The assessment of instability in the osteoarthritic knee

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- Patient-reported instability is a common complaint amongst those with knee arthritis.
- Much research has examined the assessment of self-reported instability in the knee; however, no definitive quantitative measure of instability has been developed.
- This review focuses on the current literature investigating the nature of self-reported instability in the arthritic knee and discusses the possibilities of further investigation.

Keywords: knee; osteoarthritis; instability; gait analysis

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Introduction

Instability - reported as a feeling of buckling or giving way - is a commonplace symptom in osteoarthritic (OA) knees, being found in up to 72% of individuals.1–5 In an attempt to understand and treat instability, either through total knee replacement (TKR), osteotomy or through physical therapies, it is important to define what is characteristic about the unstable in comparison with the stable OA knee. Since the first published incidence of instability in the OA knee,1 factors with potential association to instability have been examined such as joint laxity, muscle strength, proprioception, knee joint stiffness, disease severity and gait parameters, finding several significant associations.2,6-9 This is of importance as recent findings make clear that instability in the native knee has associations with poor function,3,10 and this instability may persist post-operatively after TKR with a nearly doubled rate of fear of falling, and significantly increased limitation of activities.5 Currently ‘patient-reported instability’ is the benchmark used in clinical studies as well as in clinical assessment.

Patients usually describe the sensation of ‘giving way’, ‘slipping’, ‘buckling’ or a lack of confidence in the knee; this has been recognized in arthritic populations for over a decade and extensive research has been done into its prevalence following arthroplasty surgery, with several recent papers reviewing the nature of instability post-operatively.11-15 However, such an extensive body of literature does not exist for the native knee. Recent research has attempted to provide an objective description of instability by investigating various kinematic and biomechanical factors that may describe it.6-9,16-23 While these studies are heterogeneous in design and do not lend themselves to meta-analysis, a qualitative overview of this work can give insight into understanding instability. Therefore, this review aims to explore current definitions and prevalence of self-reported instability, before examining various components of knee structure and function that have been examined in an attempt to quantify instability. The results of these studies will be examined together to form conclusions as to the biomechanical nature of self-reported instability and to suggest further direction for research to produce a quantifiable marker for knee instability. PubMed and Medline searches using the terms ‘knee’, ‘osteoarthritis’, ‘unstable’, ‘instability’, ‘stability’ and ‘buckle’ were performed to identify relevant studies. Papers were reviewed in full, with their references used to identify additional sources.

Self-reported instability

The importance of patient-reported outcomes and subjective assessment has become increasingly clear in the pre-operative assessment for arthroplasty. Several commonly used patient-reported outcome measures (PROMS) look at functional components of knee health (e.g. Western Ontario and McMaster Universities Osteoarthritis Index (WOMAC), Oxford Knee Score (OKS), Knee Outcome Survey – Activities of Daily Living Score (KOS-ADLS)), but it is only the OKS and KOS-ADLS that specifically question subjective instability. With answers scored on a five- and six-point Likert scale, they ask respectively: ‘have you felt that your knee might suddenly “give way” or let you down?’; and ‘to what degree do each of the following symptoms affect your level of daily activity’ with ‘giving way’, ‘buckling’ or ‘shifting of the knee’ as options. The
self-reported incidence in patients awaiting knee arthoplasty has been shown to be as high as 72%. While earlier work has looked at subjective instability as a co-variable in knee arthritis, more recent work has looked directly at the association between subjective instability and objective measures of assessment. These measures will be discussed in turn.

**Anterior cruciate ligament**

While work has been performed identifying the deficient nature of the anterior cruciate ligament (ACL) in OA, its relationship with subjective instability has not been established. The anterior cruciate ligament is known to be the primary stabilizer of the knee in anterior translation of the tibia against the femur. Traumatic ACL rupture is known both to cause instability in the knee and to predispose to OA. OA is known to cause degeneration and rupture in the ACL. Macrophscopic ACL deficiency of any aetiology has been observed in 6% to 22% of OA knees at TKA. However, histological appearance of the remaining ACL has been found to be abnormal, even in the macroscopically normal ACL. Worse macroscopic and histological appearance has been associated with severity of arthritis, higher BMI, increasing age and increased coronal deformity. Varying abnormalities have been found in the ACL of OA knees, including myxoid degeneration, vascular proliferation, chondroid metaplasia, cystic changes and reorientation of fibres, with more significant abnormality found in the posterolateral bundle in comparison with the anteromedial. Correlation between ACL deficiency and OA scoring systems has been mixed with no association found with OKS, but a lower Knee Society score found with ACL deficiency.

While extensive work has examined both the macroscopic and macroscopic appearance of ACL in OA and found the OA ACL to be frequently damaged, no studies were found assessing the association between symptomatic instability of the OA knee and macro- or microscopic condition of the ACL. No reports of intra-operative posterior cruciate ligament (PCL) structure or function in relation to OA and instability have been found.

**Joint laxity**

One proposed difference between subjectively stable and unstable knees has been joint laxity - the ‘looseness’ of the joint; however, the evidence does not support this suggestion. It has been hypothesized that increased laxity in the joint would more likely result in a feeling of instability. While joint laxity is routinely assessed in any knee examination, quantitative examination has been assessed using stress radiographs, isokinetic dynamometry, non-invasive image free navigation and under anaesthetic using bone-anchored computer navigation. These methods allow a repeatable measurement of joint laxity in a static situation to be assessed. Dynamically, varus-valgus (V-V) movement during walking has been assessed using optoelectronic gait analysis, in particular analysing the stance phase between foot strike and the point of maximum weight acceptance. However, despite an expectation of increased V-V laxity in subjectively ‘unstable’ in comparison with ‘stable’ knees, none of these studies has found any connection.

**Muscle strength, power and activation**

A further hypothesis as to the cause of subjective knee instability is that it is a consequence of decreased muscle strength in comparison with subjectively stable knees. Several methods exist to quantitatively assess muscle strength and varying results have been found with regards to muscle strength comparison between ‘stable’ and ‘unstable’ knees (Table 1). When measuring power independent of function, no difference was found between quadriceps strength between ‘stable’ and ‘unstable’ individuals with knee OA in several small studies. While weaker quadriceps muscle power was found in ‘unstable’ versus ‘stable’ knees in several studies, none were statistically significant. However, in a larger study of 283 well-matched individuals (191 ‘unstable’ versus 92 ‘stable’ OA knees), subjectively unstable knees were found to be significantly weaker in quadriceps extension in comparison with subjectively stable knees when examined using an isokinetic dynamometer at 60 degrees/second and normalized for patient weight. It is notable that in this larger study the unstable group contains 5% more women, is slightly older, more painful and with a longer duration of symptoms; while none of these parameters reached statistical significance individually, the combined effect of these factors may have had an influence on the outcome. In a study of 388 patients undergoing TKR, Fleeton et al showed no association between quadriceps strength post-operatively at six weeks and six months following surgery and the persistence of pre-operative knee instability. However, when the more functional stair climb test is used, reduced power - calculated as a function of weight, speed and height of stair climb - was found to be an independent predictor of instability post-operatively.

A lack of muscular co-contraction may contribute to instability. This hypothesis has been tested when comparing OA to healthy knees, but only in small subgroup analysis comparing stable and unstable OA. Contradictorily, while greater co-contraction in the vastus medialis and medial hamstring was found in a symptomatically unstable group, greater co-contraction was also identified in a symptomatically stable group.
While many studies failed to reach statistical significance, it is noteworthy that all had non-statistically significant weakness in the unstable compared with stable group, suggesting an association of weaker quadriceps in individuals with symptomatic knee instability. Some confounding factors, such as the effect of pain and the influence of gender, need further exploration; although an association potentially exists, whether quadriceps weakness is a cause or an effect of the instability is unclear. Further studies involving knee power measures and muscular co-contraction are warranted to clarify potential links and mechanisms of instability.

**Proprioception and postural control**

One hypothesis is that instability in the OA knee is due to impairment in either proprioception or postural control, with the patient unable to clearly identify the position of, and thereby control, the knee joint in space. However, this hypothesis has not been proven.

Impaired proprioception was not associated with self-reported instability in 283 patients when tested with regards to passive knee flexion sensitivity; however, the same group did find that impaired proprioception was associated with the retention of instability at two years. Instability was found not to be associated to the ability to perform a one-legged unsupported balance in 284 patients with knee OA, once confounding variables of BMI, pain, muscle strength and range of active flexion were removed in the regression analysis. Further, no difference was found between strength training and a combination of strength and proprioception training in reducing the incidence of instability in a randomized controlled trial of 159 patients.

In a study examining vibration sensation in OA, 14 of 16 knee OA individuals were found to experience feelings of instability during a high stepping task, attributed to a reduced vibration perception. Overall, proprioception does not appear to be associated with subjective instability nor does proprioceptive training improve symptoms.

Normal proprioception in the subjectively unstable knee may lead to patient recognition of feelings of buckling and instability that may or may not lead to falls, leading to the voluntary reduction of activity. Three studies reported results of differing physiotherapy intervention in knee OA individuals with and without knee instability using self-reported knee instability as their stratification point. Outcomes of these studies were limited to qualitative self-reported function with regards to instability. While two studies showed no improvement in outcome with additional training for proprioceptive feedback in addition to strength training alone, the third reported a subgroup analysis showing that for those individuals with already good quadriceps strength and instability, the addition of stability training did improve outcome. This may suggest that while quadriceps strength alone is not the cause of the instability, and proprioception is not lost, the strengthening of those abilities may allow individuals to better control their instability - in a disordered and unpredictable joint, increased muscular strength may be required to prevent buckling and giving

### Table 1. Published literature relating to muscular strength and instability in the knee

| Author, date         | Study size | Measurement protocol                                                                 | Study design                                                                 | Result                                                                 |
|----------------------|------------|--------------------------------------------------------------------------------------|-------------------------------------------------------------------------------|------------------------------------------------------------------------|
| Schmitt et al, 2008  | 52         | Isokinetic dynamometer, isometric, knee flexed to 90°, electrode monitoring,         | Comparison of strength between stable and unstable individuals                | No relationship between muscle power and instability                    |
| Schmitt and Rudolph, | 20         | Isokinetic dynamometer, isometric, knee flexed to 90°, electrode monitoring,         | Baseline data taken between stable and unstable patients                      | No statistical difference between stable and unstable group            |
| Knop et al, 2012     | 283        | Isokinetic dynamometer, flexion and extension at 60°/second, normalized for         | Regression analysis for multiple variables in patients with OA instability vs  | Reduced muscle strength associated with self-reported instability      |
| Skou et al, 2014     | 100        | Isokinetic dynamometer, isometric, 60° flexion, normalized for body mass, quadriceps only | Regression analysis for multiple variables between OA patients looking at knee confidence | Association between worse knee confidence and lower quadriceps power |
| Farrokhi et al, 2015  | 53         | Isokinetic dynamometer, isometric, 60° degrees flexion, normalized for body mass, quadriceps only | Baseline data taken between stable and unstable individuals                  | No statistical difference between stable and unstable group            |
| Gustafson et al, 2016 | 35        | Isokinetic dynamometer, isometric, 60° flexion, normalized for body mass, quadriceps only | Baseline data taken between stable and unstable individuals                  | No statistical difference between stable and unstable group            |
| Fleeton et al, 2016  | 388        | Handheld dynamometer mounted on a jig, isometric, 60° flexion, normalized for body mass, knee flexion and extension | Pre- and post-operative testing between stable and unstable knees undergoing TKR, regression analysis for multiple variables | No association between muscle strength and groups of stable, unstable and resolving instability |
way that may otherwise be prevented by intrinsic joint stiffness.

**Knee joint stiffness**

Stiffness, defined and measured as the moment required to produce an angular rotation at the knee, is a defining characteristic of OA of the knee, which has also been investigated as a factor in stability in the OA knee in both the frontal and sagittal plane, non-weight bearing and while walking.

Passive mechanical stiffness in the frontal plane at 20° of flexion was found to be reduced for those with symptomatic instability in a study of 73 patients with medial knee OA, leading the authors to hypothesize that increased V-V stiffness was a mechanism for stability. Moreover, those with self-reported instability have been characterized as walking with reduced sagittal plane stiffness. While these appear to be the only studies examining the effect of stiffness on knee stability, there appears to be a clear pattern of reduced stiffness in both sagittal and coronal plane associated with subjective instability in the OA knee.

**Other Factors Identified during Gait analysis**

Several parameters of gait have been found to differ between subjectively stable and unstable knees. While extensive investigation has been performed comparing normal and OA gait, until recently less attention has been given to the differences between ‘stable’ and ‘unstable’ knee OA gait. Three studies have commented on walking speed, all noting that those OA patients with instability in the knee walk with slower self-selected speed in comparison with those with no reported instability.

Increased knee flexion range of motion during stance phase is noted by two studies but no agreement is found with regards to knee flexion angle at heel contact. Internal contact mechanics of the knee has been assessed in three studies through the use of dynamic stereo radiography. A three-dimensional bony model of the knee joint was created from a CT scan and matched with high frequency bi-planer radiographs of the knee taken during treadmill walking to determine the internal joint motion and contact points. Comparisons were made between healthy individuals and those with knee OA and subjective instability during downhill treadmill walking. OA individuals were noted to have greater V-V movement in the weight acceptance phase compared with controls, as well as a decreased flexion range of motion. It had been hypothesized that instability in the OA knee would result in increased movement of the tibia with respect to the femur during loading in comparison with healthy controls; however, this was not found to be the case, with no significant differences found between groups. However, the medial tibiofemoral contact point was found to move a greater distance and at greater velocity in those with OA and ‘instability’, compared with those with OA and no ‘instability’ and to those without OA, with no differences found between controls and those with OA and no ‘instability’. Variability, defined as the average of the standard deviations at each recorded time point across weight acceptance phase, was examined for both knee joint rotation and tibiofemoral contact point. Anteroposterior (AP) contact point mobility was higher in the OA unstable group compared with the ‘stable’ OA and control groups, while ‘stable’ OA patients exhibited the least stance phase sagittal plane variability with ‘unstable’ OA patients being the most variable. Gait in subjectively ‘unstable’ knee OA differs from the subjectively stable with slower self-selected pace, increased knee flexion range of motion during stance and greater knee joint internal contact point variability.

**Disease severity**

The influence of OA severity of subjective instability has been examined in a study of 192 patients with OA, in which instability was stratified using a four-point Likert scale, with the results dichotomized. Severity was assessed using joint space narrowing, osteophyte formation, and the Kellgren and Lawrence (K-L) scale from an AP and lateral standing radiograph. No associations were found between severity of OA and subjective instability. An association between worsening varus alignment and instability has been shown in one small study but not replicated in another. Taken together, and to date, there is no evidence of a link between disease severity and instability.

**Discussion**

While it may seem intuitive that ‘instability’ and increased laxity go hand in hand, several studies have found that neither V-V laxity in non-weight-bearing conditions nor during movement are related to ‘instability’. Similarly, it may be reasonably postulated that a knee is unstable due to inadequate strength. However, most studies looking for associations between strength and instability are not conclusive. In the one study that found a significant difference, it was unclear whether weakness was a cause of ‘instability’ or the effect of reduced activity caused by the ‘instability’. As impaired proprioception has not been shown to be associated with ‘instability’ in the OA knee, it must be concluded that...
the cause of symptomatic instability may be found in aetiologies other than V-V laxity, muscular weakness and one’s sense of joint position.

Stiffness is one of the cardinal features of OA and it is therefore unexpected to find that stiffness in the knee is reduced in ‘instability’. However, the reduced passive stiffness in the knee in the frontal plane in the first few degrees around the neutral axis and in the sagittal plane during walking presents a picture of a knee that is more difficult to control, lacking the restraining characteristic that stiffness brings to perturbation under small load. This seems consistent with individuals with ‘unstable’ knees walking slower, with increased knee flexion and with an increased movement and variability of contact points within the knee.[7,8,20] Contradictorily, these characteristics, taken together, point to a knee that, while not objectively loose with regards to supine ligamentous laxity nonetheless displays the characteristic of a joint lacking passive control during gait.

The lack of correlation between K-L grading and symptomatic instability points to an aetiology beyond simply bone and cartilage damage, to a whole joint process. The ACL in the OA knee has been shown to be absent or damaged frequently in OA knees; while the correlation between subjective instability in OA and ACL status has not been examined, it is clear to see how a dysfunctional or absent ACL may contribute to a more internally mobile and unstable joint. Further work to identify both the relationship between macroscopic ACL condition and symptomatic instability, but also to characterize the biomechanical function of the ACL in the OA knee, is warranted.

Limitations and future developments

One clear limitation in any study examining instability in the knee is lack of consensus over the definition of subjective instability. As discussed previously, several methods of description exist, but all are based upon a single questionnaire giving an ordinal result, often transformed dichotomously. Validation of this method is not possible due to the lack of comparison; however, it is widely accepted throughout the literature. Due to the nature of biomechanical studies, several involve small participant numbers. While their conclusions remain valid, it is important, particularly with regards to subgroup analyses, to interpret their results with caution due to sample size and multiple comparisons.

To overcome the shortfall of self-reported instability and to determine a more quantified measure, biomechanical characteristics of ‘unstable’ movement must be identified. To make this of practical, clinical relevance requires the development of a portable device capable of demonstrating small, rapid movements at the knee during movement. Recent work has been ongoing to develop portable gait analysis devices using accelerometers, with some success, while the use of accelerometer in examining varus thrust has well-established foundations. Computational analysis techniques such as Fast Fourier Transform or wavelet filtering of accelerometer data allow exploration of frequency domain in knee movement. This will allow the exploration of fast knee vibration and oscillation that is suggested by reduced stiffness and increased contact point variability in the subjectively unstable knee in comparison to stable. While no successful results of these technologies have yet been demonstrated, the known characteristics of instability in the knee lends itself this method and therefore work should be directed towards such practically useful technologies.

There is potential for more understanding between the behaviour of the restraining ligaments of the knee and knee instability. Devices to determine in vivo ligament stiffness are required to understand healthy, pathological and OA ligamental contributions to knee stability.

Conclusion

The subjective sensation of instability and buckling in the OA knee has been linked to reduced stiffness, reduced walking speed, increased flexion and increased internal contact point movement variability in comparison to the stable OA knee. Work should be undertaken to assess the impact of ACL function on subjective instability in the OA knee. It appears that the subjectively unstable OA knee exists in a state of unpredictability and reduced stiffness both during walking and while static, in contrast to the classic symptom of OA stiffness. Practical methods of quantifying this reduced stiffness should be pursued in an effort to quantify knee instability in patients with knee OA in the orthopaedic clinic.

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