the high back, shoulder, elbow and wrist are most frequently injured on the left or “lead” (closer to target) side (Sugaya, Tsuchiya, Moriya, Morgan & Banks, 1998). Moreover, Nicholas, Reidy and Oleske (1998) discovered from their epidemiological survey of golf injuries, that females and higher handicap-holding golfers had a greater risk of upper extremity injury, while young and overweight golfers were more likely to suffer back pain. The following sub-sections contain injury details for each joint, along with simple explanations for medical terminology (in parentheses), all of which have been sourced from Stedman’s Medical Dictionary (2016).

Spine – lumbar and thoracic. The spine, consisting of 33 segments termed “vertebrae”, is divided into five regions – cervical, thoracic, lumbar, sacral and coccygeal - based on the general design of the vertebrae in these regions. The vertebral bodies of the cervical (except the first two), thoracic and lumbar vertebrae are separated by intervertebral discs, and the bodies are also connected, on their posterior aspects, through articulations known as facet joints. Movement of the spine is facilitated by motion at the discs and facet joint structures, while the ligaments (fibrous tissue connecting two or more bones) act to restrain excessive movement (Armstrong, 1994). Armstrong stated that typically seen golf-related LBP was either spondylogenic or discogenic. He further described spondylogenic pain as either emanating from changes to

vertebral bones or from degeneration to muscles, ligaments or fascia (fibrous tissue surrounding muscle). Discogenic pain results from intervertebral disc degeneration or herniation (protrusion).

An epidemiological study conducted in Australia (Finch, Sherman & James, 1998) was based on data collected from sports medicine clinics and hospital emergency departments. The researchers found that the lower back was the most frequently seen area of injury (in 25% of all cases seen). The lower back/lumbar spine pathologies were categorized as being either intervertebral disc or facet joint injuries, and typically resulted from overuse.

One study of elite and professional male and female Japanese golfers found that 55% of their questionnaire respondents had low back injury (Sugaya et al., 1998), with 22% of total cases being from each of the PGA (male) and LPGA (female) Tours of Japan, and 10% being from the senior PGA Tour of Japan. Of those with LBP, 51% had right-side symptoms, 28% left-side and 21% had central symptoms (all right-handed golfers), and those having right-side symptoms showed an association of pain with the impact and follow-through swing phases. The researchers found that right-handed golfers with LBP showed significantly higher right-side vertebral osteophyte (bony outgrowth/protuberance) formation and right-side facet joint osteoarthritis (OA) than non-golfing controls with LBP.

In a review article (Reed & Wadsworth, 2010) back pain was said to be caused by mechanical pain, which includes muscle strain/spasm, discogenic pain (involving an intervertebral disc), spondylogenic (degeneration of spine) pain, or pain related to facet arthropathy (pathology of a joint). In older golfers, hip OA may be a factor in LBP. Herniation of a disc can also cause LBP, as can spondylolysis (degeneration of the pars interarticularis part of a vertebra; typically seen in younger golfers), and spondylolisthesis (forward slip of a vertebra on the one below it) subsequent to bilateral spondylolysis. Facet joint pain, according to the review,

can be similar to spondylolysis, and can manifest either as pain in the joints in younger athletes or as OA in older golfers. Additionally, the article also stated that sacroiliac joint dysfunction can account for up to 40% of low back injuries and is especially common among younger, more athletic golfers. Another review article stated that when golfers are aged 50 and above, they can suffer from vertebral compression fractures (Cabri, Sousa, Kots & Barreiros, 2009). Finally, one 1993 case study article (Ekin & Sinaki 1993) reported that three postmenopausal women suffered multiple vertebral compression fractures in the thoracic and upper lumbar regions while playing golf. This latter phenomenon was ascribed to reduced bone mineral density, not to swing mechanics.

Another review article (Tucker, 2016) specified that the paraspinal muscles (adjacent to the spinal column) typically sustain tears and strains. Moreover, besides the facet joint and lumbar disc injuries seen, when there is injury to the posterior arch of a vertebra, spondylolysis may result. Typically, then, LBP can be said to include disc, facet joint, or spondylolysis-related injuries in the overuse category.

Thoracic muscle strain was referred to by Thériault and Lachance (1998) in their golf injuries review article. As mentioned earlier, compression fractures have been observed in healthy postmenopausal women, and the most common site of stress was the thoracolumbar region, which is a transition segment of the spinal column (McHardy, Pollard, Luo, 2006). One epidemiological study (Fradkin, Cameron & Gabbe, 2005) found that better golfers (median handicap 10) were also more likely to sustain injury in this region. Compression fractures were thus the main injuries reported for the thoracic region.

Spine – cervical. The neck or cervical section of the spine, according to the 1998 epidemiological study by Finch et al., frequently sustains either disc or facet joint injury, and 15% of all cases treated in their sports medicine clinics had such damage. Conversely, another epidemiological study (Sugaya et al., 1998) found that the neck or high back region, mostly on the left side of right handed golfers, was injured in 33% cases, second only to low back issues. However, the 2005 study by Fradkin et al., found that only a few neck injuries occurred, and when they did, golfers were middle aged (median age 52 years) and had higher handicaps (median handicap 22.5). The lead side of the neck is thus more likely to be injured, either through disc or facet joint damage.

Shoulder. An epidemiological study of Scottish golfers (McNicholas et al., 1998), found that in general, upper limb injuries tended to involve enthesopathies (disease at the insertion of tendons or ligaments into bone), and ligament sprains and tendinitis, especially in golfers over 40 years of age. The most proximal (nearest to the trunk) joint of the upper extremity is the shoulder or glenohumeral (GH) joint, consisting of the humerus bone of the (upper) arm, which attaches to the glenoid fossa on the lateral aspect of the scapula (shoulder girdle) and its surrounding labrum (ring of fibrocartilage around the rim of the glenoid fossa). This joint is a ball-and-socket joint allowing maximum freedom of upper limb movement, but this mobility is achieved at the expense of joint stability. The large head of the humerus articulates with the much smaller glenoid fossa on the lateral side of the scapula. The relationship has been likened to a golf ball resting on a tee that has been placed sideways. The humerus is held against the scapula mainly by the four rotator cuff (RC) muscles – suprapinatus, subscapularis, infraspinatus and teres minor - and their tendons (Andrews & Whiteside, 1994). A tendon is that part of a muscle that attaches

it to bone, but is very different from muscle in its morphology and biomechanical properties (Kumar, 2001).

The most frequently seen overuse injury to the shoulder typically involves the RC tendons. In fact, RC movement capability reduces with aging, as a result of reduced vascularity and degenerative change (Bait, 1993). The subacromial space (below the acromion process of the scapula) can then be compromised as a result of osteophyte formation and reduced muscle viability. The damage at or close to the location of the juxtaposed tendons of four RC muscles' insertions at the greater or lesser tubercle of the humerus can include tendinitis (inflammation of a tendon), subacromial bursitis (inflammation of the bursa, or synovial fluid filled sac, below the acromion process of the scapula) and impingement (tissue trapped and colliding with, or rubbing against, other tissue in a confined space), which can lead to RC tears (McCarroll, 2001).

RC tendon injury, swelling of the subacromial bursa and damage to other tissues in the subacromial region have been associated (Andrews & Whiteside, 1994) with specific positions of the shoulder when the gap between the head of the humerus and the subacromial space reduces.

One case each of impingement and RC pathology were seen in an epidemiological study that assessed 34 injured golfers (Finch et al., 1998), so that the two shoulder injuries comprised 6% of the population that was studied. Additionally, McNicholas et al. (1998) found shoulder impingement to be the most frequently seen injury in the shoulder region. According to McHardy and Pollard (2005), shoulder injury is mainly seen in the lead shoulder. They stated in their review article that shoulder pain could be found in the acromioclavicular (AC) joint region and could be from OA / arthrosis (degenerative joint change), or osteolysis (destruction of bony tissue). The AC joint, formed by the union of the clavicle and the acromion process of the

scapula can be injured by overuse, typically from positions attained at the top of the backswing and the end of the follow-through. The authors also stated that posterior shoulder instability and subacromial (bursal) impingement have been seen in golfers, also usually at the top of the backswing. A study (Mallon & Colosimo, 1995) that looked at only AC joint injuries (in competitive golfers, handicap 3 or less) found that 18 of 34 golfers with left shoulder pain (53%) had pain isolated to the AC joint, and of them, 41% showed evidence of AC joint OA.

Another review article (Bayes & Wadsworth, 2009) once again stated that most shoulder pain in right handed golfers was seen in the left shoulder. This study explained that at the top of the backswing the lead shoulder could be subjected to AC joint compression, impingement of the humerus on the anterior part of the GH labrum; or a stretching of the posterior capsule of the GH joint. The same shoulder could, at the end of the follow-through, be prone to injury to the posterior GH labrum or an impingement of the posterior RC with the posterior labrum. Overall, the authors found that RC injuries are typically the most common injuries to golfers' shoulders and are generally chronic. According to them, while RC injury to the trail shoulder is usually well tolerated, pain in the lead shoulder at the top of the backswing interferes with movement.

One 2013 review (Cohn, Lee & Strauss, 2013) identified two basic types of impingement syndromes – external and internal. External impingement (subacromial impingement), refers to impingement outside the joint capsule, and occurs when the shoulder is elevated, which reduces the space between the humerus and the acromion process of the scapula. This position, is, for example, attained by the lead shoulder at the top of the backswing, and can lead to the formation of spurs (bone thickening) or inflammation of the subacromial bursa. The result can be either tendinitis or even partial tears of the RC muscle, and the authors mention that RC tendinitis and impingement together are the second most common cause of shoulder pain in elite golfers.

Internal impingement (within the GH joint itself), on the other hand, is typically seen in the lead shoulder at the top of the backswing and at the end of the follow-through.

The lead shoulder attains a position of external rotation and abduction at the end of the follow-through, which may cause labral tears, RC tears and lesions to the articular region of the humeral head (Bayes & Wadsworth, 2009). Finally, the lead shoulder can also develop instability in younger golfers, especially when they have preexisting hyperlaxity of the joint which is then combined with overuse. Posterior instability takes place in the lead shoulder at the transition between the top of the backswing and the downswing, while anterior instability is more commonly seen at the end of the follow-through. Shoulder instability can lead to several injury symptoms. Andrews and Whiteside (1994) provided greater detail about anterior and posterior shoulder instability. In anterior instability, the humeral head may be subjected to subluxation (incomplete dislocation) if there is any stretching of the anterior part of the joint capsule, which, with repetition, can cause anterior labral lesions (pathologic change in tissue), among other injury. Posterior instability tends to be caused by genetic, not sports related, predisposition. Pathologies develop more easily in unstable joints.

A retrospective review (Hovis, Dean, Mallon & Hawkins, 2002) on shoulder injury explained that lead shoulder elevation may be reached by a competitive golfer up to 2000 times per week. This position has been known to cause subacromial impingement, AC joint arthrosis and posterior GH instability. In their shoulder injury review, Kim, Millett, Warner and Jobe (2004) included further shoulder pathologies. For instance, golfers complaining of lead shoulder pain at the end of the backswing or the start of the downswing could have tears to the superior part of the labrum of the glenoid fossa. Such injuries are referred to as superior labrum anterior-to-posterior (SLAP) tears. Anterior shoulder pain during the later follow-through phase could be

indicative of biceps tendinitis, also known as inflammation of the tendon of the long head of the biceps brachii muscle. One study (Jacobson, Miller & Morag, 2005) differentiated between lead shoulder injuries seen in older and younger golfers. In older golfers osteophytes in the acromioclavicular region can cause impingement and RC pathology, while in younger golfers the more commonly seen injury is hyperlaxity of the GH joint, weakness or imbalance of the RC muscles or a tightness of the posterior joint capsule.

A recent epidemiological (Lee et al. 2017) shoulder injury study of 77 Korean amateur golfers with shoulder pain combined an injury questionnaire with an ultrasound assessment. Participants were divided into an RC tear group (because this is the most frequently seen shoulder injury among golfers) and a non-RC injury group. The former was further divided into supraspinatus muscle tear (the most frequently seen) and non-supraspinatus muscle tear (i.e. one with involvement of the other three RC muscles – subscapularis, infraspinatus and teres minor). Supraspinatus tears were the most frequently noted injury, with 5 in trail and 12 in the lead shoulders. Similarly, two trail shoulder and seven lead shoulder subscapularis tears were seen. Subacromial or subdeltoid bursitis and AC joint OA were observed in both shoulders, and biceps tenosynovitis (inflammation of a tendon and the sheath surrounding it) in the lead shoulder, all as findings accompanying the RC tears.

One article (Sutcliffe, Ly, Kirby, & Beall, 2008) which compared golf related injuries with the associated magnetic resonance images (MRI), summarized the differences between RC injury between older and younger golfers, stating that pathology typically occurs because of excessive rotation of the shoulder joint at the ends of the range of motion. While older golfers typically have more degenerative changes such as acromioclavicular osteophytes which

can impinge on the subacromial bursa and/or the supraspinatus tendon, younger players more typically have impingement of the supraspinatus tendon because of repetitive impingement-causing motions. The resulting injury can be subacromial bursitis, RC inflammation (tendinitis) or RC tearing. This paper mentioned that the two most frequently affected RC muscles are the supraspinatus and the infraspinatus.

In summary, lead shoulder impingement of the supraspinatus, infraspinatus and subscapularis RC muscle tendons, subacromial bursitis, and AC joint arthritis are the most commonly seen lead shoulder injuries, followed by labral damage and biceps tendinitis. The most frequently observed injury to the trail shoulder is RC (supraspinatus, subscapularis) damage.

Elbow. The main elbow joint is a hinge joint formed by the humerus of the upper arm and the ulna and radius bones of the forearm. The two prominences of the distal humerus just proximal to the elbow joint are the lateral and medial epicondyles, and many muscle tendons attach to these structures (Stanish, Loebenberg & Kozey, 1994). Most forearm muscle-tendons originate from the two epicondyles - the wrist flexor muscles from the medial epicondyle and the wrist extensors from the lateral epicondyle. The muscle forces generated within the relatively small attachment-areas of several muscle tendons create significant stress, which is why elbow tendinitis, usually referred to as elbow epicondylitis (inflammation of an epicondyle) is the most frequently seen elbow injury among golfers. Elbow tendinitis occurs so frequently in sports that lateral epicondylitis is often referred to as “tennis elbow” while medial epicondylitis has been termed “golfer’s elbow”. The term “lateral” refers to a position further from the midline of the body compared to “medial” which is closer to the midline, when the body is in an upright

standing posture and the palms are facing forward. The right handed golfer will typically suffer from left arm tennis elbow and/or right arm golfer's elbow.

Amateur golfers are known to injure their elbows more frequently than professionals and, among them, females have a greater incidence of elbow injury than males. In addition, in some studies more pain has been experienced on the lateral side of the elbow than the medial side (Stockard, 2001), while in others (Sutcliff et al., 2008), lateral epicondylitis of the lead elbow has been seen as frequently as medial epicondylitis of the trail elbow. Lateral elbow injury is more frequent during the fourth and fifth decades, and is said to be equally likely among males and females (Ellen & Smith, 1999).

Ulnar neuropathy/neuritis (disorder of a segment of a nerve) in the cubital tunnel region near the elbow, has been seen among golfers, as a co-morbid condition accompanying medial epicondylitis of the trail elbow in up to 20% cases. Ulnar collateral ligament injury has also been seen in golfers, typically at the medial side of the trail elbow (Stockard, 2001). Therefore, elbow injury in golfers mainly comprises lead elbow lateral epicondylitis, trail elbow medial epicondylitis, and trail elbow ulnar neuritis.

Wrist/hand. The wrist or radiocarpal joint comprises the radius of the forearm and some of the carpal bones of the wrist/hand. Strong flexor and extensor muscles with their origins in the area of the elbow, insert into various bones in the hand, to produce wrist flexion and extension as well as radial (abduction; away from the body's midline) and ulnar (adduction; towards the body's midline) deviations.

According to one review (Murray & Cooney, 1996), in golf the lead wrist and hand are more frequently injured than the trail wrist. The most common type of overuse injury seen is

tendinitis. Lead wrist tendinitis takes place in several areas. Extensor carpi ulnaris (ECU) tendinitis is associated with the start of the downswing. De Quervain's disease is a fibrosis (fibrous tissue formation) of the sheath of two tendons of thumb muscles (abductor pollicis longus and extensor pollicis brevis). It is caused by, or exacerbated at, the top of the backswing. Flexor carpi radialis (FCR) tendinitis and flexor carpi ulnaris (FCU) tendinitis have both been reported in golfers, typically in the trail hand. Additionally, the golfer's trail wrist is also susceptible to flexor tenosynovitis, which may manifest itself as carpal tunnel syndrome (median nerve entrapment in the palmar or anterior side of the wrist).

One epidemiological study of elite golfers (Hawkes, O'Connor & Campbell, 2013) also found that most wrist injuries were to the lead wrist. On the ulnar or medial side, ECU tendinosis (more appropriately, tendinitis) was seen in more cases than ECU subluxation. ECU tenosynovitis was also seen in the lead wrist. On the radial or lateral side of the wrist, the lead hand was diagnosed most frequently with de Quervain's tenosynovitis. The authors stated that the most serious wrist injuries involved damage to the retinaculum (a retaining band) of the ECU tendon of the lead wrist, and were typically acquired at impact.

Finally, a more recent review article (O'Connor et al., 2016) described three commonly seen wrist injuries in elite golfers. They are: lead wrist ECU tendon pathologies including overuse tendinopathy and tenosynovitis, sheath subluxation and dislocation on the ulnar (medial) side; and lead wrist De Quervain's tenosynovitis on the radial side. The third injury is a dorsal (posterior) side carpal impingement syndrome seen on the trail wrist of elite golfers. Rettig (1994) mentioned that there are several injuries possible at the wrists/hands which could include tendinitis, impingement, the formation of ganglia, sprains, stress fractures especially to the hook of the hamate carpal bone, distal radioulnar joint pathology, nerve compression issues (at the

carpal tunnel and Guyon canal of the hand) as well as vascular problems. In fact, Rettig (1994) stated that hand and wrist injuries had been found to be 37% of the total injuries in one studied population, with lead wrist and hand injuries being the most frequently seen.

Therefore, the most commonly seen wrist injuries of the lead wrist include ulnar side ECU tendinitis, and radial side de Quervain's disease/syndrome. On the trail wrist, FCR and FCU tendinitis and flexor tenosynovitis manifested as carpal tunnel syndrome may be seen, along with carpal impingement on the dorsal side.

Hip. Injuries to golfers' lower extremities are not as common as to the upper limbs. Hip injuries are seen far less frequently (in 1% professionals and 3.1% amateurs) than knee (in 6.6% professionals and 9.3% amateurs) and ankle (in 2% professionals and 2.5% amateurs) injuries. When they do occur, lower extremity injuries prevent proper downswing weight shift and rotation (McCarroll, 1994).

The hip joint connects the head of the femur (ball) with the acetabulum (socket) of the pelvic girdle. It is a stable ball and socket joint with movement possibilities in all three planes of motion (Moore, Dalley, & Agur, 2010). Dickenson et al. (2016) assessed the prevalence of hip pain in professional golfers and found the difference in laterality reported for the lead (11.9%) and trail (9.1%) hips to be insignificant, although lead hip quality of life scores were significantly lower. It may therefore be said that both hips may be subjected to certain types of tissue injury. According to McCarroll (1994), one frequently seen injury is trochanteric bursitis (bursa of the greater trochanter of the femur), which is more commonly seen in female golfers. Hip cartilage damage of either hip, in the form of OA, is not seen merely in older golfers, but in individuals as young as 35 years too. A recent review article (Lee & Lee, 2017) described the most common hip injuries as including labral tearing and femorocetabular impingement. This

latter injury is typically not related to the actual movements of the golf swing. Thus, typical hip overuse injuries include trochanteric bursitis, OA, and joint laxity (looseness) with the associated labral tearing.

Knee. The knee includes a joint between the femur of the thigh and the tibia of the leg/shank, as well as the articulation of the patella (knee cap) with the femur. Considered a modified hinge joint, it is mainly designed for a large range of flexion and extension but it also permits some internal and external rotation (McCarroll, 1994). McCarroll reported that knee injuries usually involve either tearing of the menisci (crescent shaped fibrocartilaginous structures on the superior part of the tibial bone) or pathology at the patellofemoral (PF) joint.

Meniscal tearing usually involves the posterior horn of the medial meniscus, and such injuries occur most commonly among older golfers. Although the author did not mention which knee is typically affected, it is presumed that he referred to the lead knee, as that is the knee which undergoes repetitive twisting and bending (at high speed), which are known causative factors for meniscal injury. PF pain can result from a variety of overuse syndromes, but may also be a result of morphological factors, a topic which is beyond the scope of this paper.

One surgeon (Guten, 1996) assessed 35 golfing knee injuries seen at a private orthopedic practice, and discovered that 34 of them were from overuse. He found that there was no correlation between a player's age or handicap and golf-related injuries. All golfers in his study (28 males and 7 females) were right handed and between the ages of 21 and 73. Of them, 17 had trail knee problems, 15 had lead knee issues and three had pathology in both knees. This was in agreement with an earlier study (Stover & Mallon, 1992) which had found a 10% incidence of trail knee pain compared to 7% lead knee pain. Tissue-wise, the damage consisted of, in decreasing order of incidence, medial meniscus tears, OA, lateral meniscus tears,

chondromalacia of the patella (softening of patellar cartilage) and loose bodies (small, free-floating fragments of cartilage or bone) within the knee joint. Guten did not report the laterality of tissue-specific injuries.

A review article by Marshall & McNair (2013), stated that the most commonly seen knee injuries are to the anterior cruciate ligament (ACL), the lateral or fibular collateral ligament (LCL) and the medial or tibial collateral ligament (MCL). These authors also stated that the material structure of the medial menisci changes with age, suggesting that degenerative tears can occur after middle age. A recent systematic review (Baker et al., 2017), found that most previous studies do not report the structures involved in knee injury, and relied on several case studies to acquire more information. The Baker et al. study's literature search revealed that medial meniscus injuries had been reported most frequently, followed by degenerative changes to the knees from OA. The authors stated that professional golfers had a greater incidence of overuse knee injury than amateurs.

The lead knee is more susceptible to injury based on the mechanisms of loading it endures (Baker et al., 2017), which appears to contradict the information from earlier studies regarding the laterality of greater injury (Guten, 1996; Stover & Mallon, 1992). It may therefore be stated that the most frequently seen knee injuries are tears of the medial and lateral menisci, OA, and ACL, MCL and LCL ligamental damage, with both lead and trail knee tissue being prone to damage.

Ankle/Foot. Ankle sprain is the most common injury seen at the ankle and can take place during the actual swing from "abnormal weight shift" (Lee & Lee, 2017), a concept which has not been adequately defined. Sprains moreover, are typically acute, and are often the result of accidents when players trip over an obstacle, or slip, especially when carrying a golf bag (Batt,

1992; Gosheger et al., 2003). The other frequently seen ankle injuries such as peroneal (fibular) tendinitis, Achilles tendinitis and plantar fasciitis, are perhaps more related to walking on the golf course than to the swing. For the above reasons, ankle injuries will not be discussed further in this study.

Ribs. The main injury to the ribs involves stress fractures (fatigue fractures), and one study claimed that many cases may go unnoticed because of the usually rapid “spontaneous healing” (Kuroda & Ichikawa, 1977, p. 182). Rib fractures are predominantly seen in beginner golfers and usually take place in the lead upper posterior ribs, typically from ribs four to seven (Rasad, 1974). One study of 11 beginner golfers (Lin, Chou & Hsu, 1994) found that all lesions were to the posterolateral sides - eight on the lead side and six on the trail side - while three golfers had bilateral injury. Those with trail side stress fractures had a history of divot taking with their swings, and the injured players hit approximately 400 balls per week.

A study (Kuroda & Ichikawa, 1977) of 13 right-handed Japanese golfers of ages ranging from 24-42 years, found that there were stress fractures on 12 lead-side ribs and one trail-side rib. Injuries were seen on all ribs from the first to the 12th, and the golfers were within three days to 24 months of commencing golf, or were recommencing it after a considerable break. The study also reported 15 other Japanese case studies with golfers ranging in age from 33-53 years. They all had lead sided rib fractures, with the onset of the injury ranging from a few days to 12 months from initial commencement of golf. Across all the case studies and the 34 golfers reported, only two were females. Finally, a review (Lee, 2009) of all stress fractures related to the golf swing found that rib fractures were most frequently seen on lead side ribs, specifically on the fourth to sixth ribs. They occurred almost exclusively when the players were new to golf and participated in high quantities as well as high frequencies of practice or other training. Thus,

posterolateral stress fractures of the upper lead-side ribs comprise the most frequently seen injury to the ribs.

Part 2. General Etiology

All injury to human tissue can be categorized as having risk factors which are genetic, morphological, psychological or biomechanical (Kumar, 2001). The focus of this section is to report the expected causative factors of injury to musculoskeletal tissues which are biomechanical in origin. Such tissues include ligaments, tendons, muscles, cartilage, bones, and even nerves. All of these tissue types can be damaged through movements during which excessive force is applied, which then creates compressive, tensile (stretching), shear, or torsional loads, on a recurring basis (Kumar, 2001). This section collates information on all the motions and loads which have been ascribed to overuse injuries. Commonly seen pathogenesis or mechanisms of each injury, whether observed in golf or other similar sports, are described, to create an understanding of which movements and loads are typically correlated with specific tissue damage.

Spine – lumbar and thoracic. The lumbar spine, based on the design of its discs and facet joints, allows significant forward flexion and extension, some lateral flexion (side-bending) and limited horizontal plane axial rotation (Gluck, Bendo, & Spivak, 2008). In the context of those specific capabilities and limitations, the four directionally different forces imposed on the lumbar region of the spine during the golf swing are: shear forces directed anteroposteriorly (front-to-back), shear forces directed mediolaterally (side-to-side), vertical compressive forces (caused by back muscles that pull downwards to stabilize the body from falling forwards), and torsional forces around the vertical axis of the body (Hosea, Gatt & Gertner, 1994). As a point of

distinction, “axial loading” involves compression along the vertical axis of the body, while “axial rotation” refers to rotation about the vertical axis (spinal column) of the body.

One early study (Sugaya et al., 1998) combined epidemiological findings with those of radiographs, to understand not only the prevalence of LBP, but also to discover the location of injury, the swing phase during which symptoms were reproduced, and the actual tissues injured during the golf swing. The authors concluded that the main contributing factor for lumbar degeneration and other injury was a combination of the lateral bending angle and rotational velocity of the torso, which they termed the “crunch factor”. The crunch factor increases during the downswing, and reaches peak values just after impact.

A subsequent study (Cole & Grimshaw, 2014) that further analyzed the crunch factor (related to lateral bending angle and trunk rotational velocity), compared golfers with and without LBP. Both groups showed increasing crunch factor from mid-downswing to impact and slightly beyond. The authors concluded, based on a comparison with a study of cricket fast bowlers, that lateral bending velocity combined with rotational velocity might be more predictive of injury and also opined that the iron clubs would cause more injury than the wood clubs because of the greater lateral bend and lateral bending velocity involved. Finally, a study comparing LBP in elite female cricket fast bowlers with and without LBP, concluded that it was lateral bending rather than shoulder counterrotation (angle between shoulders and pelvis, similar to that seen in golf’s X-Factor angle) which would be more likely to cause pain (Stuelcken, Ferdinands, & Sinclair, 2010). The X-Factor in golf involves a maximization of the hip-shoulder separation angle, tends to increase torque on the lumbar spine, and has been implicated in LBP in golfers (Gluck et al., 2008).

Lumbar disc trail side. Disc herniation has been ascribed to a combination of compression, torsion and lateral bending (Lee & Lee, 2017). As little information exists on the mechanisms of injury for the individual tissues injured, a report from other sports or from cadaveric studies is often useful. In a very early cadaveric study (Roaf, 1960), the author stated that the healthy disc, as well as vertebral joints and ligaments are resistant to compression and stretching created by spinal flexion and extension, but vulnerable to rotation and horizontal (side-to-side) shear. The lumbar disc, specifically, is fairly resistant to a compressive force, and prolapse (sinking down) takes place only if its inner nucleus pulposus has lost its typical turgor, such as occurs with aging. Conversely, according to the author, a combination of rotation and compression can cause most spinal injuries.

One review paper (Gluck et al., 2008) also stated that disc herniation in a healthy disc typically takes place as a result of lateral bending combined with compression and torsion, all of which, the authors remarked, may be seen during the golf swing. Finally, Lindsay and Vandervoort (2014), mentioned that compression loads (higher in professionals than in amateurs) may also cause disc herniation, because such loads, seen across all skill levels of golfers, are greater than those found in a cadaveric study to cause disc prolapse. The authors also suggested that the side bend seen in the golf swing causes mediolateral shear, which may be harmful to the discs, as it is mainly those structures, not vertebral bone, that resist sideward-directed forces. Thus downswing lateral flexion with axial rotation, the velocities of both movements, and compressive forces, are the main factors implicated in lumbar disc herniation.

Lumbar facet joint trail side. As the lumbar spine is not designed for rotation, (Gluck et al., 2008) specifically because of the sagittal (front-to-back) orientation of its facet joints, even a 2° to 3° intersegmental rotation (as seen in any swing using limited pelvis versus large torso

rotation, termed the X-Factor in golf) can produce facet joint microtrauma. Facet joint pain can be similar to pain from spondylolysis, according to one review article (Reed & Wadsworth, 2010), and is caused mainly by hyperextension, although compression and rotation can be precipitating factors too.

Lumbar spondylolysis. Based on his cadaveric study, Roaf, (1960), believed that the so-called hyperextension (as seen in the “reverse-C” finish of the modern golf swing) and hyperflexion injuries are really rotation injuries. However, according to a prospective study of injury incidence among pole vaulters of both genders, a spondylolytic fracture results from repetitive hyperextension of the lumbar spine (Rebella, 2015). A golf review article (Reed & Wadsworth, 2010) summarized the information by stating that spondylolysis, which typically occurs in younger golfers, is caused by repeated lumbar hyperextension, and exacerbated with rotation.

Thoracic compression fractures. These fractures, especially in postmenopausal, osteoporotic females are caused by, as the name suggests, vertebral compression. As stated by Roaf (1960) in his cadaveric study, vertical (compression) pressure is more likely to cause a fracture of the cancellous bone of the vertebral body than the herniation of a healthy disc.

Spine – cervical. The cervical (neck) region of the spine is an important area to study with respect to golf injury because, as reported in one review on cervical spine injury in athletes, the risk for non-contact, high-velocity sports’ injury in the neck region is as great, if not greater than, for contact sports (Morganti et al., 2001). The morphological (structural) makeup of that section of the spine permits almost equal amounts (approximately 90°) of flexion and extension as well as rotation to either side. Lateral flexion, however, is limited to between 20° and 45° (Swartz, Floyd & Cendoma, 2005). Could the difference in movement capabilities of the different spinal

sections help to explain why most injury to the lumbar spine happens on the trail side, yet cervical injuries are more commonly seen on the lead side?

In one epidemiological study, the authors commented anecdotally that during the takeaway (until the end of the backswing) golfers “rotate the shoulders, hips, knees, and lumbar and cervical spines while the head remains stationary” (McCarroll & Gioe, 1982, p. 66). They further added that after hitting the ball the lumbar and cervical sections of the spine rotate and hyperextend. The questionnaire of their study of professional male and female golfers elicited information regarding the phase of the swing related to cervical injury and found that the neck could be injured during the takeaway phase (up to the top) or the follow-through phase. From their findings it may be said that the unique position of the head compared to the torso during the backswing, and the rotation and hyperextension of the neck during the follow-through, are important causative factors for cervical injury. The difference between cervical spine movement compared to the rest of the spine, will be of interest in determining other likely mechanisms of neck injury in golfers.

Lead side disc. Cervical disc injury has an opposite laterality to that of thoracic and lumbar injury. A review article on injuries to the cervical region of the spine in athletes (Chang & Bosco, 2006) stated that acute cervical intervertebral disc disruptions, termed “soft disc disease”, could be ascribed to the lateral bending of the neck. Another review article on cervical injury among athletes (Maroon & Bailes, 1996) stated that in the sport of wrestling, albeit a contact sport unlike golf, axial compression, axial rotational, and medio-lateral or horizontal shear forces can all be causative factors for both intervertebral disc and facet joint injuries.

Lead side facet joint. A common causative factor of cervical facet joint injury is

“whiplash” (a flexion-extension injury), although this phenomenon has not been used to explain golf-related facet-joint injury. Whiplash can result in joint capsule strain from a combination of shear and compression, as seen even in low-speed rear-side automobile collisions (Lu, Chen, Kallakuri, Patwardhan, & Cavanaugh, 2005). Moreover, whiplash can affect several tissues according to Pearson, Ivancic, Ito and Panjabi (2004), with compression affecting the facet joint itself, and strain (from elongation) damaging the capsular ligaments (CL), as individual vertebrae slide anteroposteriorly relative to one another. Whiplash has two phases – the first occurs when cervical vertebrae go from a neutral position into extension, at the peak of which compression is maximal. During the second phase, they flex to return to neutral, and maximum CL strain is known to take place in the middle of the second or forward neck movement phase. A prospective cohort study of injuries in international rowers reported a surprisingly large number of cervical injuries, most of them to the facet joints. These injuries were all sculling (two oars per rower) not sweep (one oar per rower) injuries, and were ascribed to the need for this type of rower to be frequently “looking behind”, which involves rotation at the neck (Wilson, Gissane, Gormley, & Simms, 2010). Cervical injury, whether disc or facet, is said to result from compression combined with rotation, flexion/extension and medio-lateral shear.

Shoulder. The lead shoulder is the one more frequently injured one, as it achieves a position of considerable horizontal adduction and internal rotation at the top of the backswing, and external rotation and abduction at the end of the follow-through. Many pathologies can result during both of those positions which are at the extremes of the GH joint’s range of motion (Bayes & Wadsworth 2009; Kim et al., 2004). In fact, one study (Hovis, Dean, Mallon & Hawkins, 2002) indicated that lead shoulder elevation (associated with horizontal adduction) can reach above 120°, that is, 30° above the plane of the shoulders. At the end of the backswing, a

golfer could experience either anterior or posterior shoulder pain. Anterior shoulder pain indicates impingement of the head of the humerus and the anterior glenoid labrum, or AC joint impingement and degeneration, while posterior pain may be indicative of posterior capsulitis or tightness of the RC muscles. If pain is felt on the lead shoulder at the end of the follow-through, it might indicate impingement of the head of the humerus with the posterior rim of the glenoid fossa, the posterior labrum, or the inferior surface of the RC muscles (Cohn, Lee & Strauss, 2013; McHardy, et al., 2006). These positions can lead to RC injury and/or labral tears.

RC tendon injuries are the most frequently seen shoulder injuries, and are referred to by several names such as tendinitis and tendinosis, all of which fall under the umbrella term of tendionopathy (Seitz, McClure, Finucane, Boardman, & Michener, 2011). The RC's mechanism of injury is said to be either extrinsic (to the joint capsule) or intrinsic, as described briefly in Part 1. Extrinsic injury causes compression of the tendons, and involves two types of impingement. Impingement has classically been thought to take place when the subacromial space narrows during elevation of the arm and the humerus moves closer to the acromion process of the scapula, resulting in compression of any tissues between these two structures. Such structures include the RC tendons which insert onto the humerus, and the subacromial bursa. Besides "subacromial impingement", another more recently recognized extrinsic impingement is "internal impingement" in which an RC cuff tendon directly impinges on the posterior superior aspect of the glenoid rim, when the arm is in full external rotation, abduction and extension. Both the extrinsic causative factors involve overhead shoulder movements, similar to those that take place in the trail arm at the top of the backswing or the lead arm at the end of the follow-through. The third mechanism of RC injury is an intrinsic, degenerative, overuse mechanism, which

occurs under tensile loads, such as when lead arm's RCs are stretched during the backswing, or the trail arm's are stretched at the end of the follow-through (Seitz et al., 2011).

Lead rotator cuff tendon impingement/damage. The four RC muscles together generate most of the compressive force as well as force that resists shear around the GH joint (Meister & Andrews, 1993). RC damage or tearing, especially of the tendon of the supraspinatus muscle (located most superiorly on the humerus), occurs when the muscle moves below the acromion, and suffers impingement. This subacromial region of the supraspinatus has poor vascularity (blood supply) and impingement can lead to inflammation followed by tendinitis or tearing of the muscle (Seminati & Minetti, 2013). Continual impingement resulting from overuse constitutes the main causative factor of cuff pathology, and a supraspinatus tear can lead to the tearing of other RC muscles such as the infraspinatus or the subscapularis (Lee et al., 2017). The authors ascribed full thickness lead shoulder supraspinatus tears to impingement during the follow-through phase of high velocity swings. Meanwhile, the subscapularis, involved with shoulder internal rotation, can suffer damage because it is the most active of the RC muscles throughout the swing, and especially when it acts eccentrically during the braking action of the downswing (Lee et al., 2017). Lead side RC injury can therefore take place both at the top of the backswing when the shoulder is maximally adducted, internally rotated and elevated, and at the end of the follow-through when the shoulder is maximally externally rotated, abducted and extended.

Lead labral damage. Shoulder instability, typically seen in younger golfers, is a result of existing joint laxity along with overuse (Cohn et al., 2013). Posterior GH instability is seen in the lead shoulder at the top of the backswing (Hovis et al., 2002), and anterior instability at the end of the follow-through. According to Andrews and Whiteside (1994), while posterior subluxation

(partial dislocation) is more typically a result of genetic, and not sports-related, joint laxity, stretching in the anterior part of the lead shoulder can produce lesions in the region of the anterior labrum. Such stretching would occur when the shoulder is extended, abducted and in external rotation (Andrews & Whiteside, 1994), a situation seen with the lead shoulder towards the end of the follow-through.

Lead subacromial bursitis. Lee et al. (2017) studying the causative factors of shoulder injury, discovered that the cases of subacromial (and subdeltoid) bursitis were concomitant with supraspinatus muscle tears, leading the authors to opine that such bursitis might also be the result of overuse, secondary to impingement, and probably resulting from positions similar to those seen in RC injury.

Lead AC joint arthritis. Force across the AC joint is known to be maximal when the shoulder is abducted or adducted (Mallon & Colosimo, 1995). The AC joint of the lead shoulder gets compressed at the end of the backswing, during a movement often referred to as “cross body adduction” (Bayes & Wadsworth 2009; Mallon & Colosimo, 1995). This may be a cause of the OA/arthritis seen in this joint, as also described by Hovis et al. (2002).

Lead biceps tendinitis. Lee et al. (2017) indicated that biceps tendinitis is caused by excessive flexion of the lead elbow during the follow-through phase. Kim et al., (2004) indicated that biceps tendon disorders (and superior labrum lesions) do occur during the golf swing, even though it does not have a truly overhead motion. Additionally, according to the authors, in biceps tendinitis, pain is felt on the anterior shoulder, especially during the late follow-through when the lead arm is maximally abducted and externally rotated as well as extended. It may be deduced that excessive elbow flexion combined with shoulder abduction, external rotation and extension can cause damage to the lead biceps tendon during the latter part of the follow-through.

Trail rotator cuff tendon impingement/damage. The only injuries reported at the trail shoulder involve the supraspinatus and subscapularis muscles (Lee et al. 2017), as this shoulder is placed in external rotation during the backswing and in horizontal adduction at the end of the follow-through (McHardy et al., 2006), although the injuries and positions described by McHardy have not been linked in any study. However, if the trail shoulder's position at the top of the backswing is compared to the lead shoulder's at the end of the follow-through, which has been described to be compromised in maximal external rotation, abduction and extension, then this position may cause trail shoulder injury. Similarly, if the trail shoulder's position at the end of the follow-through is compared to that of the lead shoulder at the top of the backswing, this may be a causative factor for RC injury too.

Elbow. Many long wrist flexor and extensor muscles originate as narrow tendons at the elbow's epicondyles. These tendons lie between strong muscles and rigid bone, making them vulnerable to injury when they are subjected to tensile (stretching) forces (Stanish, Loebenberg, & Kozey, 1994). That is why the most common overuse pathologies seen at the elbow are lateral and medial epicondylitis of the lead and trail elbows respectively.

Lead elbow lateral epicondylitis. One review article (Hume, Reid, & Edwards, 2006) on epicondylar injury in all sports stated that when the wrist extensor muscles are used excessively and overstretched as well as torqued, lateral epicondylitis can result. Another review (Grimshaw, Giles, Tong, & Grimmer, 2002) summarized the mechanism of lateral epicondylitis as including repetitive elbow extension in combination with a twisting motion; frequently with an excessively forceful grip. This tight grip is said to increase tensile loads on the wrist extensors (Wadsworth, 2007). Wadsworth (2007) also opined that an excessively straight lead arm might have tension in it which could be a further cause for lateral epicondylitis. In summary, in the

words of Stockard (2001), lateral epicondylitis is caused by “repetitive forceful extension of the forearm accompanied by a twisting motion, especially if associated with excessive gripping of the golf club” (p. 509).

Trail elbow medial epicondylitis. In an EMG study that compared professional and amateur golfers, the pronator teres (PT) muscle originating at the medial epicondyle, generated considerably more activity during the forward swing (earlier part) and acceleration phases of the golf downswing in amateur golfers. The authors correlated this fact with having seen trail arm medial epicondylitis more frequently than in the lead arm in their clinical experience (Farber, Smith, Kvitne, Mohr, & Shin, 2009), and thus concluded that the PT muscle might be the main contributor to medial epicondylitis in amateur golfers. They surmised that amateur golfers “push” the trail arm through the swing’s arc, rather than “pull” it, which latter action was what the greater lead arm PT muscle activity of the professionals seemed to indicate.

Another EMG-based study (Glazebrook, Curwin, Islam, Kozey, & Stanish, 1994) compared low and high handicap golfers with and without medial epicondylitis, and found that there was significantly greater trail wrist flexor EMG activity during the address and backswing phases only, in all the symptomatic participants compared to the non-symptomatic ones. This was contrary to the results of an earlier study, the authors stated, in which pre-impact and impact were found to be the swing phases which maximally stressed the common flexor tendon at the medial epicondyle, while the wrist was in a valgus position (hand further from the body’s midline than the forearm), often termed a “cocked” position in golf. Therefore the authors concluded that the excessive address and backswing activity of the wrist flexors combined with the burst of flexor activity, from an extended position, that takes place close to impact, might be a causative factor for medial epicondylitis.

One typically implicated mechanism of injury in medial epicondylitis of the trail elbow besides repetitive muscular contractions, is sudden deceleration when the club encounters the ground or thick grass (Sutcliffe, Ly, Kirby & Beall, 2008). To summarize, risk factors for trail-side medial epicondylitis are forearm pronation and wrist flexion during the downswing (Hume et al., 2006), more so during deceleration when the wrist goes rapidly from an extended position to a flexed one.

Trail elbow ulnar neuritis. The ulnar nerve, as it passes superficially (close to the surface) through a narrow groove at the elbow termed the cubital tunnel, can be exposed to tension (stretching) when the elbow is repetitively flexed, causing ulnar neuritis (Ellen & Smith, 1999). Additionally, a combination of shoulder abduction, forearm pronation, wrist extension and radial deviation, along with elbow flexion, as seen during the end of the golf backswing and early downswing, increases tension on the ulnar nerve considerably (Byl, Puttlitz, Byl, Lotz, & Topp, 2002; Wright, Glowczewskie, Cowin, & Wheeler, 2001).

Wrist/hand. Tendinitis is the most common wrist problem seen in golfers. It is said to be the result of ulnar and radial deviation in the lead wrist; flexion and extension in the trail wrist; and pronation and supination of the forearms (Rettig, 1004). Both tension and compression can cause tendon injuries.

Lead wrist ulnar side ECU tendinitis. Rettig (1994), while discussing tendinitis, explained that although the range of ulnar deviation of the lead wrist at impact is small, it is the speed of the movement which places it under considerable stress. Rapid ulnar deviation, usually combined with forceful forearm supination and wrist flexion, can often result in damage to the ECU tendon on the ulnar side of the wrist. Injury could range from tenosynovitis

to subluxation to a frank dislocation (Chauhan, Jacobs, Andoga, & Baratz, 2014). One epidemiological assessment of wrist injuries in professional European golfers found that the most serious wrist injury seen in their study was to the ECU tendon, often caused by sudden deceleration as when striking the ground, when extrinsic wrist muscles must contract to maintain a stable wrist as the decelerating club tries to force the wrist into radial deviation (Hawkes et al., 2013). ECU tendinitis, therefore, results from wrist ulnar deviation combined with forearm supination and wrist flexion, when movement takes place at considerable speed and precedes rapid deceleration.

Lead wrist radial side de Quervain's syndrome. De Quervain's syndrome or disease is a type of tenosynovitis, as it affects a tendon sheath. It is typically believed to result from excessive pronation and supination of the forearm (Sutcliffe, Ly, Kirby, & Beall, 2008). An article dedicated to golf-related wrist injuries (Murray & Cooney, 1996) stated that de Quervain's syndrome could occur during ulnar deviation of the lead wrist close to impact. The authors believe it could also take place in the same wrist during the top of the backswing or during downswing initiation, when the abducted thumb is subjected to resistance from the golf club. The tendons, according to the authors, could be further stressed if the golfer uses a "casting" type movement from the top of the backswing which causes an early "hand release" or abrupt wrist ulnar deviation, with the lead thumb trapped in a fixed position between the trail hand and the golf club. Loads leading to De Quervain's tenosynovitis could be imposed on the lead thumb, at either the top of the backswing or during early downswing, when it is abducted or ulnarly deviated, and the injury is perhaps exacerbated by forearm rotation.

Trail wrist FCR tendinitis. With an FCR tendinitis, there is pain on the volar (palmar or anterior) radial part of the wrist, and the causative factor is said to be repetitive palmar flexion against resistance (Murray & Cooney, 1996).

Trail wrist FCU tendinitis. Injury to the FCU tendon typically results from the microtrauma of repeatedly hitting the ground while taking divots, because this increase in resistance to movement loads the FCU muscle's tendon (McHardy et al., 2006). Repeated ulnar deviation, such as that seen in the act of hammering, has also been suggested as a mechanism of this type of injury in golf (Yamabe, Nakamura, Pham, & Yoshioka, 2012). Another likely causative factor for FCU tendon injury is the range of flexion and extension that the trail hand makes during the swing (Murray & Cooney, 1996). In summary, trail FCU injury risk factors include ulnar deviation accompanied by a large range of flexion and extension.

Trail wrist carpal tunnel syndrome. When moving the wrists (while grasping with the fingers) is a repetitive action, flexor tenosynovitis followed by carpal tunnel syndrome can result. Carpal tunnel syndrome involves a compression of the median nerve in the hand, and this can take place from the large range of flexion and extension movement during the golf swing, as well as from a tight grip (Murray & Cooney, 1996).

Trail wrist dorsal impingement. Dorsal impingement of the wrist (O'Connor et al., 2016) is said to be one type of carpal impingement syndrome which affects elite golfers and typically takes place when there is forced dorsiflexion or repetitive hyperextension of the trail wrist, especially when combined with axial loading (along the long-axis of the arm).

Hip. In general, it could be said that hip injury in the athlete is more likely from torsional or twisting movements, while uni-planar movements such as running straight are not as problematic (Byrd, 2007).

Trochanteric bursitis. Bursitis or inflammation of the bursa of the greater trochanter (a bony prominence) of the femur has been seen in female athletes with wide pelves or a more prominent trochanter, and in runners who considerably adduct the thigh towards the body's midline (Anderson, Strickland & Warren, 2001). Running on banked surfaces makes the level of the pelvis uneven, and that too can be a risk factor for trochanteric bursitis. Pain in the greater trochanter is also caused when the hip is extended. A case report (Karpinski & Piggott, 1985) of 15 persons studied specifically for greater trochanteric pain syndrome (which the authors said was often incorrectly diagnosed as trochanteric bursitis) indicated that patients' pain was reproduced during any of resisted abduction of the thigh, forward spinal flexion, or spinal flexion to the contralateral side. All these movements stretch the insertions of the gluteus medius and gluteus minimus muscles and can cause pain. Many of the positions and movements mentioned in the above articles (Anderson et al., 2001; Karpinski & Piggott, 1985) such as hip extension, leg abduction, spinal forward flexion and opposite-side lateral bending, are seen with respect to the lead hip during the downswing, while its adduction is seen during the backswing. Pain in the hip region may thus be a result of overuse, and while swinging from uneven terrain.

Osteoarthritis. OA involves the degeneration of joint cartilage, and takes place when forces are transmitted to cartilage because muscles and tendons can no longer support repetitive activity. One case study of treatment for a right-handed golfer with lead side hip OA, stated that OA can lead to a loss of medial and lateral rotation as well as abduction and extension of this joint (D'Amico, Betlach, Senkarik, Smith & Voight, 2007).

OA risk factors include, among others, gender (more males before age 50, and more females after), and greater body weight (Felson & Zhang, 1998). Hip OA has been seen in elite sportspersons including golfers (Lequesne, Dang, & Lane, 1997). When golf is practiced at a

medium intensity level, it can have a relative risk of 1.5 (taking into account several factors including physical loads on the hip joint). This is almost the same as long distance running (relative risk 1.7) and greater than soccer (relative risk 1). OA may thus be said to be caused by, in the case of sports, contact stress (Ganz, Leunig, Leunig-Ganz, & Harris, 2008) or compression. Additionally, joint cartilage may be more compressed when loaded in a “close-packed” position in which the head of the femur and the acetabular fossa within which it resides fit together with maximum congruence (Flynn, 1973). The hip’s close-packed position occurs during full extension, and slight medial (internal) rotation, a position the lead hip attains during the downswing. Moreover, greater external loads will be applied to the lead lower limb as pressure shifts onto it during the downswing, which makes the lead hip more likely to sustain OA damage. To summarize, OA may be more likely in the lead hip, during extension with internal rotation, especially during swing phases when that hip is subjected to greater external load.

Labral tears. Intra-articular (within the joint) injuries are receiving greater recognition with the advent of arthroscopic techniques, and are an important cause of pain and diminished performance in athletes (Phillipon & Schenker, 2005). The primary author of the paper, a surgeon, examined 16 professional golfers, all of whom had labral tears along with capsular laxity and rotational instability. The authors stated that in many sports movements, including the golf swing, the hip joint is forced into rotation, especially external, with axial (downward) loading. This is the movement that can cause elongation of the joint capsule, localized instability of the joint, and eventually, labral tearing. Therefore it may be said that

external rotation and axial loading, which are seen in the trail hip during the golf downswing and follow-through, may be causative mechanisms for joint instability and perhaps labral tearing in that joint.

Knee. One general, non-medical description of knee positions that are risk factors for injury during the golf swing was made by McCarroll (1994), who said that during the backswing the lead hip is rotated, the lead foot rolls inwards, and the lead knee also moves inwards, until it points behind the ball. This backswing position may be described as a pronated position of the foot and a valgus position of the knee (in which the tibia is further from the body's midline than the femur). It may place stress on some medial-side knee tissues, such as the MCL. During transition and the downswing, when the lead leg and knee move towards target and pressure is shifted onto that side, stress is placed on some lateral-side knee tissues such as the LCL. The trail knee may also have stress placed on some of its medial-side tissues such as its MCL, as it moves towards the lead knee during the downswing (McCarroll, 1994).

Early golf-swing knee research (Gatt, Pavol, Parker, & Grabiner, 1998) looked at resultant joint forces and moments, which were calculated from motion and force plate data to indicate the actions of body tissue such as muscles and ligaments on the knee joint. Resultant joint forces are linear and reflect a combination of bone-on-bone reaction force as well as muscle pull. Resultant joint moments are rotary forces generated by muscles, in order to move the segments they connect, about their joints. Ligaments tend to be strained (stretched) in order to resist motion in a particular direction. For instance, a ligament such as the anterior cruciate ligament (ACL) which is designed to resist tibial motion in an anterior direction or in internal rotation will produce posterior force and an external rotation moment to resist excessive anterior and internal rotation movements. While ligaments stretch to prevent excessive movement,

muscles contract to create movement. For instance, the quadriceps femoris, a group of anterior thigh muscles which act to extend the knee, will apply an extensor moment on the knee (Gatt et al., 1998). It is important to note that, during the golf swing the lead knee (tibia) is said to be rotating externally during the backswing and internally during the downswing, while the opposite description applies for the trail knee.

The rotational loads (around a vertical axis and around a frontal plane axis), as well as the anterior-posterior translational loads placed on the knee joint during the golf swing are resisted by the ligaments and the menisci of the knee (Marshall & McNair, 2013). During the downswing, the lead knee is mainly subjected to internal rotation forces as well as posterior and varus (tibia moved towards the body's midline) forces, while the trail knee is subjected to external rotation forces along with anterior and valgus (tibia moved away from the body's midline) forces (Lindsay et al., 2000). In general, knee tissues that resist joint compression and internal tibial rotation are vulnerable.

The lead knee is more vulnerable to damage during the golf swing, according to many studies including that of Baker et al. (2017). The main injury causing movements and loads include rapid knee extension when the knee is flexed between zero and 30° (angle between vertical femur and bent tibia); internal tibial rotation, strong compression of the tibiofemoral (TF) joint because of quadriceps femoris (anterior thigh) muscle activity, and large external ground reaction forces (GRF) acting on the knee (Baker et al., 2017). One study of 308 golfers (Chu et al., 2010) with a mean age of 43.2 ± 15.6 years and a mean USGA handicap of 8.4 ± 8.4 , showed that lead foot vertical GRF (VGRF) as a percent of body weight, changed from 29.0 ± 12.1 at the top of the backswing to 93.9 ± 28.5 and 95.1 ± 30.5 during the acceleration and last

40 ms phases of the downswing respectively. As a comparison, the trail foot experienced its maximum VGRF of 64.5 ± 14.3 at the top of the backswing.

Medial meniscus injury. According to the Marshall and McNair (2013) review, the menisci are most susceptible to injury during knee rotation, especially during weightbearing. This, the authors stated, is seen in the golf downswing when the lead knee has greater medial compartment loading along with horizontal rotation. The authors also stated that degenerative tearing of the menisci can occur from middle-age onwards, and that meniscal damage can progress to OA. A similar statement was made by McCarroll (1994), who opined that meniscal injury typically results from twisting or bending, and the older golfer is particularly susceptible. Posterior horn flap tears are seen frequently in this population. Such meniscal tears are a result of many years of friction as well as twisting, both of which occur during the golf swing. It may therefore be stated that greater loads are placed on the medial compartment of the knee when it is adducted and twisted (as seen in the trail knee in the backswing or the lead knee in the follow-through), making it vulnerable to medial meniscal injury.

Lateral meniscus injury. The only reference to lateral meniscal tearing (McCarroll, 1994), states that pain, once again, results from bending or twisting. It is very rarely seen in the older age group of golfers.

Osteoarthritis. OA at the knee can be either at the patellofemoral joint (PF) or at the TF joint. Increased PF joint forces results from excessive knee flexion (Roos, Barton, & van Deursen, 2012), and women are particularly vulnerable to PF injury, particularly if they have larger Q angles resulting from their wider pelves, as well as foot pronation (McCarroll, 1994). This female-specific morphology, combined with the typical “dynamic valgus” position (a combination of knee flexion, hip internal rotation and foot pronation) of both the knees as seen

during the golf swing, make female golfers more likely to suffer from PF pain or damage (McCarroll, 1994).

The main (TF) knee joint can be subjected to compressive forces which, with long term, repeated loading, can damage the cartilaginous patellar (anterior) surface of either femur. A study assessing exclusively frontal plane moments on the lead knee (Lynn and Noffal, 2010), found that an external adduction or varus moment, typically seen immediately after impact, increases the loads borne by the medial compartment of the knee, which could cause or exacerbate medial knee OA. The authors also hypothesized that the large abduction or valgus loads that the lead knee is subjected to just prior to impact would be borne by the smaller lateral compartment of the knee, which could be a risk factor for lateral compartment OA. Although no study has discussed the possible causes of trail knee injury, the same theories could be true, especially for lateral compartment loads on the trail knee, when the knee is in an increasingly valgus position, such as during early downswing. Finally, the recent review (Baker et al., 2017) stated that excessive rotation can cause contact between bony aspects of the femur and the tibia, in places where the cartilage is not as strong, which can also lead to damage. Based on the loads known to cause TF OA, the lead knee is perhaps more vulnerable, especially when it sustains large varus or valgus loads or when it is excessively rotated.

ACL injury. The ACL is designed primarily to restrain the anterior translation of the tibia, and is stretched mainly in sagittal plane activities. Overuse combined with a forceful swing on a planted foot during the downswing may be a causative factor for lead ACL damage (Marshall & McNair, 2013). It is known that maximum anterior tibial translation takes place when knee flexion is in the range of 20° - 45° (Dargel et al., 2007). Additionally, the ACL may also be stretched under combined loads, as it is known to provide a restraint during internal

rotation of the tibia on the femur (Marshall & McNair), such as that seen in the lead knee during the downswing. It is therefore the lead-side ACL that is more likely to be injured when stretched, resulting from anterior translation and internal rotation of the tibia.

A 1998 analysis (Gatt et al.) studied forces and moments (twisting forces) that are generated in the lead and trail knees during the golf swing. The authors observed that the main forces affecting the ACL ligament are anterior shear forces (external forces from the ground against which the ACL must pull the tibia back), varus moments (forces resulting in the pull of the tibia away from the body's midline) and internal rotation moments (forces generating internal rotation). All these forces and moments are especially disruptive when the knee is flexed between 0° and 30° . Some of the Gatt et. al research findings were corroborated by Marshall & McNair (2013) who stated, in their review article, that there is greater stress on the ACL when the knee is more extended than flexed.

Lynn and Noffal (2010), in their golf research looked at frontal plane lead knee moments only, and stated that the external abduction or valgus moment (acting from the ground up) seen in their study immediately before impact, can increase stress on the ACL. A recent research article, also on the lead knee (Purevsuren et al., 2017), stated that peak forces and strain on the ACL occur immediately post-impact and into the follow-through. As the authors concluded, a combination of less knee flexion, more internal tibial rotation, greater joint compressive force, and external knee adduction moment during the downswing or follow-through, places considerable strain on the ACL. It may be noted that these authors describe the tibia as being in external rotation during the downswing and internal rotation during the follow-through.

Female athletes, as described in a literature review of ACL injury among soccer players, are more prone to ACL injury, especially during deceleration movements, when the knee is

extending and in a valgus position, and weight is being shifted over onto the lead leg while the foot is fixed to the ground (Alentorn-Geli et al., 2009). The review also stated that lateral trunk displacement in the direction of the lead knee, is a strong predictor of knee injury. As these are all positions and loads relevant to the lead knee during the golf downswing, this review may help to further elucidate the likely mechanisms of lead ACL injury in golf. It may be summarized, then, that dynamic knee valgus combined with forceful quadriceps activity leading to rapid knee extension (during the phase of 0° to 30° flexion) are pre-impact causative factors of ACL injury. At the same time, post-impact injury mechanisms include adduction and internal rotation of the joint.

MCL damage. The MCL stretches to resist abduction forces (when the knee is closer to the body's midline), as well as internal and external rotation in the flexed knee, and can be subjected to strain when those positions are exaggerated, as they may be close to the top of the golf backswing (Marshall and McNair, 2013).

LCL damage. The LCL is known to limit external rotation of the knee in the horizontal plane, especially when the joint is close to full extension (Marshall and McNair, 2013). It can thus be damaged when it is greatly stretched during excessive external rotation with extension, as seen in the lead knee close to club-ball impact.

Ribs. The most common golf-related injury to the ribs involves stress fractures, which are said to be the result of excessive muscle force applied to bone (Taneja, Negromonte & Skaf, 2013).

Stress fracture lead side ribs. During the back- and through-swings, a force couple is created by scapular retraction and protraction, with strong muscle forces being generated.

The main muscle involved in this action is the serratus anterior, which serves to keep the scapula in close contact with the thoracic wall (Cabri et al., 2009). Repeated striking of the ground (Lee, 2009) and/or sustained serratus anterior activity have been implicated in lead side rib fracture.

Stress fracture trail side ribs. Trail side rib fractures are not common, especially in experienced golfers. However, one case report of a 45 year old, nine-handicap, right-handed male golfer, discussed the likely mechanism of his right-side, fifth rib fracture (Read, 1994). The author surmised, based on the patient's ability to reproduce pain even after his normal swing had returned to a pain-free status, that a failure to rotate through the ball could be a causative factor. He stated that, "This golfer was a hooker of the ball but would block the shot when he failed to rotate through the ball, rather sliding his right side under". (Read, 1994, p. 206).

Part 3. Biomechanically Evaluated Golf Movements and Loads

In Part 3, an attempt has been made to more formally correlate actually measured swing mechanics with those that have been claimed to either cause or exacerbate commonly seen golf injuries. A search of published biomechanical research was made for this purpose. It was considered to be an important exercise because movements and loads have previously been ascribed to golf movements and golf-related injury without the data to substantiate the proposed injury mechanisms. Additionally, many authors have implicated "poor swing mechanics" as a reason for injury causation, but no formal scientific research exists that has validated what exactly poor swing mechanics might constitute, according to Sherman & Finch (2000).

An exemplar collection of relevant information has been reported in this section, and no attempt has been made to source only that data which directly correlates to the assumed causative factors described in Part 2 of the Results section. The purpose for including considerable content germane to the topic is so that as researchers are able to identify new causes

for tissue-level damage during the unique movement of the golf swing, many more of the biomechanically-derived findings reported in this article will become relevant. In some cases, the researchers had already correlated their results with typical injury-causing mechanisms for specific tissues. In other cases, data has been extracted from biomechanics research, and analyzed in a manner suitable for the purpose of this study, even if the authors of the concerned journal articles used their data to examine quite different aspects of the golf swing. In the latter case, it was not possible to know significance or effect size, but merely the magnitude of the raw data provided.

Some definitions used in this section, and already explained in the previous section, are “force”, which is linear and “moment”, which is angular. Movements, forces and moments can be either positive or negative depending on the convention used by the researchers to describe their inertial reference frame. While the signs have been reported here as presented in the original articles, a description of the respective movement, force or moment has been included in all cases, to better explain each situation. For instance, some articles may attach a negative sign to movement away from target, and a positive sign to movement towards target; in this paper, the data is always accompanied by a description such as “away from target sway”.

Spine – lumbar and thoracic. It is important to know the active (self-generated) ROM of the lumbar spine in order to have a basis for comparison with movements seen during the golf swing. A study (Tojima, Ogata, Yozu, Sumitani, & Haga, 2013) which compared two different methods of measurement (optical cameras vs electrogoniometer) in seven healthy males of mean age 30.3 years, found that the maximum amounts of ROM measured in any one direction were: flexion 50°, extension 24°, lateral flexion 16° and axial rotation 8°. Moreover, it should be noted

that overuse injury likelihood is exacerbated as the speed at which movements are made increases.

Kinetics and EMG. Forces on the spine can be compressive (directed downwards), as well as in the anteroposterior and mediolateral directions. A recent study by Dale and Brumitt (2016) calculated peak spinal forces for the full swing of 13 experienced golfers. Results showed that peak full swing forces, all normalized to body mass were: compression, $7.6 \text{ N} \pm 0.4 \text{ N}$, taking place at an average of 0.02 s prior to impact; lateral shear, $3.2 \text{ N} \pm 0.2 \text{ N}$, an average of 0.05 s before impact; and anteroposterior shear, $2.0 \text{ N} \pm 0.2 \text{ N}$, 0.08 seconds on average before impact. All these loads occur mere milliseconds prior to impact and considerably load the lumbar spine, during a swing phase when it is being moved at near maximal speed.

A 1990 study (Hosea, Gatt, Galli, Langrana & Zawadsky) used an inverse dynamics-based calculation of loads at the L3-L4 lumbar motion segment along with EMG. The study compared four professional and four amateur (average handicap 16) male golfers, who all used a 5-iron club. Peak lateral bending load for amateurs was $963.13 \text{ N} \pm 298.37 \text{ N}$, which was 81% greater than that generated by professionals, at $531.19 \text{ N} \pm 232.87 \text{ N}$. Both groups showed trail-side side-bending during the downswing leading up to impact. Compressive loads were seen to be lower in amateur golfers $6100.08 \text{ N} \pm 2413.05 \text{ N}$, compared to those generated by professionals ($7584.52 \text{ N} \pm 2422.44 \text{ N}$). When these forces were normalized by body weight (as forces in the Dale and Brumitt were), both groups generated peak compressive forces that were over eight times body-weight (8.13 and 8.57 respectively).

Finally, the spine is also subjected to rotational forces termed torque or moment, and peak torque for amateurs (Hosea et al., 1990) was $85.40 \text{ Nm} \pm 34.21 \text{ Nm}$. This was about double that of the professionals, who generated $56.83 \text{ Nm} \pm 28.03 \text{ Nm}$. EMG activity was reflective of

the typical loads generated by the two groups, and during the time of peak loads (downswing and early follow-through), the trail side external obliques, rectus abdominus and paraspinal muscles (erector spinae muscle group) were active, with amateurs showing greater peak muscle activation than professionals. The study concluded that professional golfers were able to accelerate the club with lower spinal loads, in all directions except compression (Hosea et al., 1990).

Yet another research paper (Lim, Chow, & Chae, 2012), studied lumbar spinal loads with respect to body weight, in young, right handed, male golfers ($19.4 \text{ years} \pm 0.9$) with a mean handicap of $0.8 (\pm 1.1)$. Peak compressive loads (normalized, and expressed in terms of, body weight) were: -6.53 ± 1 and -6.29 ± 1.46 during the forward (early) downswing and acceleration (latter part of the downswing) phases of the swing, with the minus sign indicating a downward direction. These loads were reflected by the high, electromyographically studied, trail side erector spinae and lead side rectus abdominus muscles' activity during the forward swing. The acceleration and early follow-through phases are known to be the shortest phases of the full-swing, yet are phases when maximum stress is imposed on the lumbar spine. The considerable rotational speed of these two phases is generated by the trail external oblique and lead internal oblique abdominal muscles.

The activity of the trail sides of those two muscles of trunk rotation, along with that of the rectus abdominus muscle showed increased activity (7%, 6% and 19% increases respectively, compared to average EMG activity) during the acceleration to early follow-through phases, indicating lateral bending activity. Compared to peak compressive loads, the maximum AP shear load (normalized by body weight) was far less, and was the highest in the early and late follow-through phases: 0.97 ± 0.35 and 1.64 ± 0.35 respectively (positive numbers indicating a tendency for the L5 vertebra to move forwards relative to L4). Maximum mediolateral shear

(normalized by body weight), away from target during the early downswing and towards target from the acceleration phase (-0.04 ± 0.42) until late follow-through (-0.44 ± 0.56), was much smaller than the other two forces (Lim, Chow, & Chae, 2012). Overall, across all studies, compressive loads on the spine were greater than either anteroposterior or mediolateral shear forces.

Kinematics and EMG. A study of elite golfers' swings found that professional golfers' peak X-Factor (the angular difference between pelvis and upper torso rotation), occurring during the early downswing, was $56^\circ \pm 4^\circ$. Peak S-Factor (three dimensional shoulder obliquity compared to the horizontal plane) occurring post-impact, was $48^\circ \pm 4^\circ$. Finally, peak O-Factor (three dimensional pelvic obliquity compared to the horizontal plane) occurring approximately at impact was $16^\circ \pm 4^\circ$. These numbers indicate a considerable difference in thorax versus pelvis torsion, as well as in combined rotation and lateral flexion, as seen at the pelvis and shoulders. The movements place the different regions of the spine into very divergent positions (Meister et al., 2011).

As X-Factor has been implicated in lumbar spine injury because of the differential rotations of the thoracic and lumbar spines, it is useful to know more about the angles reached from a study which compared low-skilled male and female golfers of mean handicap 30.8 with a similarly mixed group of skilled golfers with mean handicap 0.80 (Okuda, Gribble, & Armstrong, 2010). Both groups had similar upper trunk and pelvic rotations at the top of the backswing, giving the two groups a mean differential rotation between the shoulders and the pelvis in the range of 46° to 54° . However, the skilled golfers had significantly less pelvic rotation ($10.1^\circ \pm 7.9^\circ$) by approximately mid-downswing compared to the low-skilled golfers ($20.7^\circ \pm 13.7^\circ$). With similar shoulder positions but less pelvis rotation towards target by mid-

downswing, the skilled players created a greater angle (X-Factor Stretch; the downswing version of X-Factor) between the shoulders and pelvis (Cheetham, Martin, Mottram, & St Laurent, 2001) at mid-downswing.

As compression, which depends on the amount of forward flexion in the pelvic region, is the largest force on the lumbar spine, the study comparing low-skilled with skilled golfers is informative (Okuda et al., 2010). While the former had minimal forward flexion at both ball impact and mid follow-through, the latter had significantly greater forward flexion at both those stages: $6.6^\circ \pm 5.2$ and $10.5^\circ \pm 5.8^\circ$. When comparing male and female professional golfers with respect to forward flexion (Zheng, Barrentine, Fleisig, & Andrews, 2008), females had significantly less forward flexion at address, top of backswing and impact ($32^\circ \pm 4^\circ$, $25^\circ \pm 4^\circ$, and $29^\circ \pm 6^\circ$ respectively) compared to males ($35^\circ \pm 4^\circ$, $31^\circ \pm 4^\circ$, and $33^\circ \pm 3^\circ$ respectively). These results have been corroborated by the fact that compressive loads during the downswing are greater in professionals than in amateurs (Hosea et al., 1990), and are possibly also greater in male professionals than in female professionals.

One early (1994) study on golf swing spine and hip motions in male golfers (McTeigue, Lamb, Mottram, & Pirozzolo) compared angles between 51 PGA Tour players, 46 Senior PGA Tour players and 34 amateur players (mean handicap 17.5, range 5-36). Forward bending was maximal at address (ranging from a mean of 23° for seniors to 28° for PGA Tour players) and remained between a mean of 16° for the seniors and amateurs to 19° for the Tour players, at impact. Lateral flexion started at $6^\circ \pm 1^\circ$, $8^\circ \pm 1^\circ$ and $7^\circ \pm 1^\circ$ respectively to the trail side for the three groups (PGA Tour, Senior PGA Tour and amateurs) at address, and was then between a mean of 3° (Tour players) and 16° (amateurs) to the lead side, at the top of the backswing. From there, all three groups moved into greater trail-side lateral flexion than was seen at address, to

reach impact with $31^\circ \pm 1^\circ$, $28^\circ \pm 2^\circ$ and $21^\circ \pm 2^\circ$ of trail-side side bend respectively. Forward and side bending angles were measured between the pelvis and the mid-thoracic spine. All three groups had their upper bodies (measured at the level of the mid-thoracic spine) $5^\circ \pm 1^\circ$ open (facing target) at address. They then rotated their upper bodies closed at top of the backswing: PGA $87^\circ \pm 3^\circ$, Senior PGA $78^\circ \pm 4^\circ$ and amateurs $87^\circ \pm 4^\circ$; with their pelvis also being closed by $55^\circ \pm 3^\circ$, $49^\circ \pm 3^\circ$ and $53^\circ \pm 4^\circ$ respectively. Finally, at impact all three groups were open in the upper body by: $26^\circ \pm 3^\circ$, $28^\circ \pm 4^\circ$ and $27^\circ \pm 3^\circ$ respectively, and were open in the pelvis by $32^\circ \pm 3^\circ$, $34^\circ \pm 4^\circ$ and $35^\circ \pm 3^\circ$ respectively. Thus, overall it could be said that golfers retain forward flexion throughout the swing. They also change from lead-side lateral flexion during the backswing to trail-side lateral flexion at impact. Finally, the trunk rotates from a slightly open position at address, to closed at the top, and then considerably open at impact, with the upper torso rotating in a differential manner from the pelvis.

Another group of researchers who compiled the results of 308 male and female golfers (Chu et al., 2010), also provided information on trunk lateral bending, pelvis axial rotation and pelvis superior-inferior shift. The highest measured lateral bend was at impact ($14.4^\circ \pm 6.5^\circ$), at which time its velocity was $66.3^\circ/\text{s} \pm 54.0^\circ/\text{s}$. Pelvic axial rotation went from $49.0^\circ \pm 12^\circ$ closed at the top to $0.4^\circ \pm 12.7^\circ$ closed by the acceleration phase, achieving a maximal downswing velocity of $388.1^\circ/\text{s} \pm 77.4^\circ/\text{s}$ during that very brief time period. The pelvis continued into an open position of $35.2^\circ \pm 12.3^\circ$ for impact, but had begun to decelerate by then. Finally, superior-inferior movement of the pelvis went from $0.03 \text{ m} \pm 0.05 \text{ m}$ down at the top of the backswing, to $0.05 \text{ m} \pm 0.05 \text{ m}$ up by the acceleration (late downswing) phase, with a maximal

velocity of $0.44 \text{ m/s} \pm 0.26 \text{ m/s}$. The trunk continued up after the acceleration phase reaching a height of $0.09 \text{ m} \pm 0.06 \text{ m}$ at impact. These findings once again corroborate the fact that there is considerable change in direction of movement from the top of the backswing to impact.

Maximum spinal movements in all three planes with the driver and 7-iron clubs were measured in a research study of 44 healthy male professional golfers (Lindsay, Horton, & Paley, 2002). The authors discovered that these golfers had $28.9^\circ \pm 10.9^\circ$ and $35.1^\circ \pm 12.8^\circ$ forward flexion for the driver and 7-iron respectively, at address. Maximum forward flexion ROM seen during the entire swing for the two clubs was $45.6^\circ \pm 9.7^\circ$ and $51.0^\circ \pm 9.9^\circ$ respectively. Trail-side lateral flexion went from $6.9^\circ \pm 3.4^\circ$ for the driver at address to a maximum across the entire swing of $26.3^\circ \pm 5.2^\circ$; and went from $6.7^\circ \pm 3.2^\circ$ at address to a maximum of $27.9^\circ \pm 4.8^\circ$ for the 7-iron. This side bending range of movement took place at velocities of $109.2^\circ/\text{s} \pm 25.3^\circ/\text{s}$ (driver) and $121.7^\circ/\text{s} \pm 24.8^\circ/\text{s}$ (7-iron). Additionally, towards-target rotational velocity was $194.8^\circ/\text{s} \pm 54.6^\circ/\text{s}$ and $180.3^\circ/\text{s} \pm 50.8^\circ/\text{s}$ for the driver and 7-iron respectively. The 7-iron had significantly greater forward flexion as well as trail side lateral bending velocity than the driver, while the driver had significantly greater target-side axial rotation than the 7-iron. Thus it could be said that both clubs were able to impose considerable loads on the spine. Moreover, lead-side lateral flexion during the backswing was $7.1^\circ \pm 6.0^\circ$ for the driver and was significantly greater for the 7-iron at $9.8^\circ \pm 5.9^\circ$, indicating that there was both greater lead- and trail-side lateral bending with the 7-iron, which movements, the authors suggested, could cause LBP.

Finally, as lateral flexion has been implicated in lumbar injury, the comparative results between female and male professional golfers, both using their driver-clubs, is informative. The angles were similar for both genders, with lead-side lateral flexion of $-11^\circ \pm 10^\circ$ and $-10^\circ \pm 12^\circ$ respectively at the top, changing to a trail side lateral flexion of $29^\circ \pm 6^\circ$ and $31^\circ \pm 5^\circ$

respectively at impact (Zheng et al., 2008). Combining results for lateral bending from the top of the backswing to impact, from this and other previously mentioned studies, with the information on trail-side lateral bending velocities (Chu et al., 2010), may indicate that trail-side lateral flexion velocities have to be greater to move through the increased ROM created by moving from lead-side lateral flexion to trail-side lateral flexion within the limited time of the downswing. Additionally, with respect to lumbar spine injury, the magnitude of lateral flexion in the Zheng et al. and previously mentioned studies should be compared to the normal active lumbar lateral flexion ROM of a maximum of 20° in either direction (Tojima et al., 2013).

One study (Horan, Evans, Morris, & Kavanagh, 2010) comparing linear and angular displacement between 19 male and 19 female skilled golfers using the driver club found that mean backswing mediolateral sway for males, for the thorax and pelvis respectively, was $-7.3 \text{ cm} \pm 2.7 \text{ cm}$ and $-4.5 \text{ cm} \pm 3.1 \text{ cm}$ (the minus sign indicating movement away from target). However, at impact, while the thorax remained away from target by $-4.2 \text{ cm} \pm 3.4$, the pelvis swayed towards target by $10.9 \text{ cm} \pm 2.9 \text{ cm}$. This trend continued when sway was adjusted by body height and mass, indicating a considerable mediolateral difference in the positions of the upper and lower sections of the spine at impact. This pattern was seen in female golfers too, who had a top of backswing thorax and pelvis sway of $-6.1 \text{ cm} \pm 2.5 \text{ cm}$ and $-5.1 \text{ cm} \pm 2.6 \text{ cm}$ respectively, which changed to $-1.3 \text{ cm} \pm 4.1 \text{ cm}$ and $10.1 \text{ cm} \pm 4.5 \text{ cm}$ respectively at impact. While mediolateral shear forces may be smaller than compressive forces (Dale & Brumitt, 2016; Hosea et al., 1990), this amount of “sway” between the thorax and pelvis at the considerable speed of the downswing, may indicate sizeable shear between specific vertebral segments.

Yet another research project (Horan et al., 2010) served to corroborate the movements of the lumbar region that were seen in the previously mentioned studies. The authors observed that

mean anteroposterior (AP) pelvic tilt at the top of the backswing was $24.7^{\circ} \pm 3.2^{\circ}$ and $23.4^{\circ} \pm 5.7^{\circ}$, and decreased to only $4.7^{\circ} \pm 4.7^{\circ}$ and $5.2^{\circ} \pm 5.7^{\circ}$ for males and females respectively at impact. This indicates that a considerable amount of the typically expected forward flexion of address is retained to the top as well as while returning to impact, for both groups. In the same study, pelvic lateral tilt at impact for males and females was $11.0^{\circ} \pm 3.2^{\circ}$ and $6.1^{\circ} \pm 2.9^{\circ}$, with pelvic lateral tilt angular velocities being $107^{\circ}/s \pm 49^{\circ}/s$ and $69^{\circ}/s \pm 38^{\circ}/s$ respectively.

Additionally, it would appear that lateral tilt velocity is not maximized at impact, as maximum lateral tilt velocity in the pelvic region (Horan et al., 2010) reached still higher values ($199^{\circ}/s \pm 57^{\circ}/s$ and $136^{\circ}/s \pm 41^{\circ}/s$ for males and females respectively). These findings may indicate that the lateral tilt angle continues to increase post impact. These angles and velocities have been reported here for the pelvis alone, and the trends were found to be similar for the thorax, indicating, overall, considerable angular tilt in the forward and lateral directions, which take place at rapid velocities. All angles and velocities reported here were significantly lower for females than for males.

Spine tissue injury mechanisms. Summarizing the findings of spinal biomechanical research, it may be said that as the spine has been observed to be subjected to compression, lateral flexion and axial rotation occurring at the high velocities at which these latter two movements take place, disc injury is plausible. The existence and magnitude of spinal hyperextension during the follow-through has not been measured; however both compression and rotation have been observed in the golf swing, which are risk factors for lumbar facet injury and for spondylolysis.

Additionally, movements that rotate different segments of the spine through dissimilar or oppositely-directed transverse-plane (e.g. X-Factor) and frontal plane (e.g. lateral sway) motions may be problematic, as torsional or shear forces can be created between individual vertebral segments. As stated by Gluck et al. (2008), even 2° to 3° of inter-segmental rotation can induce microtrauma to the lumbar facet joints, as they do not have the orientation to permit excessive rotation. Such rotation can be seen with the X-Factor, which is known to achieve a peak difference in upper torso relative to pelvis rotation of $56^{\circ} \pm 4^{\circ}$ (Meister et al., 2011). Moreover, one review article on scoliosis (lateral and rotational curvature of the vertebral column) stated that even a normal spine can be altered during normal movement, when there is an imbalance of forces along the spine from asymmetric loading (Hawes & O'Brien, 2006). Such asymmetric loading was observed (Horan et al., 2010) in the oppositely directed pelvic and thoracic sways during the downswing.

Spine – thoracic. As female professional golfers (Zheng et al., 2008) have significantly less forward trunk flexion at address, top of backswing and impact ($32^{\circ} \pm 4^{\circ}$, $25^{\circ} \pm 4^{\circ}$, and $29^{\circ} \pm 6^{\circ}$ respectively) compared to males ($35^{\circ} \pm 4^{\circ}$, $31^{\circ} \pm 4^{\circ}$, and $33^{\circ} \pm 3^{\circ}$ respectively), it would be expected that their thoracic spines also have less forward flexion, and thus less compressive loads placed upon them. It is therefore difficult to explain, in biomechanical terms, why they may have greater compression fractures in the thoracic region. As post menopausal women are more prone to sustaining such fractures, the main reason could be hormonal rather than biomechanical.

Spine – cervical. One study of four right-handed, elite golfers reported head movements of one of those participants, which, while not an adequate sample size to base conclusions upon, may serve to indicate that the head (and thus some parts of the cervical spine) does move

throughout the swing. The participant, a professional golfer, moved his head 39 mm (3.9 cm) vertically upwards from address to the top of the backswing while using his 7-iron. He then moved it 51 mm (5.1 cm) downwards by the club-shaft vertical position or early downswing, after which it remained in a similar position until impact (Gryc, Zahalka, Maly, Mala, & Hrasky, 2015). The up-then-down movement of the cervical spine is in opposition to the down-then-up movement of the trunk (Chu et al., 2010).

The head/neck region is also known to have changing directions of lateral flexion at different phases of the swing. A study (Zhang & Shan, 2014) looking at golf swing consistency with the driver club found that the head (in 22 golfers of mean handicap 12.3 and mean age 35.1) is tilted $2.5^{\circ} \pm 0.6^{\circ}$ away from target at takeaway or the beginning of the backswing, and gets tilted further to the trail side by $4.2^{\circ} \pm 1.3^{\circ}$ at impact; however this information was partial as the change from takeaway to the top of the backswing is also required to assess the entire range of movement that the head goes through during the golf swing.

When the Gryc et al. (2015) results describing vertical changes in head position are combined with those of Horan and Kavanagh (2012), it can be seen that the head and neck are moved considerably and not in the same manner as the thoracic and lumbar parts of the spine. Horan and Kavanagh showed that, for 14 male professional golfers, peak speeds as a percentage of downswing time (impact = 100%), of the head, thorax and pelvis were achieved in the order of pelvis first at 76 ± 5 , head second at 79 ± 19 and thorax third at 83 ± 9 .

The three body segments also had very different peak speeds, $507^{\circ}/s \pm 52^{\circ}/s$, $650^{\circ}/s \pm 60^{\circ}/s$ and $210^{\circ}/s \pm 56^{\circ}/s$ for the pelvis, thorax and head respectively. As the golf swing is said to have ground-up, proximal-to-distal kinematic sequencing (Cheetham et al., 2008), it makes sense for the thorax to speed up after the pelvis, but the results show that the head, which is moved by

muscles connecting it to the cervical spine, is not involved in the sequencing process, as it reached peak velocity immediately after, yet at a far slower speed than, the pelvis.

There is also a very mixed order of the time of peak velocity as a percentage of the downswing (Horan & Kavanagh, 2012), when comparing velocities of forward tilt (FT), lateral tilt (LT) and axial rotation (AR). The overall order was, head LT 67 ± 23 , pelvis AR 71 ± 6 , head AR 77 ± 19 , thorax AR 77 ± 12 , thorax LT 78 ± 12 , pelvis LT 81 ± 6 , head FT 82 ± 11 , pelvis FT 89 ± 8 and thorax FT 95 ± 5 . Thus the head moved laterally (to the trail-side) first, then there was AR of all three parts, then LT of the thorax and pelvis, and finally FT of the head followed by FT of the pelvis and thorax. All these movements occur within the 0.34 s or less that any golf downswing lasts (Zheng et al., 2008), and indicate that head movements take place out of sequence from those of the thorax and pelvis which are known to be tightly coupled (Horan & Kavanagh). Finally, the authors also noted that head peak lateral flexion velocity ($156^\circ/\text{s} \pm 57^\circ/\text{s}$) was significantly greater than axial rotation velocity ($67^\circ/\text{s} \pm 114^\circ/\text{s}$), so that the head and neck go from an attempted neutral at the top into trail side lateral flexion at impact, at great speed, which may be a factor related to injury.

Another head motion is its translation away-from, and then towards, the target during the back- and down-swings, which is greater in elite golfers, as reported by Sanders & Owens (1992). In their study on novice versus elite male golfers, they found that the chin, considered a good indicator for lateral body movement, moved away from target during the backswing and towards target during the downswing and follow-through. The chin's most away-from-target position was $-29.3 \text{ cm} \pm 8.1 \text{ cm}$ in elite golfers, and its most measured forward position was $19.5 \text{ cm} \pm 8.0 \text{ cm}$, post-impact. During impact, however, elite players minimized the lateral

movement of their chins. This sudden reduction of chin (and thus head/neck) movement may further corroborate the idea of head movement being delinked from that of the torso.

The anecdotally described causes for cervical fractures (Part 2) were a stable head compared to a rotating torso during the backswing, and hyperextension during the follow-through. The limited information available on head/neck movement during the golf swing does indicate that the head and torso move in different directions at different times, and that no coupling exists between the thorax and the head/neck region (unlike the strong pelvis-thorax coupling). Together these un-coupled translational (sideways and upwards) and rotational movements of this area of the spine might cause certain tissues to be subjected to tensile or compressive loads. Additionally, the head appears to be going into extension up to the top of the backswing and then into flexion during the downswing, which is a combination of movements that resembles those seen during whiplash. It may thus be stated that disc and facet injury in the cervical region are plausible during the golf swing; however, the lead-side laterality of both injuries cannot be explained given the available information.

Shoulder.

Kinematics. Mitchell, Banks, Morgan, & Sugaya (2003) focused on lead and trail shoulder movements, in degrees, and compared the joint angles between college-aged, middle-aged, and senior male golfers, all using the driver club. The sample sizes for the three groups were 19, 24 and 22 respectively. The mean ages for the three groups were 20 years, 36 years and 68 years respectively, while their mean handicaps were 3, 9 and 14 respectively. Lead arm maximum horizontal adduction in the transverse plane (90° adduction was defined as a horizontal movement of the humerus in front of the trunk) was seen at the top of the backswing: $125^\circ \pm 7^\circ$, $126^\circ \pm 7^\circ$ and $119^\circ \pm 6^\circ$ for the three groups, respectively. Vertical elevation of the

lead side shoulder (described as the angle between the arm and a vertical line) was $110^{\circ} \pm 10^{\circ}$, $107^{\circ} \pm 9^{\circ}$ and $94^{\circ} \pm 8^{\circ}$ respectively.

End range positions for the trail arm during the follow-through were similar to those of the lead arm at the top of the backswing, for the three groups of the Mitchell et al. (2003) study. Maximum trail arm horizontal adduction was $121^{\circ} \pm 8^{\circ}$, $114^{\circ} \pm 7^{\circ}$ and $108^{\circ} \pm 8^{\circ}$. Vertical elevation was $112^{\circ} \pm 8^{\circ}$, $114^{\circ} \pm 11^{\circ}$ and $103^{\circ} \pm 11^{\circ}$ respectively. It can thus be seen that younger golfers tend to have greater lead and trail side across-the-body adduction and elevation ranges at both extremes of swing motion, that is, at the end of the back swing and the end of the follow-through.

In one study on eight female Ladies Professional Golf Association (LPGA) players (Lemak, Fleisig, Welch, Marting & Zvijac, 1994), maximum lead arm horizontal adduction (measured as the angle between the shoulder line and the arm line, when the arm is anterior to the shoulder line) was $133^{\circ} \pm 5^{\circ}$ in the backswing (indicating an angle of about 57° between the shoulders and the lead arm). The maximum “abduction” angle between the trunk and the lead shoulder was $79^{\circ} \pm 14^{\circ}$ at the top of the backswing, and this angle may be considered to be similar to a lead arm elevation. The trail shoulder’s horizontal abduction was $51^{\circ} \pm 19^{\circ}$ (measured as the angle between the shoulder line and the arm line, with the arm posterior to the shoulder line), and this is an indication of how extended the arm was at the top of the backswing. Finally, maximum trail shoulder abduction (measured as the angle between the arm and the trunk) was $39^{\circ} \pm 14^{\circ}$ at the top of the backswing. The authors did not report whether these angles were for the driver, five-iron or pitching wedge clubs used in their study.

A more recent study (Chu et al., 2010) measuring several biomechanical variables, had 266 male and 42 female participants with an average handicap 8.4 ± 8.4 , who also used the

driver club. This study showed a mean leading arm angle of $222.7^\circ \pm 9.5^\circ$ (from a horizontal line aimed in the direction of the target) at the top of the backswing, which may be interpreted as a lead arm elevation that is almost 45° above horizontal. The authors did not report trail arm abduction at the top. Thus, two studies (Chu et al., 2010; Mitchell et al. 2004) indicate that the lead arm is elevated considerably above horizontal at the top of the backswing, while one (Lemak et al., 1994) reveals considerable trail arm abduction and horizontal abduction, similar to extension.

Finally, a research analysis of 72 male golfers divided into four groups - Tour professionals, low handicap, mid handicap and high handicap golfers – measured shoulder joint kinematic variables, among others (Zheng et al., 2008). At the top of the backswing, lead arm shoulder horizontal adduction (defined as the angle between the humerus and a vector from the trail shoulder to the lead shoulder) was $125^\circ \pm 6^\circ$, $123^\circ \pm 5^\circ$, $119^\circ \pm 6^\circ$ and $115^\circ \pm 8^\circ$ for the four groups respectively, with a larger number indicating less space between the shoulder line and the lead arm. The lead arm then changed its direction of movement, and generated peak angular abduction velocities at the following percent of downswing completion for the four groups: $72\% \pm 13$, $75\% \pm 18$, $87\% \pm 17$ and $91\% \pm 18$. The professional golfers thus had greater shoulder horizontal adduction at the top of the backswing, and their downswing velocities in the opposite direction peaked earlier.

The trail arm, in this same study, was at maximum external rotation of $66^\circ \pm 11^\circ$, $61^\circ \pm 15^\circ$, $47^\circ \pm 24^\circ$, and $46^\circ \pm 17^\circ$ for the four groups respectively, at the top of the backswing, and then went into internal rotation, during the downswing, with angular velocities which were: $522^\circ/\text{s} \pm 234^\circ/\text{s}$, $518^\circ/\text{s} \pm 150^\circ/\text{s}$, $456^\circ/\text{s} \pm 225^\circ/\text{s}$ and $326^\circ/\text{s} \pm 124^\circ/\text{s}$ respectively. Once again

the professional golfers had maximum trail shoulder external rotation at the top, and reached higher angular velocities of shoulder internal rotation during the downswing.

EMG. An EMG study of shoulder muscle activity during the golf swing (Jobe, Moynes, & Antonelli, 1986), measured activation levels compared to maximum manual muscle test (MMT), of several shoulder muscles, for seven male golfers. The authors stated that 30%-60% MMT is considered moderate activity, whereas greater than 60% MMT is marked activity. This article did not provide numerical data. Their graphs showed lead side subscapularis (shoulder internal rotator) activity to start at over 80% at takeaway, and, while dropping slightly during the forward swing (early downswing), it increased to greater than 100% of MMT during the acceleration and follow-through phases. The lead side latissimus dorsi and pectoralis major (both are shoulder adductors, internal rotators and extensors) also showed their maximal activity during the acceleration phase, which was greater than 80% in both cases.

The trail side subscapularis, in the meantime, showed marked activity during the acceleration and the follow-through phases. The trail side latissimus dorsi and pectoralis major fired at almost marked levels during the forward swing, reaching marked levels during the acceleration phase (greater than 100%), and remaining marked during the follow-through. The authors explained that maximal activity bursts of over 100%, seen in many of the muscles, were able to take place because the MMT values only represent sustained, not instantaneous, effort. Based on the results of this study it may be said that the subscapularis RC muscles of both the lead and trail sides remain considerably active throughout the swing, and the trail shoulder's latissimus dorsi and pectoralis major are very active during the downswing (Jobe et al., 1986).

A review paper describing shoulder muscle recruitment during the golf swing (Escamilla & Andrews, 2009), showed that, as a percent of maximum voluntary isometric contraction

(MVIC, similar to MMT) forces, the lead supraspinatus (shoulder abductor) had its maximal activity ($28\% \pm 20\%$ and $28\% \pm 14\%$ respectively), during deceleration and follow-through, typically termed early and late follow-through. The lead infraspinatus (shoulder external rotator) had peak activity ($61\% \pm 32\%$) during deceleration and the lead subscapularis had moderate or almost moderate activity (as defined by Jobe et al., 1986) throughout all five phases of the swing from takeaway to late follow-through ($33\% \pm 23\%$, $29\% \pm 24\%$, $41\% \pm 34\%$, $23\% \pm 27\%$ and $35\% \pm 27\%$). The trail supraspinatus had an activity level of $25\% \pm 20\%$ (compared to percent of MMT) during takeaway, and was less active than that during the other phases. The trail infraspinatus was also maximally active during the takeaway ($27\% \pm 24\%$) and did not have much activation thereafter. Finally, the trail subscapularis had moderate or marked activity during four phases, from the forward swing (early downswing) to the end of the follow-through ($49\% \pm 31\%$, $68\% \pm 67\%$, $64\% \pm 67\%$ and $56\% \pm 44\%$).

Shoulder tissue injury mechanisms. Synthesizing the information from shoulder motion and EMG studies, considerable horizontal adduction and vertical elevation of the lead and trail arms is seen at the ends of the backswing and the follow-through respectively. Muscle activation patterns complement kinematic data to indicate that the lead side shoulder extensors (latissimus dorsi and pectoralis major) fire during the late downswing, a lead shoulder internal rotator (subscapularis) is moderately active throughout the swing, while a lead shoulder external rotator (infraspinatus) is active in the early follow-through (Escamilla & Andrews, 1990; Jobe et al., 1986).

The trail shoulder reaches considerable abduction and horizontal abduction/extension at the top of the backswing. Then there is almost marked, followed by marked activity of the trail internal rotators (latissimus dorsi, pectoralis major, subscapularis) and adductors (latissimus

dorsi and pectoralis major) during the downswing. Kinematic data also indicates considerable trail shoulder cross-body adduction and elevation during the follow-through (Mitchell et al., 2003; Zheng et al., 2008).

Based on available data, it may be surmised that as there is considerable shoulder ROM at the end of the backswing and the end of the follow-through, risk factors exist for RC injury, subacromial bursitis, AC joint damage in both shoulders, as well as labral tears and biceps tendinitis in the lead shoulder. Younger and professional golfers would be more susceptible to such injuries, because of greater horizontal adduction and elevation at both the top of the backswing and the end of the follow-through (Mitchell et al., 2003; Zheng et al., 2008).

In the trail shoulder, RC injury-causing positions have been observed at the top of the back-swing (Lemak et al., 1994; Zheng et al., 2008). The expected causative mechanisms of other trail shoulder injuries such as subacromial bursitis and AC joint compression have also been detected.

Elbow.

Kinematics. The kinematic analysis of 72 male golfers divided into four skill levels (Zheng et al., 2008) measured lead and trail elbow flexion angles at address, top of backswing, and impact, as well as maximum angular velocities and percent of downswing time at which those peaks angular velocities occurred. The only significant difference in the four groups, all using a driver, was lead elbow flexion at impact, which was highest ($45^{\circ} \pm 8^{\circ}$) for the high handicap golfers and lowest ($31^{\circ} \pm 8^{\circ}$) for the low handicap golfers. The same group of authors (Zheng et al., 2008) also compared both elbow angles in male and female professional golfers using the driver club, and found that both groups had similar lead elbow angles at both the top

and at impact. Together, their two studies indicated that only high handicap golfers had more flexed, or less extended, lead elbows at impact.

Another study compared seven male to five female golfers, who all used their driver clubs and had mean handicaps of 6.6 versus 6.1 (Egret et al., 2006). The authors found that females had greater (not significant, possibly because of small sample size) lead elbow flexion at both address and impact than males ($45.1^\circ \pm 12.2^\circ$ vs $38.0^\circ \pm 5.3^\circ$ and $67.6^\circ \pm 10.5^\circ$ vs $50.9^\circ \pm 7.7^\circ$ respectively). Females, however, had less trail elbow flexion than males, from address to the top to impact (significant only at the top of the backswing). The angles were $37.4^\circ \pm 12.2^\circ$ versus $41.8^\circ \pm 8.4^\circ$; $109.7^\circ \pm 17.3^\circ$ versus $131.5^\circ \pm 12.2^\circ$ and $49.2^\circ \pm 5.2^\circ$ versus $57.3^\circ \pm 20.1^\circ$ respectively, for the three phases. The researchers measured elbow flexion as forearm movement past zero degrees which was the angle between the arm and the completely extended forearm. Thus it may be stated based on results from the Egret et al. (2006) research findings that females may, (with larger sample sizes), have greater lead elbow flexion, and less trail elbow flexion, at impact.

Additionally, differences were seen in peak angular velocities. Lead elbow extension velocities for the professionals, low handicap golfers, mid-handicap golfers and high-handicap golfers were (Zheng et al., 2008): $235^\circ/s \pm 61^\circ/s$, $255^\circ/s \pm 86^\circ/s$, $234^\circ/s \pm 90^\circ/s$ and $166^\circ/s \pm 61^\circ/s$ respectively, with significance seen between the high handicap golfers and all the other groups. Not only did the more skilled golfers develop higher lead elbow extension velocities, they did so significantly closer to impact: $83\% \pm 20\%$, $78\% \pm 18\%$, $77\% \pm 15\%$ and $69\% \pm 14\%$ respectively. This would indicate that for the more skilled golfers, the lead elbow reached its maximum extension closer to impact, when the arm-club system was travelling at its highest speed (Zheng et al., 2008).

Maximum trail elbow angular velocities for the four groups were: $854^{\circ}/s \pm 150^{\circ}/s$, $783^{\circ}/s \pm 129^{\circ}/s$, $726^{\circ}/s \pm 122^{\circ}/s$ and $551^{\circ}/s \pm 126^{\circ}/s$, and were significantly higher for the more skilled-golfer groups, while the time to peak as a percent of downswing time was significantly later in the downswing for the professionals: $99\% \pm 2\%$, $94\% \pm 4\%$, $94\% \pm 7\%$, $91\% \pm 5\%$ (Zheng et al., 2008). Thus maximum velocity was reached much closer to impact for the professionals compared to the other skill levels of golfers. Of greater interest for the purpose of this study however, is a comparison of (despite significance not being known), the differences in mean peak angular velocities (although the standard deviation is quite high) reached for each arm, and the times at which they occurred. The difference in mean peak elbow extension angular velocities between the two elbows was $619^{\circ}/s$ for the professional golfers, with the lead elbow reaching peak angular velocity 17% of downswing time earlier than the trail elbow, which would indicate that the lead elbow was already decelerating by the time the trail elbow reached maximum velocity. For the highest handicap golfers, the difference in mean peak elbow extension angular velocity was a far lower $385^{\circ}/s$, but the difference in time to peak was slightly greater, with the lead elbow extending an average of 22% earlier in the downswing than the trail elbow (Zheng et al., 2008). It is not known, based on currently available information, whether greater differences in speed between the two arms as seen in the professional golfers or greater differences in time taken to reach individual peaks for the lead and the trail arm, might be meaningful indicators of asymmetry between the movements of the two elbows, especially because both upper limbs are connected at the golf club's grip.

While the Zheng et al. (2008) study found mean lead elbow flexion angles (converted here to the angle between the arm and the forearm for easier comparisons) at impact ranging from 135° for the highest handicap group to approximately 146° for the other three groups of

their study, another group of researchers (Bradshaw et al., 2009) found that skilled golfers (handicap range 0 to 1) had a mean lead elbow flexion angle at impact of $182.1^\circ \pm 4.6^\circ$, while nonskilled golfers (handicap range 18 to 25) had a significantly lower mean angle of $174.5^\circ \pm 9.0^\circ$. These latter angles may indicate a slightly more extended lead elbow for both groups, compared to the Zheng et al. (2008) study. Greater elbow extension has been considered a cause of lateral epicondylitis. Finally, another important movement with respect to elbow injury is forearm supination. One group of researchers assessed kinematic variables for a single semiprofessional golfer with a single-digit handicap and found that his lead forearm supination reached approximately $470^\circ/\text{s}$ at impact (Teu, Kim, Fuss, & Tan, 2006).

A study (Neal, Lumsden, Holland, & Mason, 2007) that looked at the sequencing and timing of different body segments, compared self-reported well-timed and mistimed swings of 13 male and 12 female highly-skilled golfers when using their driver clubs. No significant differences were found in segmental timing-related variables between the well-timed and mistimed shots, and no comparison was made between males and females. The mean time to impact of peak angular velocity during the downswing for the lead arm of male participants was 73 ms (standard error of mean [SEM] = 1.8 and 2.3 ms respectively) for both well-timed and mistimed shots. However, the time to peak forearm velocity was 53 ms (SEM = 2.8) prior to impact when well-timed, but 46 ms (SEM = 3.8) prior to impact when mistimed, and the lag time between arm and forearm segments increased from 23 ms (SEM = 3.4) to 29 ms (SEM = 4.4) respectively.

The mean time to impact of female lead arms' peak angular velocity during the downswing was 83 ms (SEM = 2.3 ms) for the well-timed, and 79 ms (SEM = 2.4 ms) for the mistimed shots. Their forearms reached their peak velocities 19 ms (SEM = 2.2) and 18 ms (SEM = 2.9) before impact, for the well-timed and mistimed shots respectively,

with lag times of 66 ms (SEM = 2.8) and 63 ms (SEM = 4.1) for the well-timed and mistimed shots respectively. Females therefore reached peak elbow extension velocities later than males, and also had slightly greater overall forearm speed (mean 895°/s vs 905°/s for well-timed shots), which could be interpreted as late and excessive use of lead side force to extend the elbow rapidly.

Additionally, although the results from the Neal et al. (2007) research study were not significant and no effect size was calculated, the delay in the “catching up” of the lead forearm to the lead upper arm in the mistimed shots for male golfers may be indicative of maximal lead elbow extension taking place closer to impact, which may indicate that the lead extensor muscles place greater stress on the lateral epicondyle of the lead elbow. A rationale for this concept is that if the lead elbow attains maximum extension velocity later in the downswing, it also extends more fully at a swing phase during which the lead shoulder is higher (Chu et al., 2010), and there is greater centripetal force on the lead arm-club system (Muir, 2001). Their delayed elbow extension may be one reason that female (amateur) golfers have a greater risk for elbow injury, as has been reported in epidemiological data (Stockard, 2001).

EMG. An EMG analysis (Farber et al., 2009) of four representative forearm muscles compared muscle activity levels between professional (below 4 handicap) and amateur golfers (handicaps between 10 and 20), in both arms. Although the forearm muscles – both the extensors and flexors - act to move the wrist and hand, they originate at, or close to, the common flexor and extensor origins at the medial and lateral epicondyles of the elbow respectively, indicating their relevance to this section. The four muscles studied were the extensor carpi radialis brevis (ECRB), a wrist extensor whose tendon is the main one implicated in lateral epicondylitis; the flexor carpi radialis (FCR) which acts to flex and radially deviate the wrist; the flexor carpi

ulnaris (FCU) which also flexes the wrist and ulnarly deviates it, and the pronator teres (PT), which is mainly a pronator of the forearm but also flexes the elbow joint (Moore et al., 2010). The only significant difference between professionals and amateurs, with respect to the elbow was seen in the PT muscle, which showed significantly greater lead arm activity during the acceleration phase of the professional golfers; and trail arm activity during the forward swing (early downswing) of the amateurs. Could such activity patterns indicate greater risk of lateral epicondylitis in professionals as the lead forearm is held in pronation and only supinated much later in the downswing when it is likely to achieve greater speed of movement? Moreover, could there be a greater risk of medial epicondylitis in amateurs as the trail forearm pronates early in the downswing, while the elbow also perhaps flexes earlier, both assumed from the earlier pronator teres activity?

Additionally, based on the Jobe et al., (1986) convention rather than on significance, there was marked lead forearm muscle activity, as a percent of maximum, for both the professionals and the amateurs during the forward swing ($68.8\% \pm 24.1\%$ and $74.2\% \pm 70.9\%$ respectively) and during the acceleration ($62.4\% \pm 61.8\%$ and $94.2\% \pm 205.3\%$ respectively) phases for the ECRB, which would be expected as the lead wrist extends, leading up to impact. Moreover, mean trail side ECRB activity for the amateurs was marked: $60.3\% \pm 62.8\%$ and $105.1\% \pm 172.7\%$, during the forward swing and acceleration phases, but was only moderate ($32.9\% \pm 21.9\%$ and $41.1\% \pm 39.9\%$ respectively) for the professionals (Farber et al., 2009). That may indicate greater downswing trail wrist extension, and thus a stretching of the forearm flexors.

Lead elbow tissue injury mechanisms. Putting together all available findings, it appears that the lateral aspect of the lead elbow may be subjected to forceful extension, probably greater

in mistimed shots and in females (Neal et al., 2008). In the professional golfers the reason might be the greater lead elbow extension velocity which additionally was closer to impact. These movements, along with lead forearm supination (Choi et al., 2016), have all been implicated in lead elbow lateral epicondylitis.

Trail elbow tissue injury mechanisms. The greater trail forearm PT muscle activity (Farber et al., 2009), indicative of greater forearm pronation; the greater elbow flexion at impact (Zheng et al., 2008); and the pre-impact wrist extension expected from greater ECRB activity (Farber et al., 2009) together may strain (stretch) the wrist flexors and thus be a risk factor for medial elbow pain in amateur or higher handicap golfers. The risk factor for injury increases when there is greater trail wrist extension (Farber et al., 2009) preceding the “flexor burst”, (Glazebrook et al., 1994) which occurs for all golfers close to impact.

Finally, if the trail upper limb is in a position of shoulder abduction (Lemak et al., 1994; Mitchell et al., 2003); elbow flexion (Egret et al., 2006; Zheng et al., 2008); forearm pronation (Farber et al., 2009); and wrist radial deviation (Cahalan et al., 1991; Zheng et al., 2008) as well as extension (Cahalan et al., 1991; Glazebrook, et al., 1994), there will be greater stretching of the ulnar nerve (Wright et al., 2001). Maximal stretching of the nerve can cause strain, which makes it prone to injury at the cubital tunnel of the elbow. This position is seen at the top and during the early downswing in all golfers.

Wrist/hand.

Kinematics. Both wrists are known to move through a considerable ROM during the swing. “Where do Driver Swings go Wrong?” (Zhang & Shan, 2014) is a study of kinematic variables dedicated mostly to assessing consistency. One relevant result was lead wrist radial deviation at the top of the backswing. It was defined as the angle between the club shaft

compared to a horizontal line facing away from target at the top of the backswing. The mean value, for the 22 experienced golfers, was $170.8^{\circ} \pm 9.7^{\circ}$. This angle may, in fact, increase during “transition” which was defined by the authors as the time period between the lead hand’s deceleration at the end of the backswing, and its acceleration at the start of the downswing. For the purpose of the current study, the club shaft’s angle will be used as an indication of lead wrist radial deviation at the top of the backswing. In a study of eight LPGA players (Lemak et al., 1994), the rotation of the club was $366^{\circ} \pm 11^{\circ}$ (from a horizontal line facing target), or approximately 6° past horizontal at the top of the backswing.

Comparing lead and trail wrist angles (“cock”) at the top and at impact, Zheng et al. (2008) observed that mean lead wrist angle (approximately between the lead forearm and the club-shaft, and not measured as a three dimensional position which would separate the radial deviation from the extension) ranged, in their four groups of male professional to high handicapped golfers, between a mean of 92° to 103° at the top to between 156° to 165° at impact. Similarly, trail wrist mean angles (approximately between the trail forearm and the club) ranged between 74° and 79° at the top to between 118° and 124° at impact. Might the considerable asymmetry in radial deviation between the two wrists, especially at fast downswing speeds, create excessive force at the radial wrist?

In a study involving both female and male professional golfers (Zheng et al., 2008), the trail wrist, for the male participants, was always more cocked (smaller angle) than the lead wrist, at both the top of the backswing (a mean of 77° vs 94°) and at impact (a mean of 120° vs 163°), and there was no significant difference between females and males. The results from the two Zheng et al. studies indicate that the trail wrist had greater “cock” at the top and almost upto impact than the lead wrist. These angles might pull at the lead thumb (which lies within the trail

hand in the golf grip), placing greater strain on it, and resulting, perhaps, in injury to the radial side of the hand.

A study of the wrists' arc of motion (Cahalan et al., 1991) between an uninjured and an injured group of golfers revealed that the 25 mixed group of uninjured golfers in the study, using a driver club, had a mean arc of motion of $35^{\circ} \pm 34^{\circ}$ for the lead wrist and $103^{\circ} \pm 22^{\circ}$ for the trail wrist in the sagittal plane (flexion-extension). While these ranges of motion are within the expected functional ranges of wrist motion (Ryu, Cooney, Askew, An, & Chao, 1991), it is important to note that wrist movements in the golf swing take place with rapid changes of direction and at great speed, as the wrist is the most distal joint in the proximal-to-distal sequencing of the golf swing (Cheetham et al., 2008). These data may therefore be considered to be an indication that the trail wrist goes through a considerable range of flexion/extension motion during the swing.

In the frontal plane the total arc of motion (ulnar and radial deviation), was $36^{\circ} \pm 31^{\circ}$ in the lead wrist and $31^{\circ} \pm 15^{\circ}$ in the trail wrist (Cahalan et al., 1991). Additionally, the uninjured golfers' lead wrists, when using a driver, had a mean ulnar deviation at impact of $23^{\circ} \pm 16^{\circ}$. Although not related to the present study, the injured golfers had significantly greater lead wrist flexion/extension and radial/ulnar deviation arcs of motion, as well as greater ulnar deviation at impact. One explanation for this might be that the greater ranges of motion during their swings caused or exacerbated their injury.

EMG. The Farber et al. research of 2009 on forearm muscle EMG reported that lead FCU activity was extremely high for both their professional and amateur golfers during the forward swing using a driver club (mean values of 123.7% and 90% MMT respectively), and reduced, but remained in the marked range (terminology based on the Jobe et al., 1986

convention), during acceleration and early follow-through. The results may indicate that the FCU (which is both a wrist flexor and wrist adductor) was involved in maintaining wrist flexion, especially for the professionals who have higher FCU activity levels during the early downswing. Or the muscle's activity may signify an eccentric action to delay the rapid ulnar deviation which would be expected close to impact (Cahalan et al., 1991). Lead FCR activity was mostly in the moderate range from early downswing to early follow-through. It could thus be said that together the lead FCU and FCR may play a role in maintaining lead wrist flexion during the downswing.

Trail forearm FCU muscle activation of greater than 100% MMT was seen (Farber et al., 2009) for both the professionals and the amateurs (179% and 201.9% respectively) during the forward swing, and remained marked (82.8% and 127.9% respectively) during acceleration. The same pattern was seen for the FCR muscle, with a mean percent MMT of 119.8% and 117.6% in the forward swing and 73.9% and 105.9% during acceleration in professional and amateur golfers respectively. These results may indicate that, during the downswing, the trail-side FCU and FCR working synergistically were involved in eccentric activity to prevent a too-rapid wrist flexion, rather than either ulnar (FCU) or radial (FCR) deviation, which are their individual roles in wrist motion. That the trail-wrist remains in extension until close to impact is also known because Glazebrook et al. (1994) had observed a burst of flexor muscle EMG activity in all the golfers of their study, which reached 90.77% MVC during the "contact phase" close to ball impact.

Combined with trail-wrist extension during the downswing, the amateur golfers of the Farber et al. (2009) study also showed significantly greater trail forearm pronation than their professional cohorts during the downswing. Additionally the amateurs had higher trail ECRB

(wrist extensor) activity as well as the greater FCU and FCR (wrist flexor) activity during both the forward swing (early downswing) and acceleration (late downswing) phases. These latter results were not significant probably because of the high standard deviations typical of EMG analysis. Together these facts may indicate that amateurs might make a more delayed and forceful wrist flexion movement during or just after impact.

Lead wrist tissue injury mechanisms. Lead wrist ECU injury could arise from the observed swing positions of ulnar deviation (Cahalan et al., 1991) and flexion (Cahalan et al., 1991) combined with forearm supination (Teu et al., 2006). Similarly, there is evidence for de Quervain's tenosynovitis risk factors because of the considerable radial deviation of the lead wrist at the top of the backswing (Cahalan et al, 1991; Lemak et al., 1994; Zhang & Shen, 2014; Zheng et al., 2008), along with ulnar deviation and forearm rotation close to impact (Cahalan et al., 1991; Teu et al., 2006).

Trail wrist tissue injury mechanisms. FCU injury could be caused by the ulnar deviation and a large range of flexion and extension seen in the Cahalan et al., (1991) study. FCR injury becomes plausible given the excessive palmar flexion observed by Cahalan et al. especially when combined with the resistance experienced when golfers hit the ground before the ball or when making large divots. The large range of flexion and extension that the trail wrist moves through may also be a causative factor for carpal tunnel syndrome and dorsal impingement.

Hip

Kinematics. In the golf swing it is the pelvis that rotates about a planted leg, and therefore during the backswing the lead lower limb is said to move in external rotation (ER) and the trail hip in internal rotation (IR); the situation is reversed during the downswing, so that the lead hip experiences IR and the trail one undergoes ER. Additionally, when one lower limb is

closer to the body's midline during the swing, that leg is said to be abducted, while the one that is further from the midline is considered to be adducted (Neumann, 2010).

One group of researchers (Gulgin, Armstrong, & Gribble, 2009) who studied the hip joint's motion in 15 Division I collegiate female golfers found that, during the downswing, the lead hip's peak IR velocity ($-227.8^\circ/\text{s} \pm 96.6^\circ/\text{s}$) was greater than the trail hip's peak ER velocity ($-145.3^\circ/\text{s} \pm 68^\circ/\text{s}$), and occurred slightly later in the downswing, at 89.1% of downswing time compared to the lead hip's 85.2%. This group (Gulgin, Armstrong, & Gribble, 2010) also studied the ranges of motion of both hip joints during the back- and downswings for the same group of female golfers. In the backswing, lead hip ER range of motion was $29.7^\circ \pm 11.3^\circ$ and trail hip range of motion in IR was $8.9^\circ \pm 4.8^\circ$, while during the downswing, the ranges of motion were $34.8^\circ \pm 11.7^\circ$ of lead hip IR and $14.9^\circ \pm 9.6^\circ$ of trail hip ER. Connecting the findings of the two articles, it may be said that the trail hip moves through a smaller range of motion and achieves a lower maximum velocity, while the lead hip moves through a far greater range of motion and reaches a higher maximum velocity. These findings, albeit only for female golfers, together indicate the asymmetry of motion in this region of the body, which might be indicative of injury to either hip, as the lead side moves through a larger range of motion at greater velocity while the trail one is more restricted.

Kinematic variables for golfers using the 5-iron club were compared (Healy et al., 2011) between 15 high ball speed and 15 low ball speed male golfers and significantly different results were reported for the mid-backswing, top of backswing, mid-downswing and ball contact events. The results are incomplete as they were only published when significant differences existed between groups. The trail hip was significantly more abducted ($-17.0^\circ \pm 6.7^\circ$ vs $-4.0^\circ \pm 7.8^\circ$) at the top of the backswing, at mid-downswing ($-25.4^\circ \pm 5.8^\circ$ vs $-14.2^\circ \pm 7.5^\circ$), and at impact

($-27.1^\circ \pm 5.3^\circ$ vs $-18.5^\circ \pm 6.0^\circ$) for the faster ball speed group. The trail hip at mid-downswing, was flexed to a significantly less extent for the high ball speed group ($18.9^\circ \pm 9.2^\circ$ vs $30.2^\circ \pm 13.9^\circ$) and moved into extension at a greater velocity ($-443.2^\circ/\text{s} \pm 115.2^\circ/\text{s}$ vs $-290.4^\circ/\text{s} \pm 106.7^\circ/\text{s}$), to once again have a significantly lower flexion angle at impact ($2.3^\circ \pm 9.4^\circ$ vs $14.5^\circ \pm 13.9^\circ$). Thus the trail hip for the high ball speed group had greater downswing abduction and extension. These two movements together place the hip in a close-packed position, which may lead to injuries involving greater compressive loading.

In the same study (Healy et al., 2011), the lead hip was significantly less externally rotated for the high ball speed group at the top of the backswing ($-10.0^\circ \pm 7.3^\circ$ vs $-19.3^\circ \pm 9.4^\circ$), and was extended at a significantly faster rate during mid downswing ($-324.2^\circ/\text{s} \pm 107.6^\circ/\text{s}$ vs $-218.4^\circ/\text{s} \pm 91.4^\circ/\text{s}$) than for the low ball speed group. The lead hip therefore did not rotate as much during the backswings of the high ball speed group, but then extended further during the downswing. Could the greater extension lead to more lead hip compression, especially as pressure shifts onto the forward leg during the downswing?

Kinetics. An important consideration for all lower limb injuries is that as the center of pressure shifts towards the lead side during the downswing (Choi, Kang & Mun, 2016), there is greater loading on all lead-side lower limb joints. Foxworth et al. (2013) looked at peak hip joint internal (generated by muscles) torque differences during the entire swing, for ten young and ten senior male golfers using their driver clubs. The results were presented in Nm normalized by body weight and height. The authors found only one significant difference between the young (25.1 years ± 3.1 years) and senior (56.9 years ± 4.7 years) golfers' torque production levels, which was in peak external rotator torque (mainly generated by the gluteus maximus muscle) when averaged across both lower limbs (mean of 4.39 vs 3.22 for the two groups respectively).

At the same time, the trail limb peak external rotator torque (2.38 ± 0.84) was greater than peak lead limb external rotator torque (1.43 ± 0.49) when averaged across both groups. Another large difference, although not significant, was between combined (for both groups) leg extensor torques, which was far higher (10.64 ± 1.96 vs 5.28 ± 1.27) in the trail, than the lead, leg. Thus the younger golfers of this study were able to rotate their lead hips during the backswing and their trail hips during the downswing more forcefully than the senior golfers, while both groups had greater trail hip rotation and extension compared to the lead hip, probably during the downswing.

EMG. Useful information can be extrapolated from an EMG study of lower limb muscles which compared five low- and five high-handicap golfers (Marta et al., 2016) using a 7-iron club. Muscle activation was measured as a percent of EMG_{MAX} , (similar to MMT). Using the Jobe et al. (1986) method of making comparisons (muscle activity is “marked” if 60% of MMT or greater), reveals that low handicap (0.7 ± 2.2) golfers had marked activity levels of several muscles during the forward swing compared to high handicap (25.5 ± 3.1) golfers. Both groups showed marked trail leg biceps femoris muscle (hip extensor, knee flexor) activity ($75\% \pm 28.1\%$ vs $94\% \pm 11.3\%$) during the forward swing. Muscles for which only the low handicap golfers showed marked forward swing activity were the trail gluteus maximus (hip lateral rotator and extensor) with $82\% \pm 41.8\%$, trail semitendinosus (hip extensor, knee flexor) with $58\% \pm 12.1\%$, and both sides of the trail gastrocnemius (knee flexor, ankle plantar flexor) muscles with $68\% \pm 11.9\%$ each. It could thus be stated that low handicap golfers had more forceful trail hip rotation as well as knee and ankle flexion during the forward swing, while both groups had similar trail hip extension.

One EMG analysis (Bechler, Jobe, Pink, Perry & Ruwe, 1995), of 13 male and 3

female golfers with handicaps less than 5, found marked (Jobe et al., 1986) activity of over 60% MMT during the forward swing in lead side muscles including the adductor magnus ($63\% \pm 22\%$) and the biceps femoris ($60\% \pm 43\%$) which muscles act as hip adductor and extensor and hip extensor, respectively. The lead biceps femoris also sustained marked activity during the acceleration and early follow-through phases ($83\% \pm 58\%$ and $79\% \pm 67\%$ respectively). The lead hip could thus be said to be adducting and extending during the downswing.

Meanwhile, during the forward swing the trail side upper and lower gluteus maximus ($100\% \pm 55\%$ and $98\% \pm 43\%$), gluteus medius ($74\% \pm 36\%$), biceps femoris ($78\% \pm 35\%$) and semimembranosus ($67\% \pm 37\%$) muscles all showed marked activity. These muscles together externally rotate, abduct and extend the hip. Putting together the evidence from all the hip-related EMG information, the lead hip was adducted and forcefully extended during the downswing, while the trail hip was actively externally rotated and extended during the downswing.

Lead hip tissue injury mechanisms. Although there is limited information on laterality of hip injury in golf epidemiological literature, given the expected causative mechanisms of OA, it may be the lead hip that is more at risk for this injury. The lead hip is seen to have rapid downswing extension, especially in more skilled golfers (Healy et al., 2011), and is internally rotated during the downswing (Gulgin et al., 2010), both of which are purported mechanisms for hip OA. Moreover, when pressure shifts to the lead side (Choi et al., 2016), greater loads are placed on all lead lower limb joints.

Trail hip tissue injury mechanisms. The trail hip is subjected to external rotator torque (Foxworth et al., 2013), along with leg abduction from the top of the backswing up to impact (Healy et al., 2011), while the spine is flexed forward (Lindsay et al., 2002), and these conditions together represent a causative mechanism for trochanteric bursitis. Additionally, labral tears can

occur when there is hip external rotation (as observed by Choi et al., 2016) combined with axial loading, so that such injury may occur in the trail hip during the downswing. This latter finding is supported by a research article which found the prevalence of labral tears to be greater in the trail hips of golfers. According to the authors, as a result of years of practicing golf as adolescents, there is a change in the morphology of their hip joints, and the resulting strain may lead to labral tearing (Dickenson et al., 2016).

Knee.

The knee joint is mainly a hinge joint with a good range of flexion and extension, and some ability to rotate. However, during the golf swing, both knees move through a large range of medial and lateral rotation, and also experience abduction-adduction as well as anteroposterior translations of the medial and lateral condyles of the tibia (Murakami et al., 2016). This fairly large range of motion is facilitated by movements at the hip and ankle joints of both lead- and trail-side lower limbs. In fact, the lead knee attains a position at the top and during early downswing which has been seen in many sports and is termed a “dynamic valgus” position (Hewett et al., 2005), involving not only knee flexion but also femoral adduction, tibial abduction and ankle eversion.

Kinematics and kinetics of flexion/extension. Many research studies have looked at the range of flexion and extension the knee joints move through during the golf swing. One study (Egret, Vincent, Weber, Dujardin, & Chollet, 2003) assessed knee movement in seven male golfers with an age range of 17 to 34 years and handicaps ranging from 0 to 3, while using their driver, 5-iron and pitching wedge clubs. Comparing address, top of backswing and impact for the driver club (the other clubs had similar movement patterns), the lead knee’s flexion changed from $17.7^{\circ} \pm 5.5^{\circ}$ at address to $37.6^{\circ} \pm 9.1^{\circ}$ at the top, to $16.5^{\circ} \pm 7.9^{\circ}$ at impact. The trail knee’s angles were

$17.9^\circ \pm 6.8^\circ$, $22.8^\circ \pm 2.9^\circ$ and $27.0^\circ \pm 10.8^\circ$, respectively. In a more recent study by the same group (Egret, Nicolle, Dujardin, Weber, & Chollet, 2006), the researchers compared, once again with the driver club, the lead knee's flexion at the top of the backswing between seven male and five female golfers with mean handicaps of 6.6 and 6.1 respectively. Males had significantly greater top of backswing knee flexion ($35.3^\circ \pm 4.9^\circ$ vs $16.5^\circ \pm 5.6^\circ$).

Another study (Murakami et al., 2016), which did not report the golf club used by their participants, used radiographic image-matching to assess the movements of the femur relative to the tibia, and found that, during the golf swing, the lead knee has $18.0^\circ \pm 12^\circ$ flexion at address, $32.7^\circ \pm 7.7^\circ$ at the top and $16.5^\circ \pm 9.4^\circ$ at the end of the follow-through. The trail knee flexion angles were $16.7^\circ \pm 9.2^\circ$, $24.1^\circ \pm 7.6^\circ$ and $19.1^\circ \pm 6.5^\circ$ for the same three phases. These results are similar to those of the Egret et al. (2003) for address and the top of the backswing.

A more recent study (Purevsuren et al., 2017) which looked exclusively at the lead knee of male golfers with handicaps ranging from one to five, found that maximum knee flexion of 43.7° with the driver club, took place during the downswing, which is slightly higher than the other two studies, but the participants may have had different golf swing technique training as the study was conducted on a Korean population. Additionally, a study (Pfeiffer, Zhang, & Milner, 2014) which measured flexion in the knee joints at the instant of peak extensor moment found that it was $-52.5^\circ \pm 8.2^\circ$ in the lead knee and $-46.7^\circ \pm 6.9^\circ$ in the trail knee. This indicates a very high amount of knee flexion compared to all previously mentioned studies; however the population of this study was comprised of healthy, older male golfers, with a mean age of 57.7 years (± 8.5) and a range of 45 to 73 years. Interestingly, this study compared several knee joint kinematics and kinetics for both knees, and found that the knee flexion angle at peak knee

extensor moment was significantly higher in both knees compared to what it was during walking, so that the golf swing would place greater loads on the knees than walking.

Finally, Chu et al., (2010) found that lead knee flexion angle for 308 male and female golfers continued to reduce from the top of the backswing until impact, with no increase in that angle shown during the early downswing. This might be the difference between amateur golfers (the mean handicap for their study was 8.4 ± 8.4) and professional golfers who try to increase the GRF in the lead foot by an early shift of the body towards target (Lynn, Noffal, Wu & Vandervoort, 2012), sometimes involving more flexion of the lead knee.

When put together, the above studies indicate that the lead knee begins the backswing in some amount of flexion, which increases considerably during the backswing, and it must then extend to return to, by impact, a similar amount of flexion to that seen at address. It could thus be said that the lead knee must extend rapidly while moving through a considerable range of movement, to arrive at its observed position of impact. This can be seen from the results of one study which looked at knee extension velocity only in the lead knee (Healy et al., 2011) and found that in their group of golfers with high ball speed, it went from $-164.4^\circ/s \pm 61.5^\circ/s$ in the early downswing phase to $-238^\circ/s \pm 75.9^\circ/s$ by mid downswing. For the low ball speed comparator group of that study, the lead knee extension velocities measured during the same events were significantly lower: $-52.6^\circ/s \pm 68.7^\circ/s$ and $-177.3^\circ/s \pm 46.7^\circ/s$ respectively.

The lead knee must extend rapidly, as it has considerable flexion at the instant when it has maximal extensor moment on it (Pfeiffer et al., 2014). During that time, the quadriceps femoris group of muscles which act as knee extensors, must contract strongly, adding to the amount of compression experienced by the lead knee. This may be seen from the peak extensor

moment, which was $.069 \pm .015$ Nm/(body weight x height) in the lead knee compared to only $.033 \pm .014$ Nm/(body weight x height) in the trail knee (Pfeiffer et al., 2014).

The trail knee, conversely, starts with some flexion at address ($17.9^\circ \pm 6.8^\circ$) while using a driver club (Egret et al., 2003), and continues to flex to the top of the backswing ($22.8^\circ \pm 2.9^\circ$) and then into the downswing ($27.7^\circ \pm 10.8^\circ$ at impact), so that it does not have a rapid change in direction of movement during the downswing. A similar pattern was seen for the 5-iron and pitching wedge clubs in the seven male participants of the study. Moreover, although it also undergoes considerable flexion in the downswing, perhaps the loads on it are reduced as pressure shifts to the target side. Additionally, the trail foot almost completely leaves the ground during the downswing, which might also serve to reduce GRF loads transmitted to the trail knee joint during that phase. Thus it may be seen that the lead knee is probably more prone to injuries from excessive backswing flexion followed by rapid downswing extension, especially because considerable pressure is also being transferred to that side (Lynn & Noffal, 2010).

Kinematics and kinetics of abduction/adduction. Abduction and adduction moments have also been studied in both knees. One study (Lynn & Noffal, 2010) assessed the lead knee only, and measured only adduction (varus), and abduction (valgus) moments. Peak adduction moment, reached in the follow-through, was 0.63 Nm/kg \pm 0.23 Nm/kg, which was a significantly higher load than sustained during gait or stair ascent. Peak abduction moment, reached prior to impact, was -0.70 Nm/kg \pm 0.12 Nm/kg. Interestingly, when the foot was placed in an externally rotated or “toe out” position, a commonly used instructional technique aimed to reduce loads at the knee, adduction moment reduced significantly, but abduction moment (-0.80 Nm/kg \pm 0.9 Nm/kg) increased to an almost significant extent ($p = .07$).

Peak abduction moment was also measured by Pfeiffer et al. (2014) in their study of healthy older male golfers. It was considerably greater in the lead knee than the trail knee: $-.043 \pm .010$ (Nm/ body weight x height) compared to $-.027 \pm .008$ (body weight x height). Finally, one study (Gatt et al., 1998) measured both peak abduction and adduction moments, for both knees, in their population of 13 male golfers with handicaps ranging from 4 to 18 with a mean of 11.2, and a mean age of 35 years (± 14.2). Peak lead and trail knee abduction moment was $63.7 \text{ Nm} \pm 24.5 \text{ Nm}$, and $38.8 \text{ Nm} \pm 17.4 \text{ Nm}$ respectively. Peak lead and trail knee adduction moment was $24.4 \text{ Nm} \pm 11.0 \text{ Nm}$ and $52.6 \text{ Nm} \pm 16.1 \text{ Nm}$, respectively.

In order to synthesize the common results for peak abduction/adduction moments from the three studies, rudimentary calculations were made (by multiplying the published results by mean mass and height as required) to remove the effect of normalization by body mass (Lynn & Noffal, 2010) and by body weight and height (Pfeiffer et al, 2014), so that the two results could be compared with the raw moments presented in the third (Gatt et al., 1998) study. The mean peak lead knee abduction moment was 63.7 Nm according to Gatt et al., 50.5 Nm as calculated from the Lynn and Noffal results, and 7.4 Nm for the older golfers of the Pfeiffer et al. study. The main information to be gleaned from the three studies is that despite the supposedly lower peak abduction moment seen in the Pfeiffer et al. study, the authors found the load on the knee joint to be significantly greater than for walking as well as for stair ascent and descent.

Interestingly, a cadaver-based study (Ohori et al., 2017) applied 5 Nm of torque on porcine cadaveric knees and found that there was far greater force on the ACL at 30° knee flexion than at 60° flexion, and that the force was slightly greater under valgus (abduction) than varus (adduction) torque. Although the study used specimens from pigs, this information

indicated that 30° flexion with a valgus load of only 5 Nm, may strain the ACL more than greater knee flexion or a 5 Nm varus load.

Kinematics and kinetics of internal/external rotation. Internal and external rotation and rotation moments acting at the knee are important factors related to injury during the golf swing. Importantly, for those with replaced knees, the amount of average acceptable axial rotation is 22° at a mean low knee flexion angle of 22° (Murakami et al., 2016). Pfeiffer et al. (2014) found peak lead knee external rotation to be $14.8^\circ \pm 5.3^\circ$ which moved to a peak of internal rotation of $-19.5^\circ \pm 6.7^\circ$, indicating approximately 35° range of rotation in the lead knee, for the entire swing from address to finish. The trail knee had peak internal rotation of $-14.7^\circ \pm 5.9^\circ$ which then went to $9.7^\circ \pm 6.0^\circ$ of external rotation, resulting in a range of approximately 25°, which is considerably less than that seen in the lead knee.

In another research paper (Murakami et al., 2016), axial rotation from the top of the backswing to the end of the follow-through in five healthy males, went from $-7.5^\circ \pm 6.6^\circ$ to $10.4^\circ \pm 5.1^\circ$ in the lead knee (total range approximately 18°) and from $9.8^\circ \pm 4.8^\circ$, to $-16.0^\circ \pm 4.9^\circ$ respectively in the trail knee (total range approximately 26°). The difference between the Pfeiffer et al. (2014) and the Murakami et al. studies may be the result of age differences between the older golfers of the first study and the relatively younger golfers (mean age 34 years, range 32-36 years) of the second, with older golfers having a greater range of movement. The differences may be indicative of a generational shift in instruction-style.

Finally, Purevsuren et al., in their recent study of ten Korean one to five handicap young (age 23.2 ± 1.6 years) male golfers, stated that the lead knee started the downswing with 7.9° of

external rotation and moved to 42.4° of internal rotation in the follow-through. This large range of total lead knee motion of about 50°, may once again be a result of different swing techniques in different regions of the world.

Only one study (Gatt et al., 1998) reported internal and external rotation knee moments for both knees. Lead knee external rotation torque was $27.7 \text{ Nm} \pm 9.3 \text{ Nm}$ and internal rotation torque was $16.1 \text{ Nm} \pm 4.8 \text{ Nm}$. Trail knee external and internal rotation torques were similar to one another, and were $19.1 \text{ Nm} \pm 5.5 \text{ Nm}$ and $19.6 \text{ Nm} \pm 8.1 \text{ Nm}$ respectively. Thus it may be said that lead knee external rotation torque is the highest one, and peaks perhaps at the top of the backswing or during the early downswing.

Interestingly, the study of Korean golfers (Purevsuren et al., 2017) showed that forces on, and strain to, the lead ACL was maximum at, or just past impact. The authors ascribed this maximal strain to the tibia being in approximately 8° of external rotation prior to impact and then going into 40° of internal rotation during the follow-through. It can thus be stated that both knees undergo a large range of rotation, and have considerable torsion applied to them.

Kinematics and kinetics of anteroposterior translation. Anterior translation of the tibia has been implicated in injury to tissue such as the ACL. Moreover, Murakami et al. (2016) looked at anteroposterior femoral translation in their five recreational golfers, during the entire swing. They reported $4.6 \text{ mm} \pm 9.2 \text{ mm}$ of posterior femoral translation in the lead knee (indicative of forward tibial translation), and $4.1 \text{ mm} \pm 3.6 \text{ mm}$ of anterior femoral translation in the trail knee. Moreover, Gatt et al. (1998) had found that the lead knee experienced $295.6 \text{ N} \pm 91.9 \text{ N}$ peak anterior force on the tibia, and only $-2.8 \text{ N} \pm 19 \text{ N}$ peak posterior force during the golf swing. The trail knee in their study had similar peak anterior and posterior force values which were approximately 75 N each. The study of porcine knees (Ohuri et al., 2017) had

indicated that there is greater anterior tibial translation under varus than valgus torque, at both 30° and 60° of knee flexion. One study assessed forces on the knee of 26 healthy males, in different degrees of knee flexion and extension (Smidt, 1973). Results showed that torque produced by concentrically contracting knee extensors increased from 0° to approximately 60° of knee flexion, and increasing knee flexion also resulted in anterior tibial translation, which was maximal when the knee was in 15° of flexion, remaining fairly high up to 30° of flexion.

To summarize, the lead tibia translates anteriorly during the golf swing and has greater translation in the 15° to 30° knee flexion range. The trail tibia translates posteriorly, and the trail knee experiences smaller peak forces than the lead knee. Murakami et al. (2016) noted that a mean of 4 mm AP translation at an average of 22° of knee flexion is the accepted range for those with replaced knees.

Kinetics of compression. Gatt et al. in their 1998 study which calculated moments and extrapolated forces acting at both knees, found that average peak compressive loads were 756 N (approximately 100% body weight) on the lead knee, and 540 N (72% BW) on the trail knee. The Purevsuren et al. (2017) study of ten male Korean professional golfers looked at the peak compressive forces that the lead knee was exposed to: 375.7% body weight reached during the follow-through. This higher value may be more realistic as this latter group of researchers modeled the main tissues of the knee joint while the former extrapolated force from inverse dynamics calculations.

EMG. According to Marta et al. (2016), several lead leg muscles were markedly active (Jobe et al., 1986) for the low handicap golfers during the forward swing. They include (all described as a percent of EMG_{MAX}), the vastus medialis (66 ± 17.7) muscle (knee extensor), while the vastus lateralis (47 ± 16.5) and rectus femoris (45 ± 9.2) muscles (both knee

extensors), showed moderate activity, together indicating considerable quadriceps femoris (knee extensor group) activity in the lead leg during the forward swing. Finally, the lead peroneus (fibularis) longus muscle (ankle evertor and plantar flexor) showed marked activity across several swing phases from the forward swing to the late follow-through (73 ± 30 , 61 ± 21.6 , 55 ± 13.4 and 54 ± 26.2 respectively), perhaps indicating a pressure shift to the lead side as the evertors work eccentrically against resistance. Additionally, the low handicap group had active trail side biceps femoris and semitendinosus muscles (knee flexors, part of the hamstring group) during the forward swing (details in hip section). It may thus be said that the lead knee is extended during the downswing, while the trail knee is flexed, and the trail ankle is plantar flexed.

Knee tissue injury mechanisms. It is mainly the lead knee which is injured in golf, and some biomechanics studies, for this reason, have looked exclusively at that knee.

The ACL (especially the lead-side one) may be strained through forces and positions which include abduction moment prior to impact, adduction after impact, compressive force, internal tibial rotation and knee extension (Purevsuren et al., 2017). These suspected mechanisms of ACL injury as enumerated in the Baker et al., (2017) review have all been observed to take place in the lead knee, and occur during the downswing: rapid extension (Healy et al., 2011), internal tibial rotation (Murakami et al, 2016; Pfeiffer et al., 2014), strong quadriceps muscle activation (Marta et al., 2016) causing compression of the knee joint, and large external GRF (Chu et al., 2010). Other known causes of ACL injury are abduction and adduction, and they were observed in the Lynn and Noffal (2010) study, pre- and post-impact respectively.

The medial collateral ligament of the lead knee may be strained when the knee is in a

valgus position as observed by Lynn and Noffal (2010). Additionally, although peak lead knee adduction moment was lower than peak abduction moment, the former loads the smaller medial compartment of the knee, contributing to medial compartment OA and MCL strain (D'Lima, Fregly, Patil, Steklov, & Colwell Jr, 2012; Gatt et al., 1998).

The medial meniscus of the lead knee is more likely to be injured when the knee is twisted and the tibia is adducted. In such a position, as seen during the golf follow-through, for instance, greater loads are sustained in this region of the knee (Lynn & Noffal, 2010). The lateral meniscus of the trail knee could be susceptible to injury during twisting too, which has been observed during the downswing (Pfeiffer et al., 2014).

Female golfers are known to be more prone to OA in the PF joint than male golfers, even though their knee flexion angles at the top of the backswing were observed to be smaller (Egret et al., 2006). One reason could be because of the greater Q angle (the angle of the axis of the femur from vertical) in females, which contributes to their having greater dynamic valgus angles at the knees.

The medial side of the lead knee in golfers of both sexes, could be susceptible to OA during the adduction seen in the follow-through (Lynn & Noffal, 2010), while the lateral side of the trail knee could be similarly susceptible as it may be subjected to excessive abduction, and thus lateral compartment loading, during the downswing (Gatt et al., 1998). Although there is far lower abduction moment in the trail knee than the lead one (Pfeiffer et al. 2014), because the downswing takes places with an aggressive activation of trail side lower limb muscles such as the knee flexors, ankle plantar flexors and hip external rotators (Marta et al., 2016), there may be an increased risk for lateral compartment OA. In general, excessive rotation (Murakami et al.,

2016, Pfeiffer et al., 2014), as seen in both knees, along with high compressive forces (Gatt et al., 1998) are known to be important causative factors for knee TF OA.

Ribs.

EMG. The serratus anterior (SA) muscle serves to “directly approximate the scapula to the thorax” (Phadke, Camargo, & Ludewig, 2009, p. 4), and is a scapular protractor which has often been implicated in rib stress fractures, especially in golf. A study (Kao, Pink, Jobe, & Perry, 1995) on the activity of scapular muscles during the golf swing of competitive male golfers revealed that the trail-side SA has high activity during the forward swing as well as for the remainder of the swing, to facilitate downswing scapular protraction. Activity was measured separately for the upper and lower parts of the SA muscle, and, as a percent of maximum voluntary isometric contraction (MVIC). The trail upper SA had marked or almost marked (greater than 60% MVIC, according to the convention suggested by Jobe et al., 1986) activity during the forward and acceleration phases of the swing ($58\% \pm 39\%$, and $69\% \pm 29\%$ respectively), while the trail lower SA had moderate or almost moderate activity (between 30% and 60% MVIC) during the forward, acceleration and deceleration phases of the downswing ($29\% \pm 17\%$, $51\% \pm 33\%$ and $47\% \pm 25\%$ respectively). Altogether there was considerable trail arm SA activity during the forward swing (early downswing) to deceleration (mid follow-through) phases.

Conversely (Kao et al., 1995), the lead-side SA muscle had constant activity throughout all swing phases. The upper and lower SA individually never reached a mean MVIC activity level greater than 31%, during any phase of the swing. After combining lead arm upper and lower SA activity, the persistent SA activity would probably be in the moderate range, so

that across many repetitions, the ribs might be injured, when being subjected to continual muscle force.

Lead-side rib stress fractures. It is mainly the lead side ribs which are injured, typically in a beginner golfer. There are two explanations that are usually proffered for lead side rib fractures in golf, one of which is the fatigue levels of the constantly active lead SA muscle (Kao et al.), which would result in forces being placed on the ribs rather than the muscle. The other explanation is that when contact of the club is made with the ground rather than the ball, as beginners might do, forces would be transmitted up the club and to the SA, which would then create a pull on the ribs (Bugbee, 2010).

A third explanation is also possible, based on the “rib cage compression theory”. This theory states that when the arms are flexed, as during the forward movement of the rowing stroke, the SA is maximally activated by the pull of the oars, while the scapular retractors (mainly the rhomboids) are also active. Together, those muscles can create forces that serve to compress the rib cage. This is a similar position to that seen in the golf downswing, where it is known that lead SA activity is constant (Kao et al., 1995) and lead rhomboid activity is marked or almost marked at $68\% \pm 27\%$ during the forward swing and $57\% \pm 46\%$ during acceleration (Kao et al., 1995). Shoulder girdle muscle activity during the downswing could thus be a likely mechanism for lead-side rib stress fractures.

Trail-side rib stress fractures. The trail-side SA has considerable activity during the forward swing and until early follow-through, as it protracts the trail scapula (Kao, Pink, Jobe, & Perry, 1995). Additionally, less skilled golfers are known to swing “over-the-top” (OTT; Wrobel, Marclay & Najafi, 2012), starting the downswing with their trail-side upper body and shoulder

girdle region. The OTT movement may add additional pressure to the trail side rib cage and be a reason why beginner golfers suffer trail-side rib stress fractures.

When this information is combined with the rationale of lack of downswing rotation of the body as described in the case study of an experienced golfer who suffered trail rib injury (Read, 1994), it may be surmised that there is incorrect sequencing of upper body before lower body - the OTT movement - during the downswing. This may place greater strain on the trail ribs as it could involve greater scapular protraction than otherwise.

Discussion

This three-part literature review involved a search for golf swing-specific overuse injuries at each major body joint; the purported mechanisms of such injuries; and the biomechanically tested evidence of the existence of the positions, movements and loads attributed to them. With respect to epidemiological findings, most studies were undertaken in the 1900s, and only four took place after the year 2000. Of those, one research group took into consideration most body segments, but in females only (Fradkin et al. 2005); one looked at the wrist joint (Hawkes et al., 2013); two assessed shoulder injury (Hovis et al., 2002; Lee et al., 2017), and only one group of researchers performed a retrospective, and then a follow-up analysis, of most injuries in a mixed group of golfers (McHardy et al. 2006; McHardy et al. 2007). There is thus a paucity of research on the typical tissues injured from overuse during the golf swing; laterality is not always mentioned; and most such research is not recent enough to be adequately relevant to the more current versions of swing being used by golfers in the second decade of the 21st century.

Very few articles have combined an epidemiological analysis of golf-related injury with an assessment of the likely causes of the injuries they have observed. That role has, in fact, been fulfilled by review articles which locate injury prevalence and incidence and then conjecture

upon the likely causative factors of such injury. Many causative factors reported in this article were sourced from other movements in which the same tissue-level damage takes place.

A few exceptions do exist, as some epidemiological studies did attempt to understand the mechanical causes of the injuries revealed during their research. Sugaya et al. (1998) correlated epidemiological findings with radiographs to determine factors contributing to injury of the lumbar region of the spine. Hovis et al. (2002) looked at the positions attained by the shoulders during the swing and indicated, for instance, that any elevation of 30° above horizontal might cause impingement. Hawkes et al. (2013), looking only for the prevalence and variety of wrist injuries in professional golfers, presented, in their article, details of the type of injury, its laterality and possible causative factors. Finally, a very recent epidemiological assessment of shoulder pain in 77 Korean amateur golfers (Lee et al., 2017) combined surveys with ultrasound, and were thus able to identify the type of tissue injured, the laterality of the injury, and were then able to opine upon possible causative mechanisms.

Even when injury prevalence has been described as having specific causes, the information may be limited in its applicability to the golf swing, which has a very unique and asymmetric movement pattern compared to that which most such researchers typically assess. The golf swing, for instance, has a large rotary component, unique only to sport and not to activities of daily living. It also has a closed kinetic chain effect in both upper and lower limbs. Such an effect occurs when the distal segment of a limb is fixed and movement at one joint produces movement at all other joints in the “chain”. Despite the closed kinetic chain effects at both limbs, the golf swing must produce a ground-up, open chain, kinematic sequence (Cheetham et al., 2008), involving a rotation of the torso being preceded by that of the pelvis. All this must be achieved while ensuring that the club reaches a precise location on the ground,

subsequent to lateral, rotary and vertical movements of the body during the downswing.

Moreover all movements have to be completed within the approximately 1/3rd of a second that the golf downswing lasts (Zheng et al., 2008).

Few researchers have looked at the swings of healthy golfers for the specific purpose of studying movements and loads which may be considered causative factors for injury. In fact, most biomechanical research has focused on the factors which are correlated with the production of greater clubhead, and thus ball, speed. Moreover, most studies have had small sample sizes so that many factors do not show significance, and effect size has been reported in only one research article (Lynn & Noffal, 2010), which reported data that was not relevant to the present review. Despite the lack of detailed injury-specific information, this review article has presented evidence for positions, movements and loads which may be causative factors for tissue damage at various joints. Another constraint of existing biomechanics research is that comparisons are typically made between skilled and unskilled golfers, males and females, or younger and older players, which does not aid the understanding of injury mechanisms. Very few studies compare golf swing related injury mechanisms with other activities of daily living or sports maneuvers, to indicate the relative magnitudes of injurious loads on a joint.

The results of this study, when synthesized and analyzed, indicate that there are some typical movements which cause injury at the various joints. The lumbar spine is most vulnerable to injury when lateral bending and axial rotation are combined with compression, and the movement takes place at great speed, such as during the downswing (Lee & Lee, 2017). Thoracic spine injury is most likely from compression, and typically a result of the physiological change associated with aging. The cervical part of the spine is probably injured because its movements are dissociated with those of the rest of the spine (Part 3 of the Results section). Shoulder injury

is most commonly seen in the lead shoulder, but can be sustained in both shoulders from excessive vertical elevation and adduction at one end; and considerable abduction, extension and external rotation at the other end, of the swing's total range of motion: from the top of the backswing to the end of the follow-through (Part 3 of the Results section). Lead elbow lateral epicondylitis is typically a result of excessive elbow extension combined with wrist flexion and forearm supination, while trail elbow medial epicondylitis is related to forearm pronation with wrist (and perhaps elbow) flexion. At the wrist, many different injuries can take place, which either involve excessive radial deviation of the lead wrist at the top or its ulnar deviation at impact, while the trail wrist may be injured during excessive flexion and extension (Part 3 of the Results section).

With respect to the lower limb, the hip is susceptible to overuse injuries caused by rotation – both internal and external (Lee & Lee, 2017) – as well as extension. Either knee can be injured from a combination of rotary, adduction and abduction moments, and compressive force (Lee & Lee, 2017). The lead knee is most frequently injured, and this happens when there is considerable axial rotation combined with pre-impact abduction (dynamic valgus) and post-impact adduction of the tibia. During the late downswing, the lead knee extends rapidly when in lower angles of flexion and the quadriceps femoris group of muscles acts forcefully to extend it. Anterior tibial translation is also a risk factor for some types of knee damage. The trail knee can also be injured in similar positions. Its lateral compartment can be strained as the tibia is abducted during the downswing, especially while the trail foot remains flat on the ground. Finally, the lead side ribs may be injured, possibly from rib cage compression during the downswing, when the shoulder protractors and internal rotators are active at the same time that the rhomboids work to retract the lead scapula.

Some golf-related articles have made suggestions for changing swing positions and movements in order to facilitate the reduction of the loads on individual body segments. Thériault and Lachance (1998) recommend a more upright spinal posture, a reduction in the shoulder range of motion and trunk motion, as well as a reduction in swing speed during spinal rotation to reduce the likelihood of dorsolumbar injury. This is a recommendation echoed by Gluck et al. (2008) in their review article on the lumbar spine and low back pain in golfers. They also suggested a more upright finish than the reverse-C position that some golfers have at the end of their swings.

One group of researchers (Lindsay et al., 2002), reported that when using an iron club, specifically a 7-iron versus a driver, golfers had significantly greater lead-side lateral flexion during the backswing, followed by significantly greater trail-side lateral flexion velocity during the downswing. One research project showed that golfers with LBP had greater lead- and -trail-side lateral flexion during the back- and down-swings respectively, while another was able to eliminate LBP in a professional golfer by reducing both trunk forward flexion and lateral flexion during the downswing (Lindsay & Vandervoort, 2014). If lead-side lateral flexion during the backswing were to be reduced, it might slow down trail side lateral flexion velocity as the trail side would have a smaller range to side-bend through, and may thus require less speed to complete the motion, during the approximately 1/3rd second duration of the downswing. Additionally, the X-Factor, which differentiates the quantity of lumbar versus thoracic twist during the backswing and early downswing (Meister et al., 2011; Okuda et al., 2010), as well as the non-coupled rotations of the thoracic and cervical spines (Horan & Kavanagh, 2012) are problematic. A safer swing might thus be one that keeps the cervical, thoracic and lumbar regions of the spine aligned, in the frontal plane, from address to impact.

Two chiropractors who discussed the etiology and prevention of back pain (Seaman & Bulbulian, 2009) opined that pelvic and spinal rotators are not the most important in the production of power nor is the length of the club's arc during the backswing. They recommended a well-timed weight transfer towards target before the backswing ends, which would pre-stretch the trail side pectoralis major and latissimus dorsi muscles for greater downswing elastic energy. They also stated that, in their "back-friendly" swing, the lead arm should only move between 90° to 135° from its position at address, and not the typically seen movement of almost 180°. However, the authors did not explain how maximum possible club speed might be developed with only weight shift, while eliminating the rotary component of the swing, which uses the powerful abdominal muscles and has been linked to greater club speed in many studies.

Several recommendations have been made to reduce shoulder pain incidence. Cann, Vandervoort and Lindsay (2005) opined that a shorter backswing in which the elbows remain close to the trunk and a finish which keeps the hands low would benefit the shoulder, especially because the flatter backswing generated would reduce the chance of impingement of the lead-side RC muscles. McHardy and Pollard (2005) considered that a lack of trunk rotation might induce the much smaller shoulder rotators into becoming more activated, which may result in dysfunction, specifically instability, in the shoulders. Thériault and Lachance (1998) stated that a reduction of angular shoulder displacement during the backswing along with less arm motion during the follow-through, may reduce the likelihood of shoulder injury.

Many suggestions have been proffered regarding positions and movements that might reduce the potential for elbow injury. One is a recommendation (Farber et al., 2009) to reduce the scope for medial epicondylitis, through a decrease in trail-side PT activation during the

downswing. Such an attenuation in muscle activity would be expected to reduce forearm pronation; however no suggestion was made as to how that might be accomplished.

Another recommendation made was to avoid an excessively straight lead arm, which can create tension in the elbow and may therefore be a causative factor for lateral epicondylitis (Wadsworth, 2007). The Thériault and Lachance overview article (1998) grouped elbow, wrist and hand movements together to suggest some injury-preventive measures. The authors recommended a reduction of grip strength and a loosening of the elbows, along with a lessening of excessive wrist motion during the swing. They further opined that maintaining a good balance during weight shift would also reduce the likelihood of injury to the elbows and hands. Finally, Cann et al. (2005) considered that a “stronger” trail-hand grip (trail hand rotated more under the club’s grip) would prevent it from over-compressing the lead thumb, thus reducing the compressive loads which might cause OA in that thumb.

Limited recommendations exist for the decrease of lower limb injury potential, perhaps as they occur less frequently in golf. One useful suggestion for the lead knee might be to reduce its dynamic valgus angle at the end of the backswing, as is recommended in other sports. This may decrease the magnitude of extensor moment placed on it by the strong quadriceps femoris muscle group, and may also reduce the need for rapid extension of the joint during the downswing, reducing the loads on some knee tissues. According to a doctor who collected data on 35 golfing knee injuries, “Less knee flexion is important. Stabilizing the weight on both legs should equalize the stress on the knees” (Guten, 1996, p.127). He also suggested that a turn which was generated to a greater extent by the upper body would be safer for the knees.

Putting together the advice from golf swing researchers, a less injury prone golf swing would have altered positions during the set-up, backswing, downswing and follow-through. The

set up would have a stronger grip (with respect to lead forearm pronation and trail forearm supination) than typically seen as well as a looser one; slightly flexed elbows; and a more upright posture. The backswing would maintain the alignment of all the regions of the spine, and would have complementary amounts of cervical, thoracic and lumbar rotations up to the top, as opposed to X-Factor twisting of one part of the spine against another. Additionally, there would be less lead-side trunk lateral flexion and lead knee flexion, and the lead arm would make a shorter backswing. The elbows would be held close to the chest to produce a flatter swing plane, and there would be reduced wrist motion. The downswing would continue to maintain spinal alignment, would have less shoulder protraction/retraction and forearm pronation, and would also have a well-timed weight shift. Finally, the finish would be made with an upright, not hyperextended, spine.

Limitations and Future Research.

There were some limitations in this review article. There are so many golf-related peer-reviewed journal articles that the scope of this literature review had to be restricted to a search of the data bases mentioned, and the proceedings of the World Scientific Congresses of Golf 1990, 1994 and 1998. Additional hand searches had to be made despite an exhaustive data base search to find cross disciplinary studies from medical, sports and biomechanics research. Finally, the raw biomechanical data extracted for the purpose of this study were not a part of the original authors' analyses, and so were often not tested for significance or effect size.

Future studies should be designed specifically for injury, and should compare positions, movements and loads to those known to cause tissue damage in other sports or common human activities. Not only angular displacements, but also linear movement in space should be assessed to understand the distance a joint must move through while being subjected to large muscle

forces. It would also be of great practical value to compare movements and loads on the joints before and after making a swing change to a less injury prone swing as suggested by various researchers and synthesized in this section.

Conclusion

This review of the literature, accompanied by a synthesis and analysis, can serve as a guide to golf practitioners – both players and coaches – as to the types of injury at each joint which are typical to golf. It may also create an awareness of the biomechanically validated causal factors of each. This review could also serve as a template for the creation of similar injury assessment studies in other sports.

A recommendation has been made for load-reducing factors at most major joints. Additionally, female amateur golfers should be specifically assessed for thoracic spine, and Elbow and knee injury potential, and seniors of both genders for compressive loads at the joints which are more prone to OA resulting from golf. In conclusion, there may be causation or exacerbation of injury during the golf swing, and a swing which involves less injury-causing positions and loads may increase the pain-free span of a golfer's playing years.

References

- Alentorn-Geli, E., Myer, G. D., Silvers, H. J., Samitier, G., Romero, D., Lázaro-Haro, C., & Cugat, R. (2009). Prevention of non-contact anterior cruciate ligament injuries in soccer players. Part 1: Mechanisms of injury and underlying risk factors. *Knee Surgery, Sports Traumatology, Arthroscopy*, *17*(7), 705-729.
- Anderson, K., Strickland, S. M., & Warren, R. (2001). Hip and groin injuries in athletes. *The American Journal of Sports Medicine*, *29*(4), 521-533.
- Andrews, J.R., & Whiteside, J.A. (1994). The shoulder. In C. N. Stover, J. R. McCarroll, & W. J. Mallon (Eds.), *Feeling up to par: Medicine from tee to green*. (pp. 127-142). Philadelphia, PA: F. A. Davis Company.
- Armstrong, N. B. (1994). Back pain: Diagnosis and treatment. In C. N. Stover, J. R. McCarroll, & W. J. Mallon (Eds.), *Feeling up to par: Medicine from tee to green*. (pp. 109-125). Philadelphia, PA: F. A. Davis Company.
- Bait, M. E. (1993). Golfing injuries. *Sports Medicine*, *16*(1), 64-71.
- Baker, M. L., Epari, D. R., Lorenzetti, S., Sayers, M., Boutellier, U., & Taylor, W. R. (2017). Risk factors for knee injury in golf: A systematic review. *Sports Medicine*, *47*(12), 2621-2639.
- Batt, M. E. (1992). A survey of golf injuries in amateur golfers. *British Journal of Sports Medicine*, *26*(1), 63-65.
- Bayes, M. C., & Wadsworth, L. T. (2009). Upper extremity injuries in golf. *The Physician and Sportsmedicine*, *37*(1), 92-96.

- Bechler, J. R., Jobe, F. W., Pink, M., Perry, J., & Ruwe, P. A. (1995). Electromyographic analysis of the hip and knee during the golf swing. *Clinical Journal of Sport Medicine: Official Journal of the Canadian Academy of Sport Medicine*, 5(3), 162-166.
- Bradshaw, E. J., Keogh, J. W., Hume, P. A., Maulder, P. S., Nortje, J., & Marnewick, M. (2009). The effect of biological movement variability on the performance of the golf swing in high-and low-handicapped players. *Research Quarterly for Exercise and Sport*, 80(2), 185-196.
- Bugbee, S. (2010). Rib stress fracture in a golfer. *Current Sports Medicine Reports*, 9(1), 40-42.
- Byl, C., Puttlitz, C., Byl, N., Lotz, J., & Topp, K. (2002). Strain in the median and ulnar nerves during upper-extremity positioning. *Journal of Hand Surgery*, 27(6), 1032-1040.
- Byrd, J. T. (2007). Hip arthroscopy in the athlete. *North American Journal of Sports Physical Therapy: NAJSPT*, 2(4), 217.
- Cabri, J., Sousa, J. P., Kots, M., & Barreiros, J. (2009). Golf-related injuries: A systematic review. *European Journal of Sport Science*, 9(6), 353-366.
- Cahalan, T. D., Cooney III, W. P., Tamai, K., & Chao, E. Y. (1991). Biomechanics of the golf swing in players with pathologic conditions of the forearm, wrist, and hand. *The American Journal of Sports Medicine*, 19(3), 288-293.
- Cann, A. P., Vandervoort, A. A., & Lindsay, D. M. (2005). Optimizing the benefits versus risks of golf participation by older people. *Journal of Geriatric Physical Therapy*, 28(3), 85-92.
- Chang, D., & Bosco, J. A. III. (2006). Cervical spine injuries in the athlete. *Bulletin of the NYU Hospital for Joint Diseases*. 64. 3-4.

- Chauhan, A., Jacobs, B., Andoga, A., & Baratz, M. E. (2014). Extensor tendon injuries in athletes. *Sports Medicine and Arthroscopy Review*, 22(1), 45-55.
- Cheetham, P. J., Martin, P. E., Mottram, R. E., & St Laurent, B. F. (2001). The importance of stretching the “X-Factor” in the downswing of golf: The “X-Factor Stretch”. *Optimising performance in golf*, 192-199.
- Cheetham, P. J., Rose, G. A., Hinrichs, R. N., Neal, R. J., Mottram, R. E., Hurrion, P. D., & Vint, P. F. (2008). Comparison of kinematic sequence parameters between amateur and professional golfers. In D. Crews & R. Lutz (Eds.). *Science and golf V: Proceedings of the World Scientific Congress of golf*.(pp. 30-36). Mesa, AZ.: Energy in Motion.
- Choi, A., Kang, T. G., & Mun, J. H. (2016). Biomechanical evaluation of dynamic balance control ability during golf swing. *Journal of Medical and Biological Engineering*, 36(3), 430-439.
- Chu, Y., Sell, T. C., & Lephart, S. M. (2010). The relationship between biomechanical variables and driving performance during the golf swing. *Journal of sports sciences*, 28(11), 1251-1259.
- Cohn, M. A., Lee, S. K., & Strauss, E. J. (2013). Upper extremity golf injuries. *Bull Hosp Jt Dis*, 71(1), 32-8.
- Cole, M. H., & Grimshaw, P. N. (2014). The crunch factor's role in golf-related low back pain. *The Spine Journal*, 14(5), 799-807.
- Dale, R. B., & Brumitt, J. (2016). Spine biomechanics associated with the shortened, modern one-plane golf swing. *Sports Biomechanics*, 15(2), 198-206.

- D'Amico, J., Betlach, M., Senkarik, R., Smith, R., & Voight, M. (2007). Return to golf following left total hip arthroplasty in a golfer who is right handed. *North American Journal of Sports Physical Therapy: NAJSPT*, 2(4), 251.
- Dargel, J., Gotter, M., Mader, K., Pennig, D., Koebke, J., & Schmidt-Wiethoff, R. (2007). Biomechanics of the anterior cruciate ligament and implications for surgical reconstruction. *Strategies in Trauma and Limb Reconstruction*, 2(1), 1-12.
- Dickenson, E., O'connor, P., Robinson, P., Campbell, R., Ahmed, I., Fernandez, M., ... & Griffin, D. (2016). Hip morphology in elite golfers: asymmetry between lead and trail hips. *Br J Sports Med*, bjsports-2016.
- D'Lima, D. D., Fregly, B. J., Patil, S., Steklov, N., & Colwell Jr, C. W. (2012). Knee joint forces: prediction, measurement, and significance. *Proceedings of the Institution of Mechanical Engineers, Part H: Journal of Engineering in Medicine*, 226(2), 95-102.
- Egret, C. I., Vincent, O., Weber, J., Dujardin, F. H., & Chollet, D. (2003). Analysis of 3D kinematics concerning three different clubs in golf swing. *International Journal of Sports Medicine*, 24(06), 465-470.
- Egret, C. I., Nicolle, B., Dujardin, F. H., Weber, J., & Chollet, D. (2006). Kinematic analysis of the golf swing in men and women experienced golfers. *International Journal of Sports Medicine*, 27(06), 463-467.
- Ekin, J. A., & Sinaki, M. (1993). Vertebral compression fractures sustained during golfing: report of three cases. *Mayo Clinic Proceedings*. 68 (6), 566-570.
- Ellen, M. I., & Smith, J. (1999). 2. Shoulder and upper extremity injuries. *Archives of Physical Medicine and Rehabilitation*, 80(5), S50-S58.

- Escamilla, R. F., & Andrews, J. R. (2009). Shoulder muscle recruitment patterns and related biomechanics during upper extremity sports. *Sports Medicine*, 39(7), 569-590.
- Farber, A. J., Smith, J. S., Kvitne, R. S., Mohr, K. J., & Shin, S. S. (2009). Electromyographic analysis of forearm muscles in professional and amateur golfers. *The American Journal of Sports Medicine*, 37(2), 396-401.
- Farrally, M. R., Cochran, A. J., Crews, D. J., Hurdzan, M. J., Price, R. J., Snow, J. T., & Thomas, P. R. (2003). Golf science research at the beginning of the twenty-first century. *Journal of Sports Sciences*, 21(9), 753-765.
- Felson, D. T., & Zhang, Y. (1998). An update on the epidemiology of knee and hip osteoarthritis with a view to prevention. *Arthritis & Rheumatology*, 41(8), 1343-1355.
- Finch, C., Sherman, C., & James, T. (1998). The epidemiology of golf injuries in Victoria, Australia: Evidence from sports medicine clinics and emergency department presentations. In M. R. Farrally & A. J. Cochran (Eds.), *Science and Golf III: Proceedings of the World Scientific Congress of Golf 1998*. (pp. 73-82). London. E & F Spon.
- Flynn, M. (1973). A new method of reduction of fractures of the neck of the femur based on anatomical studies of the hip joint. *Injury*, 5(4), 309-317.
- Foxworth, J. L., Millar, A. L., Long, B. L., Way, M., Vellucci, M. W., & Vogler, J. D. (2013). Hip joint torques during the golf swing of young and senior healthy males. *Journal of Orthopaedic & Sports Physical Therapy*, 43(9), 660-665.
- Fradkin, A. J., Cameron, P. A., & Gabbe, B. J. (2005). Golf injuries-common and potentially avoidable. *Journal of Science and Medicine in Sport*, 8(2), 163-170.

- Ganz, R., Leunig, M., Leunig-Ganz, K., & Harris, W. H. (2008). The etiology of osteoarthritis of the hip. *Clinical Orthopaedics and Related Research*, 466(2), 264-272.
- Gatt, C. J., Pavol, M. J., Parker, R. D., & Grabiner, M. D. (1998). Three-dimensional knee joint kinetics during a golf swing. *The American Journal of Sports Medicine*, 26(2), 285-294.
- Glazebrook, M. A., Curwin, S., Islam, M. N., Kozey, J., & Stanish, W. D. (1994). Medial epicondylitis: an electromyographic analysis and an investigation of intervention strategies. *The American Journal of Sports Medicine*, 22(5), 674-679.
- Gluck, G. S., Bendo, J. A., & Spivak, J. M. (2008). The lumbar spine and low back pain in golf: A literature review of swing biomechanics and injury prevention. *The Spine Journal*, 8(5), 778-788.
- Gosheger, G., Liem, D., Ludwig, K., Greshake, O., & Winkelmann, W. (2003). Injuries and overuse syndromes in golf. *The American Journal of Sports Medicine*, 31(3), 438-443.
- Grimshaw, P., Giles, A., Tong, R., & Grimmer, K. (2002). Lower back and elbow injuries in golf. *Sports Medicine*, 32(10), 655-666.
- Gryc, T., Zahalka, F., Maly, T., Mala, L., & Hrasky, P. (2015). Movement's analysis and weight transfer during the golf swing. *Journal of Physical Education and Sport*, 15(4), 781.
- Gulgin, H., Armstrong, C., & Gribble, P. (2009). Hip rotational velocities during the full golf swing. *Journal of Sports Science & Medicine*, 8(2), 296.
- Gulgin, H., Armstrong, C., & Gribble, P. (2010). Weight-bearing hip rotation range of motion in female golfers. *North American Journal of Sports Physical Therapy: NAJSPT*, 5(2), 55.
- Guten, G. N. (1996). Knee injuries in golf. *Clinics in Sports Medicine*, 15(1), 111-128.
- Hawes, M. C., & O'Brien, J. P. (2006). The transformation of spinal curvature into spinal deformity: pathological processes and implications for treatment. *Scoliosis*, 1(1), 3.

- Hawkes, R., O'Connor, P., & Campbell, D. (2013). The prevalence, variety and impact of wrist problems in elite professional golfers on the European Tour. *Br J Sports Med*, 47:1075-9.
- Healy, A., Moran, K. A., Dickson, J., Hurley, C., Smeaton, A. F., O'Connor, N. E., ... Chockalingam, N. (2011). Analysis of the 5 iron golf swing when hitting for maximum distance. *Journal of Sports Sciences*, 29(10), 1079-1088.
- Hewett, T. E., Myer, G. D., Ford, K. R., Heidt Jr, R. S., Colosimo, A. J., McLean, S. G., ... & Succop, P. (2005). Biomechanical measures of neuromuscular control and valgus loading of the knee predict anterior cruciate ligament injury risk in female athletes: a prospective study. *The American Journal of Sports Medicine*, 33(4), 492-501.
- Horan, S. A., Evans, K., Morris, N. R., & Kavanagh, J. J. (2010). Thorax and pelvis kinematics during the downswing of male and female skilled golfers. *Journal of Biomechanics*, 43(8), 1456-1462.
- Horan, S. A., & Kavanagh, J. J. (2012). The control of upper body segment speed and velocity during the golf swing. *Sports Biomechanics*, 11(2), 165-174.
- Hosea, T.M., Gatt, C.J., Galli, K.M., Langrana, N.A., & Zawadsky, J.P. (1990). Biomechanical analysis of the golfer's back. In: A.J. Cochran (Ed.). *Science and golf I. Proceedings of the First World Scientific Congress of Golf*. (pp. 43-48). London.: E & FN Spon
- Hosea, T.M., Gatt, C.J. & Gertner, E. (1994). Biomechanical analysis of the golfer's back. In C. N. Stover, J. R. McCarroll, & W. J. Mallon (Eds.), *Feeling up to par: Medicine from tee to green*. (pp. 97 - 108). Philadelphia, PA: F. A. Davis Company.
- Hume, P. A., Reid, D., & Edwards, T. (2006). Epicondylar injury in sport. *Sports Medicine*, 36(2), 151-170.

- Hovis, W. D., Dean, M. T., Mallon, W. J., & Hawkins, R. J. (2002). Posterior instability of the shoulder with secondary impingement in elite golfers. *The American Journal of Sports Medicine*, 30(6), 886-890.
- Jacobson, J. A., Miller, B. S., & Morag, Y. (2005). Golf and racquet sports injuries. In *Seminars in Musculoskeletal Radiology*, 9, 346-359.
- Jobe, F. W., Moynes, D. R., & Antonelli, D. J. (1986). Rotator cuff function during a golf swing. *The American Journal of Sports Medicine*, 14(5), 388-392.
- Kao, J. T., Pink, M., Jobe, F. W., & Perry, J. (1995). Electromyographic analysis of the scapular muscles during a golf swing. *The American Journal of Sports Medicine*, 23(1), 19-23.
- Karpinski, M. R., & Piggott, H. (1985). Greater trochanteric pain syndrome. A report of 15 cases. *Bone & Joint Journal*, 67(5), 762-763.
- Kim, D. H., Millett, P. J., Warner, J. J., & Jobe, F. W. (2004). Shoulder injuries in golf. *The American Journal of Sports Medicine*, 32(5), 1324-1330.
- Kumar, S. (2001). Theories of musculoskeletal injury causation. *Ergonomics*, 44(1), 17-47.
- Kuroda, K., & Ichikawa, N. (1977). Golfer's ribs fractures. *Japanese Journal of Physical Fitness and Sports Medicine*, 26(4), 182-190.
- Lee, A. D. (2009). Golf-related stress fractures: a structured review of the literature. *The Journal of the Canadian Chiropractic Association*, 53(4), 290.
- Lee, C. H., Hong, J. Y., Jeon, P. S., Hwang, K. H., Moon, W. S., Han, Y. H., & Jeong, H. J. (2017). Features of golf-related shoulder pain in Korean amateur golfers. *Annals of Rehabilitation Medicine*, 41(3), 394-401.

- Lee, D., Oh, S. E., Lee, I. K., Sim, T., Joo, S. B., Park, H. J., & Mun, J. H. (2015). Comparison of three normalization methods for 3D joint moment in the asymmetric rotational human movements in golf swing analysis. *Journal of Biosystems Engineering*, 40(3), 289-295.
- Lee, Y. S., & Lee, S. H. (2017). Golf-related spine and lower extremity injury. *The Korean Journal of Sports Medicine*, 35(1), 1-4.
- Lemak, L. J., Fleisig, G. S., Welch, C. M., Marting, B., & Zvijac, J. E. (1994). Usefulness of partial swings in the rehabilitation of a golfer. In A. J. Cochran & M. R. Farrally (Eds). *Science and Golf II, Proceedings of the First World Scientific Congress of Golf*. (pp. 14 – 19). London: E & FN Spon 50-58.
- Lequesne, M. G., Dang, N., & Lane, N. E. (1997). Sport practice and osteoarthritis of the limbs. *Osteoarthritis and Cartilage*, 5(2), 75-86.
- Lim, Y. T., Chow, J. W., & Chae, W. S. (2012). Lumbar spinal loads and muscle activity during a golf swing. *Sports Biomechanics*, 11(2), 197-211.
- Lin, H. C., Chou, C. S., & Hsu, T. C. (1994). Stress fractures of the ribs in amateur golf players. *Chinese Medical Journal*, 54(1), 33-37.
- Lindsay, D. M., Horton, J. F., & Vandervoort, A. A. (2000). A review of injury characteristics, aging factors and prevention programmes for the older golfer. *Sports Medicine*, 30(2), 89-103.
- Lindsay, D. M., Horton, J. F., & Paley, R. D. (2002). Trunk motion of male professional golfers using two different golf clubs. *Journal of Applied Biomechanics*, 18(4), 366-373.
- Lindsay, D. M., Mantrop, S., & Vandervoort, A. A. (2008). A review of biomechanical differences between golfers of varied skill levels. *International Journal of Sports Science & Coaching*, 3(Suppl. 1), 187-197.

- Lindsay, D. M., Versteegh, T. H., & Vandervoort, A. A. (2009). Injury prevention: Avoiding one of golf's more painful hazards. *International Journal of Sports Science & Coaching*, 4(1_suppl), 129-148.
- Lindsay, D. M., & Vandervoort, A. A. (2014). Golf-related low back pain: a review of causative factors and prevention strategies. *Asian Journal of Sports Medicine*, 5(4).
- Lu, Y., Chen, C., Kallakuri, S., Patwardhan, A., & Cavanaugh, J. M. (2005). Neural response of cervical facet joint capsule to stretch: a study of whiplash pain mechanism. *Stapp Car Crash Journal*, 49, 49.
- Lyles, K. W., Gold, D. T., Shipp, K. M., Pieper, C. F., Martinez, S., & Mulhausen, P. L. (1993). Association of osteoporotic vertebral compression fractures with impaired functional status. *The American Journal of Medicine*, 94(6), 595-601.
- Lynn, S. K., & Noffal, G. J. (2010). Frontal plane knee moments in golf: effect of target side foot position at address. *Journal of sports science & medicine*, 9(2), 275.
- Lynn, S. K., Noffal, G. J., FW Wu, W., & Vandervoort, A. A. (2012). Using principal components analysis to determine differences in 3D loading patterns between beginner and collegiate level golfers. *International Journal of Golf Science*, 1(1), 25-41.
- Mallon, W. J., & Colosimo, A. J. (1995). Acromioclavicular joint injury in competitive golfers. *Journal of the Southern Orthopaedic Association*, 4(4), 277-282.
- Marshall, R. N., & McNair, P. J. (2013). Biomechanical risk factors and mechanisms of knee injury in golfers. *Sports Biomechanics*, 12(3), 221-230.
- Marta, S., Silva, L., Vaz, J. R., Castro, M. A., Reinaldo, G., & Pezarat-Correia, P. (2016). Electromyographic analysis of the lower limb muscles in low-and high-handicap golfers. *Research Quarterly for Exercise and Sport*, 87(3), 318-324.

- Martin, C., Bideau, B., Bideau, N., Nicolas, G., Delamarche, P., & Kulpa, R. (2014). Energy flow analysis during the tennis serve: Comparison between injured and noninjured tennis players. *The American Journal of Sports Medicine*, 42(11), 2751-2760.
- McCarroll, J. R., & Gioe, T. J. (1982). Professional golfers and the price they pay. *The Physician and Sportsmedicine*, 10(7), 64-70.
- McCarroll, J.R. (1994). The lower extremity. In C. N. Stover, J. R. McCarroll, & W. J. Mallon (Eds.), *Feeling up to par: Medicine from tee to green*. (pp. 165-169). Philadelphia, PA: F. A. Davis Company.
- McCarroll, J. R. (2001). Overuse injuries of the upper extremity in golf. *Clinics in Sports Medicine*, 20(3), 469-479.
- McHardy, A. J., & Pollard, H. P. (2005). Golf and upper limb injuries: a summary and review of the literature. *Chiropractic & Osteopathy*, 13(1), 7.
- McHardy, A., Pollard, H., & Luo, K. (2006). Golf injuries: A review of the literature. *Sports Medicine*, 36(2), 171-187.
- McHardy, A., Pollard, H., & Lou, K. (2007). The epidemiology of golf related injuries in Australian amateur golfers-a multivariate analysis. *South African Journal of Sports Medicine*, 19(1), 12-19.
- McNicholas, M.J., Nilesen, A., & Knill-Jones, R. P. (1998). Golf injuries in Scotland. In M. R. Farrally & A. J. Cochran (Eds.), *Science and Golf III: Proceedings of the World Scientific Congress of Golf 1998*. (pp. 65-72). London. E & F Spon.

- McTeigue, M., Lamb, S. R., Mottram, R., & Pirozzolo, F. (1994). Spine and hip motion analysis during the golf swing. In A. J. Cochran & M. R. Farrally (Eds). *Science and Golf II, Proceedings of the World Scientific Congress of Golf 1998*. (pp. 50-58). London. E & F Spon.
- Meister, D. W., Ladd, A. L., Butler, E. E., Zhao, B., Rogers, A. P., Ray, C. J., & Rose, J. (2011). Rotational biomechanics of the elite golf swing: benchmarks for amateurs. *Journal of Applied Biomechanics*, 27(3), 242-251.
- Meister, K., & Andrews, J. R. (1993). Classification and treatment of rotator cuff injuries in the overhand athlete. *Journal of Orthopaedic & Sports Physical Therapy*, 18(2), 413-421.
- Mitchell, K., Banks, S., Morgan, D., & Sugaya, H. (2003). Shoulder motions during the golf swing in male amateur golfers. *Journal of Orthopaedic & Sports Physical Therapy*, 33(4), 196-203.
- Moore, K. L., Dalley, A. F., & Agur, A. M. R. (2010). *Clinically Oriented Anatomy* (7th ed.). Philadelphia, PA: Lippincott Williams & Wilkins.
- Morganti, C., Sweeney, C. A., Albanese, S. A., Burak, C., Hosea, T., & Connolly, P. J. (2001). Return to play after cervical spine injury. *Spine*, 26(10), 1131-1136.
- Miura, K. (2001). Parametric acceleration—the effect of inward pull of the golf club at impact stage. *Sports Engineering*, 4(2), 75-86.
- Murakami, K., Hamai, S., Okazaki, K., Ikebe, S., Shimoto, T., Hara, D... & Iwamoto, Y. (2016). In vivo kinematics of healthy male knees during squat and golf swing using image-matching techniques. *The Knee*, 23(2), 221-226.
- Murray, P. M., & Cooney, W. P. (1996). Golf-induced injuries of the wrist. *Clinics in Sports Medicine*, 15(1), 85-109.

- National Golf Foundation. (2017). Golf participation in the U.S. 2017 Edition. Retrieved from http://aga-bc.org/wp-content/uploads/2017/05/99MR257S_GOLF_PARTICIPATION_US_SUMMARY_2017-copy-002.pdf
- Neal, R., Lumsden, R., Holland, M., & Mason, B. (2007). Body segment sequencing and timing in golf. *International Journal of Sports Science & Coaching*, 2(1_suppl), 25-36.
- Neumann, D. A. (2010). Kinesiology of the hip: a focus on muscular actions. *Journal of Orthopaedic & Sports Physical Therapy*, 40(2), 82-94.
- Nicholas, J. J., Reidy, M., & Oleske, D. M. (1998). An epidemiologic survey of injury in golfers. *Journal of Sport Rehabilitation*, 7(2), 112-121.
- O'Connor, P. J., Campbell, R., Bharath, A. K., Campbell, D., Hawkes, R., & Robinson, P. (2016). Pictorial review of wrist injuries in the elite golfer. *British Journal of Sports Medicine*. 50(17):1053-1063.
- Ohuri, T., Mae, T., Shino, K., Tachibana, Y., Fujie, H., Yoshikawa, H., & Nakata, K. (2017). Varus-valgus instability in the anterior cruciate ligament-deficient knee: effect of posterior tibial load. *Journal of Experimental Orthopaedics*, 4(1), 24.
- Okuda, I., Gribble, P., & Armstrong, C. (2010). Trunk rotation and weight transfer patterns between skilled and low skilled golfers. *Journal of sports science & medicine*, 9(1), 127.
- Pearson, A. M., Ivancic, P. C., Ito, S., & Panjabi, M. M. (2004). Facet joint kinematics and injury mechanisms during simulated whiplash. *Spine*, 29(4), 390-397.
- Pfeiffer, J. L., Zhang, S., & Milner, C. E. (2014). Knee biomechanics during popular recreational and daily activities in older men. *The Knee*, 21(3), 683-687.

- Phadke, V., Camargo, P. R., & Ludewig, P. M. (2009). Scapular and rotator cuff muscle activity during arm elevation: a review of normal function and alterations with shoulder impingement. *Brazilian Journal of Physical Therapy, 13*(1), 1-9.
- Philippon, M. J., & Schenker, M. L. (2005). Athletic hip injuries and capsular laxity. *Operative Techniques in Orthopaedics, 15*(3), 261-266.
- Purevsuren, T., Kwon, M. S., Park, W. M., Kim, K., Jang, S. H., Lim, Y. T., & Kim, Y. H. (2017). Fatigue injury risk in anterior cruciate ligament of target side knee during golf swing. *Journal of Biomechanics, 53*, 9-14.
- Rasad, S. (1974). Golfer's fractures of the ribs: Report of 3 cases. *American Journal of Roentgenology, 120*(4), 901-903.
- Read, M. T. (1994). Case report - stress fractures of the rib in a golfer. *British Journal of Sports Medicine, 28*(3), 206.
- Rebella, G. (2015). A prospective study of injury patterns in collegiate pole vaulters. *The American Journal of Sports Medicine, 43*(4), 808-815.
- Reed, J. J., & Wadsworth, L. T. (2010). Lower back pain in golf: a review. *Current Sports Medicine Reports, 9*(1), 57-59.
- Rettig, A.C. (1994). The wrist and hand. In C. N. Stover, J. R. McCarroll, & W. J. Mallon (Eds.), *Feeling up to par: Medicine from tee to green.* (pp. 151-163). Philadelphia, PA: F.A. Davis Company.
- Rivera, J. E. (1994). Open versus closed kinetic chain rehabilitation of the lower extremity: a functional and biomechanical analysis. *Journal of Sport Rehabilitation, 3*(2), 154-167.

- Roaf, R. (1960). A study of the mechanics of spinal injuries. *Bone & Joint Journal*, 42(4), 810-823.
- Roos, P. E., Barton, N., & van Deursen, R. W. (2012). Patellofemoral joint compression forces in backward and forward running. *Journal of Biomechanics*, 45(9), 1656-1660.
- Ryu, J., Cooney, W. P., Askew, L. J., An, K. N., & Chao, E. Y. (1991). Functional ranges of motion of the wrist joint. *Journal of Hand Surgery*, 16(3), 409-419.
- Sanders, R. H., & Owens, P. C. (1992). Hub movement during the swing of elite and novice golfers. *International Journal of Sport Biomechanics*, 8(4), 320-330.
- Seitz, A. L., McClure, P. W., Finucane, S., Boardman, N. D., & Michener, L. A. (2011). Mechanisms of rotator cuff tendinopathy: intrinsic, extrinsic, or both? *Clinical Biomechanics*, 26(1), 1-12.
- Seaman, D. R., & Bulbulian, R. (2000). A review of back pain in golfers: etiology and prevention. *Research in Sports Medicine: An International Journal*, 9(3), 169-187.
- Seminati, E., & Minetti, A. E. (2013). Overuse in volleyball training/practice: a review on shoulder and spine-related injuries. *European Journal of Sport Science*, 13(6), 732-743.
- Sherman, C. A., & Finch, C. F. (1999). The ideal golf swing: an evaluation of its mechanics and relationship to injury risk. *Safety Science Monitor*, 3. Retrieved from <http://ssmon.chb.kth.se/volumes/vol3/se2.pdf>
- Sherman, C. A., & Finch, C. F. (2000). Preventing injuries to competitive and recreational adult golfers: what is the evidence? *Journal of Science and Medicine in Sport*, 3(1), 65-78.
- Smidt, G. L. (1973). Biomechanical analysis of knee flexion and extension. *Journal of Biomechanics*, 6(1), 79-92.

- Stanish, W.D., Loebenberg, M.I., & Kozey, J.W. (1994). The elbow. In C. N. Stover, J. R. McCarroll, & W. J. Mallon (Eds.), *Feeling up to par: Medicine from tee to green*. (pp. 143-149). Philadelphia, PA: F.A.Davis Company.
- Stedman's Medical Dictionary*. (2016). Baltimore, MA: Lippincott Williams & Wilkins.
- Stockard, A. R. (2001). Elbow injuries in golf. *The Journal of the American Osteopathic Association*, *101*(9), 509-516.
- Stover, C. N., & Mallon, W. J. (1992). Golf injuries: treating the play to treat the player. *J Musculoskeletal Med*, *9*, 55-72.
- Stuelcken, M. C., Ferdinands, R. E., & Sinclair, P. J. (2010). Three-dimensional trunk kinematics and low back pain in elite female fast bowlers. *Journal of Applied Biomechanics*, *26*(1), 52-61.
- Sugaya, H., Tsuchiya, A., Moriya, H., Morgan, D.A., & Banks, S. A. (1998). Low back injury in elite and professional golfers: An epidemiologic and radiographic study. In M. R. Farrally & A. J. Cochran (Eds.), *Science and Golf III: Proceedings of the World Scientific Congress of Golf 1998*. (pp. 83-91). London. E & F Spon.
- Sutcliffe, J., Ly, J. Q., Kirby, A., & Beall, D. P. (2008). Magnetic resonance imaging findings of golf-related injuries. *Current Problems in Diagnostic Radiology*, *37*(5), 231-241.
- Swartz, E. E., Floyd, R. T., & Cendoma, M. (2005). Cervical spine functional anatomy and the biomechanics of injury due to compressive loading. *Journal of Athletic Training*, *40*(3), 155.
- Taneja, A. K., Negromonte, F. P., & Skaf, A. (2013). Stress injury of the acromion: Case report and literature review. *European Journal of Orthopaedic Surgery & Traumatology*, *23*(2), 189-192.

- Teu, K. K., Kim, W., Fuss, F. K., & Tan, J. (2006). The analysis of golf swing as a kinematic chain using dual Euler angle algorithm. *Journal of Biomechanics*, 39(7), 1227-1238.
- Timpka, T., Finch, C. F., Goulet, C., Noakes, T., & Yammine, K. (2008). Meeting the global demand of sports safety: the intersection of science and policy in sports safety. *Sports Medicine*, 38(10), 795.
- Tojima, M., Ogata, N., Yozu, A., Sumitani, M., & Haga, N. (2013). Novel 3-dimensional motion analysis method for measuring the lumbar spine range of motion: repeatability and reliability compared with an electrogoniometer. *Spine*, 38(21), E1327-E1333.
- Tucker, C. J. (2016). Golf Injuries. *Sports Medicine*.
- Wadsworth, L. T. (2007). When golf hurts: musculoskeletal problems common to golfers. *Current Sports Medicine Reports*, 6(6), 362-365.
- Warden, S. J., Gutschlag, F. R., Wajswelner, H., & Crossley, K. M. (2002). Aetiology of rib stress fractures in rowers. *Sports Medicine*, 32(13), 819-836.
- Watkins, R. G., Uppal, G. S., Perry, J., Pink, M., & Dinsay, J. M. (1996). Dynamic electromyographic analysis of trunk musculature in professional golfers. *The American Journal of Sports Medicine*, 24(4), 535-538.
- Williams, J.G.P. (1980). *Color atlas of injury in sport*. Chicago, IL: Year Book Medical Publishers, Inc.
- Wilson, F., Gissane, C., Gormley, J., & Simms, C. (2010). A 12-month prospective cohort study of injury in international rowers. *British Journal of Sports Medicine*, 44(3), 207-214.
- Wright, T. W., Glowczewskie, F., Cowin, D., & Wheeler, D. L. (2001). Ulnar nerve excursion and strain at the elbow and wrist associated with upper extremity motion. *Journal of Hand Surgery*, 26(4), 655-662.

- Wrobel, J. S., Marclay, S., & Najafi, B. (2012). Golfing skill level postural control differences: A brief report. *Journal of sports science & medicine*, 11(3), 452.
- Wulf, G. & Lewthwaite, R. (2010). Effortless Motor Learning?: An external focus of attention enhances movement effectiveness and efficiency. In B. Bruya (Ed.), *Effortless attention: A new perspective in the cognitive science of attention and action* (pp. 75-101). Cambridge, MA: The MIT Press.
- Yamabe, E., Nakamura, T., Pham, P., & Yoshioka, H. (2012, September). The athlete's wrist: ulnar-sided pain. In *Seminars in Musculoskeletal Radiology* (Vol. 16, No. 04, pp. 331-337). Thieme Medical Publishers.
- Zhang, X., & Shan, G. (2014). Where do golf driver swings go wrong? Factors influencing driver swing consistency. *Scandinavian Journal of Medicine and Science in Sports*, 24(5), 749-757.
- Zheng, N., Barrentine, S. W., Fleisig, G. S., & Andrews, J. R. (2008). Kinematic analysis of swing in pro and amateur golfers. *International Journal of Sports Medicine*, 29(06), 487-493.
- Zheng, N., Barrentine, S. W., Fleisig, G. S., & Andrews, J. R. (2008). Swing kinematics for male and female pro golfers. *International Journal of Sports Medicine*, 29(12), 965-970.