
Abstract

What factors play a role to ensure a knee joint does what it should given the demands of moving through the physical environment? This paper aims to probe the hypothesis that intra-articular joint pressures, once a topic of interest, have been left aside in contemporary frameworks in which we now view knee joint function. The focus on ligamentous deficiencies and the chondrocentric view of osteoarthritis, while important, have left little attention to the consideration of other factors that can impair joint function across the lifespan. Dynamic knee stability is required during every step we take. While there is much known about the role that passive structures and muscular activation play in maintaining a healthy knee joint, this framework does not account for the role that intra-articular joint pressures may have in providing joint stability during motion and how these factors interact. Joint injuries invariably result in some form of intra-articular fluid accumulation. Ultimately, it may be how the knee mechanically responds to this fluid, of which pressure plays a significant role that provides the mechanisms for continued function. Do joint pressures provide an important foundation for maintaining knee function? This hypothesis is unique and argues that we are missing an important piece of the puzzle when attempting to understand implications that joint injury and disease have for joint function.


Introduction

For many, a healthy tibio-femoral joint is fundamental for ambulation, getting out of a chair and enjoying sport and recreational activities. The knee is considered a tri-compartmental, complex modified ovoid synovial joint where both active and passive periarticular structures exist to ensure stability is maintained during movement [1]. This has been thought to model the conceptual framework of the spinal stabilizing system proposed by Panjabi [2]. Of the passive structures, the capsule, synovial fluid and the corresponding intra-articular pressures have received little attention in the discussion of dynamic knee joint control and movement pathomechanics in recent years. Intra-articular pressures can fluctuate with movement, in the presence, and with severity of joint disease [3-6]. For over 40 years, we have known that joint pressures are increased as a result of effusions in many individuals presenting with knee arthropathy [5,7-9]. To date, there is little discussion on this topic and given the surmounting burden of joint disease in our population and its role in affecting how active we can be in our older age, it is due time we begin to hypothesize.

Hypothesis

Current theories that pertain to why an individual’s knee, burdened by osteoarthritis, moves the way it does, stem largely from over 40 years of study on joint compressive loading and the joints response to this loading environment. It has been predominantly mechanical in nature, where only recently has the muscular system been included. The muscles provide forces to assist joint stability and movement during walking. The recent emergence of electromyographical studies in knee OA gait, has left
many researchers searching for an explanation on why these activations are so different and in some cases, quite unique from health knee joints. One hypothesis is that muscle activation is increased to stabilize the laxity of deteriorating joint compartments [10]. Others suggest muscle activation patterns are a response to decrease medial compartment loads in individuals with medial compartment disease [11]. Still others hypothesize that muscle activation is increased on the lateral side of the joint to reduce the tensile strain placed on the joint during gait in individuals with medial compartment disease [12]. While the truth is likely a combination of these mechanisms, interestingly not one paper has discussed the role of joint swelling. Effusions can occur in over 80% of patients with knee OA [13,14]. Recently, we found that individuals with knee OA and effusion walk with different kinematics, kinetics and muscle activation patterns than individuals with knee OA and no effusion [15]. Can we hypothesize that knee joint function and effusion are more closely related than what current models appear to support? Should effusion, found in many individuals with knee OA, be integrated into current pathomechanical models of knee OA gait? This paper aims to probe the hypothesis that intra-articular joint pressures, once a topic of interest, are altered with effusion presence, yet have been left aside in contemporary frameworks in which we now view knee joint function.

**Evaluation of the Hypothesis**

*A Healthy Joint*

In a joint devoid of significant pathology, intra-articular pressure has been found to be subatmospheric with average values approximating -4mmHg [3,16,17]. Gaffney et
al. [16] found resting intra-articular pressure in metacarpalphalengeal (MCP) and talocrural joints to be -2 mmHg and -3 mmHg respectively. In normal non-distended knee joints, intra-articular pressure ranged from -10 mmHg to 0 mmHg [17]. A healthy joint contains highly viscous synovial fluid that is constrained within the joint capsule and positioned between and adherent to the synovium and mobile surfaces of the articular cartilage [18]. When a joint moves, this film is subjected to shear force with resultant tensile stress that is recorded as subatmospheric pressure [18]. In a healthy, non-distended knee joint, isometric quadriceps contractions during a straight leg raise and resisted straight leg raise reduced intra-articular pressure to -43.9 mmHg and -106.9 mmHg respectively [3]. Gaffney et al. [16] found no change in intra-articular pressure to exist in the MCP or talocrural joints with isometric exercise. Intra-articular pressures remained negative and did not decrease or increase. The differences between these studies are thought to be a result of patellar movement and the fact that patellar movement can still occur during “isometric” knee extension exercises [18]. Fluctuating intra-articular pressures have also been found during gait [3] although this remains a hypothesis not well tested. Only a single study has investigated this affect and found that stance phase coincided with a phase of decreasing intra-articular pressure. Intra-articular pressures during the stance phase of gait were similar to those found during the isometric quadriceps exercises in this study. Levick [6] stated that pressure changes depend on whether active muscles distract the knee joint capsule or compress it. In a healthy, non-distended knee joint, movements of one joint surface on another are thought to draw joint tissues towards each other because of decreasing pressure, imparting a stabilizing factor and promoting normal joint tracking [18]. While this remains a hypothesis to be tested,
the notion suggests that intra-articular pressures in a healthy, non-distended knee joint may be important to consider for the provision of stability during movement. Intra-articular pressure profiles in a healthy non-distended knee joint do not represent those of a distended joint, whether acutely distended or with chronic effusion present. In fact, the exact opposite occurs.

_A Swollen Joint_

Joints that are effused (increased volume) are a common clinical finding in individuals with knee OA [15,19-21]. Levick [6] provides a detailed account of the relationship between pressure and volume with respect to synovial joints. Volume and pressure are related by wall stress, a derivation of Laplace’s Law; the greater the wall stresses at a given volume, the greater the pressure. While components of this relationship may be linear [22], investigating the overall pressure spectrum reveals a non-linear relationship that depends on a number of factors [6]. These include joint angle, the previous exposure of the joint to fluid volume, the volume time course and joint motion [6]. Intra-articular pressure increases at extreme joint ranges of motion including both flexion and extension. These changes are increasingly pronounced in the presence of greater volume [3,17]. Levick [6] concludes that given the role that time and history play on the volume/pressure relationship, acute effusions may produce higher pressures than chronic or intermittent effusions. In general, in the presence of knee joint effusions, intra-articular pressures are higher than pressures found in healthy, non-distended knee joints and the relationship between the noted effusion and subsequent pressure profile is complex.
Resting intra-articular pressures found in effused knee joints are higher than pressures in healthy non-distended joints [4,5,7,8,16]. Subatmospheric pressures at rest are absent and studies have found mean resting values to be ~20 mmHg [8], ~19 mmHg [4] in the knee joint and 14 mmHg and 16 mmHg in the MCP and talocrural joints respectively[16]. Most remarkable, is the effect of muscle contraction, where in healthy, non-distended joints, intra-articular pressure was maintained or decreased in the case of the knee joint. In the presence of positive intra-articular pressure at rest, intra-articular pressures increase. This increase is not modest and can reach levels 5-10 fold greater than resting pressures [5,8,16], and appears to depend on whether the effusion was chronic or acute [8]. In individuals with knee OA, Jawed et al. [5] found that intra-articular pressure increased from 17 mmHg at rest to 56 mmHg during maximal isometric quadriceps contraction. Merry et al. [8] also found an increase in a single individual with knee OA where intra-articular pressure at rest was 6 mmHg and during maximal knee extension increased to ~140 mmHg. These findings are not unique to isolated quadriceps contractions. Jayson et al. [3] found that in contrast to subatmospheric knee pressures during gait in healthy non-distended knees, with the lower pressures coinciding with heel-strike, no negative phase of intra-articular pressure was found in rheumatoid joints and the stance phase always coincided with peak pressure, increasing in magnitude with increasing volume of effusion. This finding was consistent when effusion (10-20mL) was introduced to healthy knee joints. The magnitude of the subatmospheric pressure diminished and the stance phase became positive and became the peak pressure [3]. Given subatmospheric intra-articular pressures have been thought to provide joint stability and promote normal articular tracking [18], these findings of opposition in the
presence of knee effusion are important to consider in deriving future hypotheses pertaining to joint function in individuals with knee pathology.

**Implications**

If individuals present with knee joint effusion, the normal sub-atmospheric intra-articular pressures are not present and this may greatly impair knee joint function. James et al. [23] found that synovial blood flow can be compromised by elevations in intra-articular pressure (>44 mmHg) which was observed during standing and walking. This pressure increase can cause synovial ischemia and manifest in a perfusion/metabolic demand mismatch in patients that present with knee effusions; hypothesized as a mechanism for hypoxic-reperfusion joint injury [24]. In a comparative study investigating intra-articular pressure dynamics in joints with acute traumatic and chronic effusions, Merry et al. [8] found distinct differences in pressure responses with quadriceps contraction. While no patients experienced pain during quadriceps contractions, a finding consistent with other studies [17,25], it was stated that all patients with acute effusions presented with a high degree of quadriceps inhibition. At rest and immediately after quadriceps contraction, the intra-articular pressure was ~19 mmHg and ~2 mmHg for individuals with chronic and acute effusions respectively. During quadriceps contraction, these pressures increased to 222.5 mmHg and 13.7 mmHg for those with chronic and acute effusions respectively. This phenomena is not only localized to the knee, as one participant with chronic shoulder effusion, also experienced greater pressures [8]. These findings provide a case for the importance of understanding both
acute and chronically effused knee joint and the role of included articular pressure in current frameworks describing mechanisms of joint function.

**Inhibition**

In the acute effusion state, there is an impression that quadriceps inhibition is protective, limiting the generation of large intra-articular pressures. In contrast, individuals presenting with chronic effusions do not appear to present with significant quadriceps inhibition (i.e. do not possess this protective response) evidenced by the large intra-articular pressures that are generated during contractions.

deAndrade et al. [25], studied the effect of joint distension on muscle reflex inhibition in the knee joint, championing our understanding of the role that joint effusion presence has in quadriceps neuromuscular function. The authors report that increasing the distension of the knee in both healthy individuals and those presenting with arthropathy after joint aspiration was associated with progressive weakening of quadriceps voluntary contraction and amplitude diminution of electromyographic signals from the vastus lateralis [25]. Subsequent studies have continued to report the effect of knee effusion on quadriceps inhibition. Fahrer et al. [26] found that aspiration of the effused knee lead to a significant increase in strength (~14%). When an increase in muscle strength was apparent, a clear increase in EMG voltage amplitude was also observed [26]. Sagittal plane tibio-femoral joint angle and chronicity of knee effusion has been shown to alter quadriceps inhibition [27], findings that are in line with intra-articular pressure studies [8,17]. Jones et al. [27] found that with the knee positioned at 90 degrees individuals with chronic effusion of arthritic cause (avg. duration ~8 years)
showed some inhibition of force generation (4-14%) where significantly more inhibition was evident when the muscles were tested in a position of knee extension (8-30%). Authors also found no obvious relationship between, amount of inhibition and size of effusion, pain experienced during contraction or muscle strength and aspiration did not have any effect on what inhibition there was [27]. These findings corroborate those of Merry et al. [8] supporting that individuals presenting with chronic effusions do not have the inhibitory influence on quadriceps contraction that individuals presenting with acute effusions do. These experimental findings, using a model of isometric quadriceps contractions can be translated into functional activities.

Jayson et al. [3] found that during gait, in the presence of an acutely effused joint, as well as in individuals presenting with chronic effusions, the knee joint experienced the greatest intra-articular pressures during the stance phase of the gait cycle. Given the findings of Merry et al, [8] it is expected that individuals with acute effusions will present with quadriceps inhibition and altered gait mechanics. The effect of experimental knee effusion on the electromyogram of knee joint muscles during gait has been studied. Using an acute effusion model Torry et al. [28] found that during 0 to 25 percent of the gait cycle the average activation of vastus medialis and vastus lateralis was reduced with increased volume of effusion and participants went in to less knee flexion, supporting the authors suggesting of quadriceps avoidance gait. Recently, Rutherford et al., [15] found that individuals with knee OA and effusion walked in greater knee flexion during the entire stance phase and had greater quadriceps and hamstring activity suggesting effusion was altering joint mechanics over and above the altered mechanics associated with knee OA. This partly contrasted the acute effusion model, particularly during early stance.
The findings of Torry et al., [28] support those of Merry et al. [8] in stating that acute knee effusions impair quadriceps muscle function. The results of Rutherford et al., [15] suggest longer standing effusion alters knee mechanics differently, not necessarily inhibiting the quadriceps as supported by others [8,27]. Further work is required to test these hypotheses, investigating the suspected underlying mechanism; intra-articular tibio-femoral pressures during gait.

Mechanisms

It is currently supported that the presence of acute knee effusion alters pre-synaptic inhibition of the quadriceps alpha motoneurons [29,30]. The integration of sensory feedback signals and motor commands within the central nervous system plays a pivotal role in controlling movement [31]. The neurological influence that effusion has on quadriceps function has been tested through the Hoffmann (H) - reflex response [29,30,32]. The H-reflex is characterized by the projection of 1a afferents onto homonymous motoneurons, an electrical analog to the patellar tendon tap reflex of the quadriceps, where a reduction in the amplitude of this reflex is assumed to indicate an increased level of pre–synaptic inhibition, and thus an indicator of motoneuron excitability [33]. Specifically, the electrical response is determined by the amount of transmitter release from the 1a afferents, and the excitability of the motoneuron pool, which can be altered in the presence of inhibitory potentials, elicited by an activation of an inhibitory pathway [31].

An inhibitory signal may come from many of the knee joint sensory mechanoreceptors in response to increased intra-articular pressure including the pacinian...
corpuscles, golgi joint receptors and ruffini endings [1,34]. The most plausible receptors intricately involved in this response is that of the 1) the pacinian corpuscle that has been found to exist in the deep layers of the fibrous capsule [35,36] and 2) Ruffini endings [29,32]. Pacinian corpuscles are extremely sensitive to capsule deformation and are rapidly adapting [1,35]. In contrast, Ruffini endings are slowly adapting [1]. Pacinian corpuscles detect joint motion and deformation caused by mechanically applied pressure and are only active during the application and removal of a stimulus [1,35]. Jawed et al. [5] found that knee joint intra-articular pressure significantly increased from 17mmHg to 56mmHg during a maximal resisted isometric quadriceps contraction in individuals with knee OA who presented with a positive bulge sign for effusion. There was also a significant correlation between the volume of fluid aspirated and the drop in intra-articular pressure [5]. Although, pacinian corpuscles may be involved in the immediate response to pressure increases, prolonged inhibition has also been found implicating the role of ruffini endings. H-reflex amplitude reductions have been shown to persist after initial effusion [29] despite a reduction in intra-articular pressure [32] leading authors to believe that slowly adapting receptors are also involved in the sensory motor integration of impaired quadricep muscle function. These findings support the possible role of distension sensitive mechanoreceptors in the subsequent inhibitory influence on quadriceps activation during voluntary activation. Moreover, in the presence of chronic effusions, joint capsule mechanoreceptors may become habituated and insensitive to alterations in pressure associated with quadriceps contraction supporting the work of Jones et al. [27] and Merry et al. [8].
Intra-articular pressures are modulated by the presence and acuity of joint effusion. If the hypotheses that intra-articular pressure fluctuation provides a mechanism for the maintenance of joint function in states where joint effusion is present were true, considerable implications exist for understanding knee mechanics and muscle activation in knee injury and disease. Few, if any investigations on gait mechanics in individuals with knee OA, report on, include or discuss joint effusion as a potential explanatory variable within their analytical methods. This hypothesis argues that we are missing important information when attempting understand implications that joint injury and disease have for joint function.

**Conclusion**

Solomonow concluded that preservation of knee joint stability should be considered a synergistic function in which bones, ligaments, joint capsule, muscles, tendons and sensory receptors function in accord [1]. This models the conceptual framework of the spinal stabilizing system proposed by Panjabi [2]. Unfortunately, this paradigm does not embrace the possible influence of intra-articular joint pressures in promoting joint tracking and joint stability during motion. Most joint lesions, including ligament injuries and various arthropathies are accompanied by a variable amount of space-occupying fluid within the joint, which inherently raises intra-articular pressure. Given the pressure response to knee joint muscle contractions, when resting intra-articular pressure is atmospheric is opposite to the response when resting intra-articular pressure is sub-atmospheric, joint function may be compromised. Although few studies exist that, employ an acute effusion model to study the effect of joint distension on
muscle function, the literature supports that chronic effusions do not respond in a similar manner. Rarely do joint lesions exist that do not demonstrate an intra-articular accumulation of fluid. It is hypothesized that joint mechanics and neuromuscular function may be influenced by the presence and chronicity of effusion however these studies have not considered the presence of effusion and possible implications that this may have for data interpretation. Future hypotheses should focus on testing intra-articular pressure profiles during gait and whether these are altered with joint injury and disease. Clues may be excised that will improve our understanding of knee injury and disease pathomechanics; aiding the development of a comprehensive framework to minimize the burden of the impaired knee joint on physical function.

Conflict of Interest Statement

The author has no personal or financial conflicts of interest pertaining to this manuscript.

Acknowledgements

There were no sources of funding for this manuscript.
References


