

Clinical Terminology for Describing Knee Instability

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Sports Medicine and Arthroscopy Reviews, 4:313-318, 1996

Great confusion has existed for many years over the terminology for describing knee instability. This confusion is enhanced when some doctors use clinical terminology for describing knee instability, whilst others attempt to describe it in biomechanical terms. It is the purpose of this paper to clarify many of the misconceptions about the definition, understanding and classification of knee instability.

DEFINITIONS

Laxity: is the measured amplitude of joint movement within the constraints of its ligaments.

Physiological Laxity: implies no ligament is stretched pathologically.

Pathological Laxity: means a ligament has been stretched by injury.

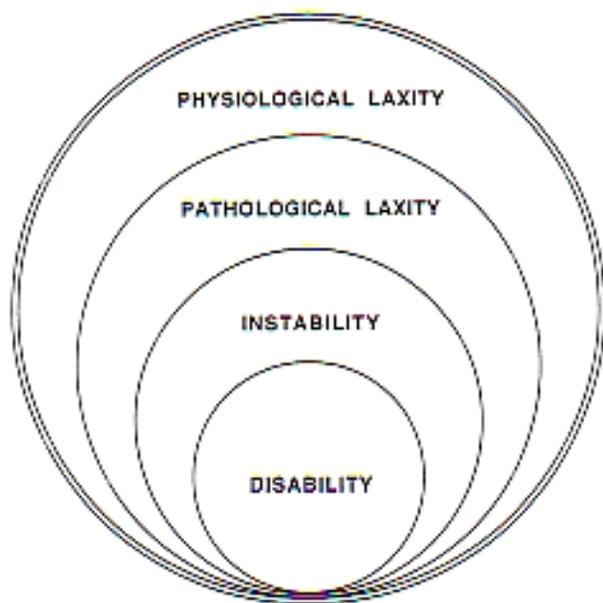
Instability: is a complaint by a patient that they lose single leg stance because the joint subluxes due to pathological laxity.

Disability: is instability that interferes with the required function of the knee.

Knee instability should be described in clinical terms. True Instability is a condition that occurs when the joint translates out of its ligamentous and muscular boundaries and the articular surfaces partially or totally disengage. The patient then loses control of single leg stance. Patients state that the knee gives way, and some understand and feel exactly what happens and state that the knee subluxed or dislocated. They may say the knee collapsed, it fell away, or it went out. One particular patient described in broken English that her knee was a “something nothing” knee. I believe that this term- “the something nothing knee” adequately describes what happens to the patients with true knee instability. Something happens (i.e., subluxation); then there is nothing under them.

There is, however, another type of “giving way”, which occurs in knees that are ligamentously intact. In these situations the knee causes pain, for example, a tear in the meniscus, and one gets reflex inhibition of the quadriceps mechanism and jack-knifing occurs as the muscles relax, patients feel their knee bending and describe this as giving way. This is not true knee instability. An experienced examiner needs to distinguish between these two types of giving way (Fig 1)

Figure 1. Relationship of laxity to instability



PHYSIOLOGICAL ANATOMY

The knee is separated into three physiological anatomical compartments: the patellofemoral compartment, the medial compartment, and the lateral compartment.

The Ligaments of the Lateral compartment

The lateral compartment is bounded medially by the anterior cruciate ligament, laterally by the lateral capsular ligament, the ilio-tibial tract, and the fibula lateral ligament and posteriorly by the arcuate complex and the posterior capsule. These structures are all supported by the iliotibial band, the popliteus muscle, the biceps muscle and an extension of the semimembranosus muscle called the oblique popliteal ligament.

The Ligaments of the Medial Compartment

The medial compartment is bounded medially by the deep third of the mid-capsular ligament, the medial collateral ligament, and the posterior oblique ligament and laterally by the posterior cruciate ligament. Anteriorly, these compartments have extensions of the medial capsule as well as patellotibial and patellofemoral expansions, as well as the patella tendon.

PROPRIOCEPTIVE CONTROL

Each of these ligaments and all of the muscles have been demonstrated to have

proprioceptive fibres (8). The ligaments and the tendons blend in local proprioceptive reflex arcs giving rise to dynamic protection as well as static protection. The cerebrum receives neurologic input from the moving joint, from joint position sensors in the skin, the capsule and the muscle spindles (8), and from the eyes and ears. Neurologic feedback to the protagonist and antagonist muscles prepares them for complicated movements such as cutting (2), sidestepping, and twisting. Pre-programming and training of all these movements have been shown to be of great benefit in the guarding of normal joint motion and preventing injury. These mechanisms can fail when some movement is unpredicted or when there is contact from an external force, or when the foot is entrapped in either cleats or a pot hole. Abnormal forces are placed both on the static and the dynamic structures and subsequent injury can occur. This injury is dependent upon the innate strength of the static stabilisers, as well as the dynamic stabilisers (proprioceptors) to recognise the abnormal forces and to come into control to protect the joints from them.

An example of the proprioceptive control of muscle is the semimembranosus muscle. Four of the seven extensions of the semimembranosus muscle, around the posterior and medial aspects of the knee tend to provide more sensory than motor function. The direct arm into the posterior aspect of the medial tibia and its anterior extension definitely have motor function because of the size of their tendons. The attachment into the medial meniscus into the posterior oblique ligament also has a motor function but also serves a sensory function. The expansion over the popliteus muscle and the oblique popliteal ligament as well as the expansion down over the medial collateral ligament all are well placed to receive proprioceptive messages from the lateral, posterior and medial capsular structures of the knee.

PATHOGENESIS OF KNEE INJURY

When any joint moves, particularly the knee joint, there is a possibility of abnormal stresses being applied to that joint. The knee joint contains an intricate mechanism of intra articular ligaments and menisci (1). The large lever arms that are placed on the knee are restricted distally, but less so proximally. The proximal ball-and-socket hip joint can move in any direction. The ankle joint is however unrestricted only in an A-P plane; therefore abnormal stresses placed through the foot and the ankle can have a marked effect on the long lever arm of the tibia. These forces can be counteracted somewhat by body mechanics above the hip joint, thus alleviating stress areas within the knee.

Yet, this protective mechanism can falter if the static and dynamic stabilizers about the knee

fail. Each of the six degrees of freedom of the knee is threatened, some more than others. Hyperflexion injury is uncommon in the knee unless there is a barrier in the popliteal fossa, but hyperextension injury is frequent. Abduction and adduction are also common, particularly from extraneous forces such as those encountered in motor vehicle accidents and body contact sports. Rotation injuries are common in all aspects of sport and are often associated with intrinsic noncontact forces.

In any injury mechanism, combinations of forces are usually applied to the knee, and therefore it is extremely rare to sustain pure injuries in one of these degrees of freedom. By this, although pure abduction, pure rotation, pure hyperextension injuries occur, there is usually also another motion (abduction, rotation, hyperextension or abnormal flexion) occurring at the time. This is one of the reasons for the confusion and misconceptions in relating clinical experiences from one observer to the other. Researchers have been experimenting in the laboratory and on the sporting field for many years in an attempt to correlate the mechanisms of injury with the specific injury to particular ligaments and thus to describe and correlate the findings at clinical examination.

The essence of understanding the pathogenesis of the knee is to be able to directly relate the findings demonstrated clinically in the examining room to the patient's history of injury; then, by identifying each injured anatomical structure, we can reconstitute that ligament and thus re-establish the static stability of the knee.

CLINICAL CORRELATION AND CLASSIFICATION

In order to have a clinical correlation and a classification system, it is essential to first define the normal knee. The knee joint is a variable joint. Much has been written about the various anomalies that can occur in the menisci, the cruciate ligaments, the bone and the articular cartilage. By definition, a normal knee is the contralateral knee, providing it has not been injured previously. This may sound elementary; however, many joints vary from patient to patient in their degrees of looseness. This term is used because there has been great debate, particularly over the last 15 years, over knee laxity where the lax knee has been interpreted as being an unstable knee. It may be that the lax knee is more prone to injury than the tight knee (10).

The extreme ends of tightness and looseness, respectively, are classified under arthrogryposis and hyperelastosis. Patients with hyperelastosis have totally dislocatable joints, but commonly do not complain of knee instability as the dynamic factors and

proprioceptor control enable them to function in activities of daily living. There is no doubt that varying degrees of genetic penetrance of these conditions can occur, and we all have seen patients with gross laxity in their joints, particularly those with recurvatum who have by all intents and purposes a grossly “abnormal” anterior draw or posterior draw sign. They have marked rotary laxities and it is possible to perform the physiological lateral pivot shift test on them to no pathological complaint.

My experience has demonstrated that there is inherent weakness in some knees, as I have patients with knee injuries with a strong family history of knee injuries, including twins with ruptured cruciate ligaments. Another manifestation of predisposition to injury in some knees is the patients who injure both knees. Thus, one can see a fundamental flaw in attempting to classify and correlate knee instability if one does not use the opposite uninjured knee for comparison.

The classic article by Hughston et al. (6) describing the straight and rotatory instabilities of the knee was based on a clinical study of a consecutive series of patients who had documented acute trauma to the knee. Early clinical examination took place mostly on the sidelines and pathology was documented at surgery. The ligaments injured were correlated specifically with the clinical findings. Many readers did not understand that this classification (which was based on the posterior cruciate ligament being intact) described medial and lateral compartment joint injuries including rotary instability (the abnormal twisting that occurred giving rise to the specific injuries). The importance of this classification and its methodology is that, with modern day arthroscopic techniques, which ignore the treatment of various capsular injuries, it will never be possible to repeat the dissections necessary to ascertain each capsular and intra-articular structure damaged in a particular injury.

Another major difficulty in understanding clinical terminology for describing knee instability is that it is extremely unusual to get a pure instability that fits exactly the various classifications employed (12). Therefore, this article describes the various combinations of signs and symptoms that patients complain of when their knee gives way. Clinically, I believe this is a possible alternate terminology.

ACL SYNDROME

Anatomy & Function

The ACL has three bands with a fourth extension to the posterior cruciate ligament called the intercruciate ligament. The function of the ACL is to control the screw-home mechanism of

the knee, which it does this by restricting anterior translation and also limiting internal rotation during the last 30° of flexion.

Historical Perspective

The ACL syndrome is the most common instability seen, particularly in the sporting fraternity. A survey of the literature reflects our understanding of this syndrome. Before the recent research and writings of O'Donoghue (11a), the anterior draw test was the gold standard in diagnosing ruptures of the ACL. Slocum and Larsen (13) introduced the concept of medial and lateral capsular laxity and recommended testing the anterior draw in external and internal rotation. Galway and Macintosh (5a) described the "lateral pivot shift" as something occurring on the lateral side of the knee during pivoting, and that something was a shift. Hughston et al. (6) clarified and described this shift as a subluxation of the lateral tibial plateau on the lateral femoral condyle. This subluxation is an alteration in acceleration, which this is known as a "jerk". Other tests such as the Lachman test and the dynamic extension test (4), have been subsequently described and introduced into our clinical terminology.

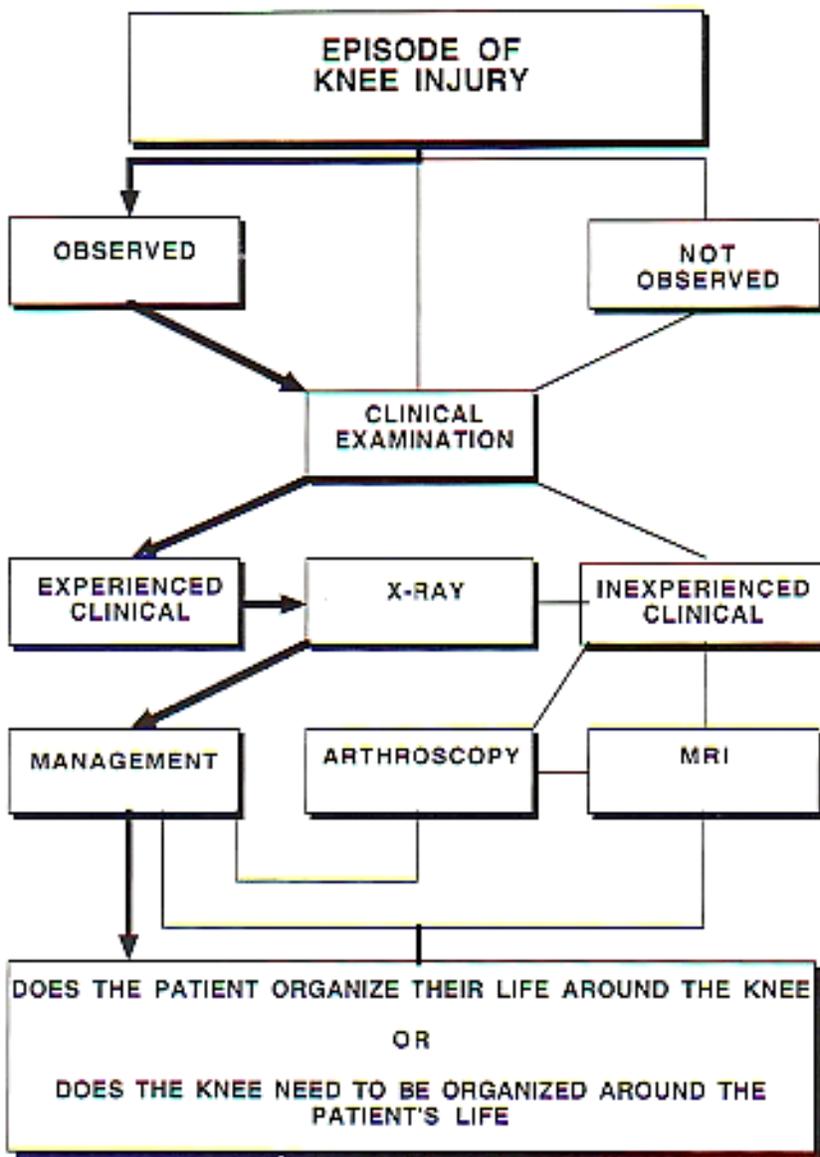
Trillat (14), in France, led the European school synchronously with the English-speaking expansion of knowledge and had an excellent classification and understanding of the instability of the knee.

Symptoms of the ACL Syndrome

The main symptom of the ACL syndrome is "giving way". Patients will complain that their knee gives way particularly if they are not running in a straight line. The history is predominantly that of a twisting injury when sidestepping, cutting, jumping or landing. This is associated with a snap, crack, or pop, and is accompanied by severe pain and, in 90% of cases, immediate haemarthrosis. The clinical examination is described as being extremely difficult, (especially to the inexperienced practitioner) and the x-rays are usually negative. The diagnosis is rarely made initially in inexperienced examiners. Experienced practitioners can make the diagnosis immediately (fig.2). Radiologically, the lateral capsular sign, or Second fracture, is evidence of the avulsed mid-third lateral capsular ligament, which is diagnostic of a rupture of the ACL.

Differentiating recurrent episodes of true giving way (the "something- nothing" variety) when cutting, landing, and sidestepping from patella instability is a strong diagnostic aid. Most anterior cruciate ruptures can be highly suspected and even diagnosed over the telephone.

Figure 2. Management paradigm for knee injury



Other symptoms of pain, locking, swelling, crepitus and weakness are usually not the patient's chief complaint, but are due to the secondary effects of the pathologic laxity and the recurrent instability. Each time the knee gives way, there is subluxation, shearing, and compression, which can cause tears of the menisci, further stretching of the capsular ligaments, and damage to the articular surface. It is interesting that in our survey, patients with a ruptured ACL that had become attached to the posterior cruciate ligament had less intraarticular damage than patients without this finding. It is also interesting to note that the amount of intraarticular damage correlates extremely well with the number of episodes of giving way.

Detecting the signs of the anterior cruciate syndrome are demanding (11). It takes 6 months for a resident or fellow seeing a large numbers of knees to become proficient at eliciting the lateral pivot jerk test. Thus, the Lachman test is usually taught as it can be measured with an arthrometer. The quality and quantity (7) of the Lachman test must be considered, however. In patients where the anterior cruciate ligament has healed onto the posterior cruciate via the intercruciate ligament may have a hard, not soft, end point. In these cases it is important to measure the quantity of the laxity.

A proficient examiner can elicit the lateral pivot jerk (i.e., failure of the screw-home mechanism) in all cases of a ruptured ACL. Because the ACL is the medial ligament of the lateral compartment, the ACL syndrome affects the lateral compartment. The lateral capsule ligament is always torn and the topography of the lateral tibial plateau enhances the subluxation especially when the lateral meniscus is nonfunctional. If the major function of the ACL is the direct control of the screw-home mechanism, which occurs during the last 30° of full extension, one can understand the pathomechanics of the jerk and subsequent subluxation. At 30° short of full extension, the iliotibial band becomes an extensor of the knee. The biceps act synergistically with the function of the ACL, which tends to prevent anterior translation of the lateral compartment as well as internal rotation of the lateral compartment, and hence causing external rotation and posterior placement. When the ACL is ruptured, this external rotation and anterior brace fails to function and it is therefore possible for the lateral tibial plateau to sublux anteriorly and rotate internally.

The dynamic extension test (4) is useful in the acutely knee as it is a “no-touch” technique that measures the anterior tibial translation by active quadriceps contraction. All other tests (9) are modifications of assessing the failure of the screw-home mechanism. The knee is examined in various positions, which allows some examiners who have difficulty with the lateral pivot jerk test to develop alternate techniques of assessing the failure of the mechanism.

Anterolateral rotary instability is another way of describing biomechanically the events that occur. Minor degrees of anterolateral translation occur with disruption of the lateral capsular ligament only; the full-blown ACL syndrome requires the ACL to be compromised.

Straight anterior instability is explained when the ACL, PCL, medial capsular ligament and the posterior oblique ligament are ruptured. When these medial capsular and posterior oblique ligaments are ruptured you have an anteromedial rotary instability. The diagnosis of straight anterior instability is made when performing the Lachman and jerk tests: Anterior subluxation

of the medial and lateral tibial plateaus is equal. The knee, in fact, dislocates.

The other instability associated with ACL is the posterolateral instability, which occurs when the arcuate complex and the popliteus are injured and the posterior capsule is stretched. This instability is also difficult to assess. The two major tests are the rotational recurvatum test performed at full extension and the external rotation test performed at varying degrees of flexion. Failure to recognise this instability is one of the reasons why we have failure of many attempts at anterior cruciate reconstruction by simply replacing the intraarticular section of the instability.

CONCLUSION

I believe if one can truly understand the ACL syndrome, then all other instabilities of the knee can be assessed and learned by relating to this syndrome. Training in clinical examination of the knee is difficult and has become a fairly low priority with the development of many arthroscopic techniques. The arthroscope cannot fully assess the unstable knee so it is vitally important to those that are practising as knee surgeons, that they take time to learn clinical examination and all of the intricate tests described. It is necessary to have exposure to a fairly large patient base and repetitive and comparable testing needs to be taught and learned. The problem of precise testing is a difficult obstacle; there is interobserver error, which needs to be decreased as more practice is achieved (11). Instrumented testing has been also demonstrated to have interobserver error, it should be encouraged while assessments are continually made.

Many difficulties in clinical examination can occur because the various types of instability do not always present as a simple isolated instability. It is, therefore, important for us to understand and to communicate in the clinical terminology already described.

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