Knee Ligament Tests

What Do They Really Mean?

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It is little wonder that the casual reader of knee orthopedic articles is left somewhat bewildered about ligament function and the clinical laxity tests used in assessing knee stability. In the past there has been wide disagreement about these issues among many knowledgeable knee surgeons. Newer clinical and biomechanical information has helped to clear up some important areas of disagreement, but correct diagnosis of knee instability is not a simple matter. Knee examinations must be very precise and meticulous to diagnose the straight and rotatory laxities. Some laxities are obscured and others are very straightforward. Still, there are practical biomechanical concepts that govern the successful interpretation of clinical laxity tests. This paper describes two of these concepts that are common to all laxity tests. Once recognized, the concepts clarify much prior confusion and help the examiner correctly interpret results of the laxity examinations.

The two biomechanical concepts or rules are as follows:

1. Ligaments can be divided by their function into primary and secondary stabilizers. For every plane of knee stability or motion, there are primary and secondary ligament stabilizers that must be recognized.
2. The amount of laxity in the knee depends on the force applied. If you apply a large pull or push (drawer test) to the knee, you get more movement (laxity). This point is actually quite simple. Unfortunately, during a clinical laxity examination, you actually apply only small forces compared to what the knee experiences with activity. Therefore, the knee may appear stable during a laxity test but may not be stable under the higher forces of activity.

We will expand on each concept individually and then put both together to explain what really is happening during the clinical laxity examination.

CONCEPT ONE: PRIMARY AND SECONDARY LIGAMENT RESTRAINTS

It is well known that ligament and capsular structures work as a system, interdependent and related one to another. Functional stability is provided by 1) the passive restraints of the ligaments, 2) joint geometry, 3) active restraints generated by the muscles, and 4) joint compressive forces that occur with activity and that force the joint together. These factors combined provide both static and dynamic stability.

There are limitations in the examination used to measure knee stability after injury. During the clinical laxity examination of the knee, we attempt to isolate and test only the passive restraints provided by the ligaments and joint geometry, not the active restraints or joint compressive forces. Thus only a limited evaluation of the factors that provide joint stability is possible by the clinical laxity tests. Also, the laxity tests are highly subjective as to the amounts of joint opening and the types of motion that are produced. This explains the frequent disparity between the laxity examination and true in vivo joint function. Laxity tests alone do not provide a reliable prediction of functional stability, a problem that we will discuss later. Muscle forces, neuromuscular coordination, and the other factors all determine whether there is functional stability or functional disability. Despite all of these limitations, the laxity examination still remains the primary means by which the clinician diagnoses ligament injuries.

Another concern about the use of examinations for stability is that many authorities still do not agree on which ligaments are being tested during the standard laxity tests. This leaves the clinician confused as to what the laxity tests really show. Over the past five years we have conducted experimental biomechanical studies in order to resolve these conflicts. We measured for the first time the actual restraining force that develops in individual ligaments during displacements of the knee.[1,4,6,7] This measurement shows which ligaments are really the primary "workers" and which are the secondary "helpers" in resisting a certain joint displacement or in providing stability. The main function of knee ligaments is to limit the motion between the tibia and femur. They do this by providing a restraining force whenever they are stretched. The total joint restraining force is simply the sum of the contributions from the individual ligaments, excluding weight-bearing forces and geometry constraints. Thus, the relative importance and function of a single ligament can be assessed in terms of the percentage of the total restraining force it provides.

We have concluded from our studies on ligament restraining forces that, for each of the planes of knee motion, only one or two ligaments act as the primary passive restraints. The other ligaments provide only a secondary restraint, but they do assist in providing stability. The preponderance of restraining forces will, however, be provided by the primary restraints. They limit joint motion and protect the secondary restraints from being stretched. Both the primary and secondary restraints work together to provide stability. Thus the designation of a secondary restraint does not lessen the importance of its role in this regard. The diagnosis and surgical treatment must be geared to restoration of all the ligament restraints, although the primary restraint must be given increased respect.

Let us examine the concept of primary and secondary ligament restraints, using the example of the anterior drawer test. We measured ligament restraining force during anterior and posterior clinical drawer tests on 12 knees from young human cadavers.[1] At 30 degrees of knee flexion on the anterior drawer test, the anterior cruciate accounted for 85 percent of the restraining force. All other ligaments combined provided only a weak secondary restraint of 15 percent of the restraining force.

CONCEPT TWO: ACTIVITY FORCES VERSUS CLINICAL EXAMINATION FORCES

Now consider an apparent clinical paradox. On occasion the anterior drawer test is only slightly positive, despite known rupture of the anterior cruciate. How can this occur if the anterior cruciate ligament is the primary restraint? Does a slightly positive anterior drawer test really indicate that true in vivo stability exists? These questions may be answered by examining Figure 1. In this biomechanical test on an intact cadaveric knee, the anterior drawer is first performed up to 5 mm of displacement, producing a restraining force of just over 100 lb (450 newtons). The joint is returned to a neutral position, and then a posterior drawer test is conducted to 5 mm of posterior tibial displacement and back to neutral. The arrows on the curves show the direction of loading, and the slight difference in the first curve for anterior and posterior loading is due to the viscoelastic properties of the ligaments. The curves define the anterior-posterior joint stability provided by the ligaments. The solid line in Figure 1 represents a knee in which all of the ligaments are intact. The dashed line shows a repeat test of the same knee after only the anterior cruciate ligament is cut. The ligament is sectioned through a patellar tendon-splitting approach so as not

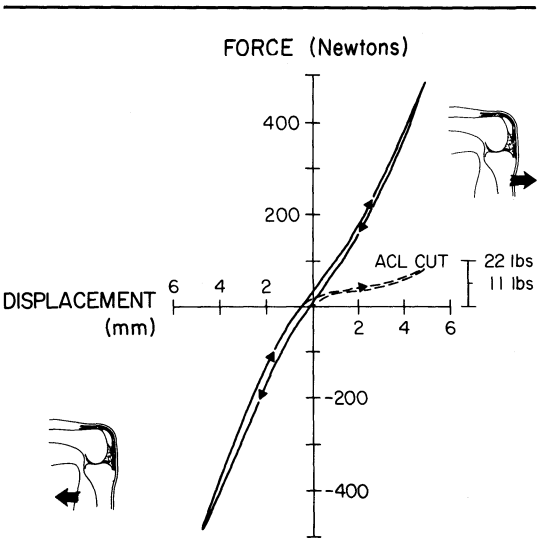

Fig. 1. Drawer test. The solid line represents a knee with all the ligaments intact. (ACL—anterior cruciate ligament.) (Reproduced with permission of author and publisher.[8])

to disturb the other ligaments. Following section of the anterior cruciate ligament, the anterior stability of the joint is markedly decreased. This shows the loss of the primary restraint. Much less force is required to produce an anterior drawer, which indicates the significant decline in anterior stability of the knee. The anterior restraining force, however, does not drop to zero, indicating the remaining presence of the secondary restraining ligaments.

It is important to realize that, during clinical drawer tests, only a small force is applied, often in the range of 20 lb (90 newtons). This is much less than the forces acting on the knee ligaments during in vivo activity, when they may easily be subjected to 100 lb or more in strenuous activities.[2,3] As shown in Figure 1, an 11-lb anterior pull in this particular knee produces only a 3-mm increase in the anterior drawer sign. This is barely detectable, yet anterior stability at higher in vivo forces is markedly affected. A much greater laxity would occur. In this case, the clinical drawer test would not predict the decline of true in vivo stability. Even a low muscle resistance in acute cases can also easily block the clinical drawer test and thus the true stability of the joint would not be ascertained.

When a primary ligament restraint is lost, the weaker secondary restraints must then resist the large in vivo forces. Often the secondary structures are not designed to resist such forces and therefore may stretch. When this occurs, the clinical laxity test results become more positive.

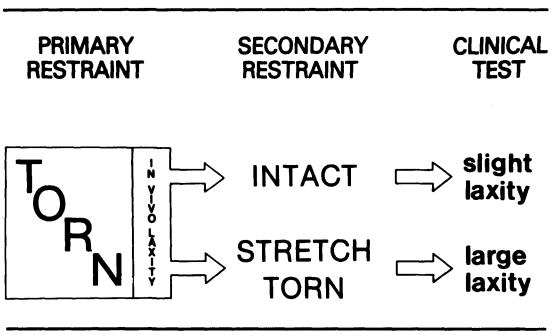

Fig. 2. The clinical laxity test may not indicate true in vivo stability or the status of the primary ligament restraint. (Reproduced with permission of author and publisher.[8])

LIGAMENT RESTRAINTS AND KNEE FORCES WORKING TOGETHER

It thus appears that the amount of laxity seen during a clinical test may be limited by the weak and inefficient secondary restraints once the primary restraint has been ruptured. The clinical laxity test therefore may not indicate true in vivo stability nor true function of the primary restraint. This interesting dilemma is summarized in Figure 2. It is our hypothesis that, if the primary restraint(s) to one plane of instability is (are) torn, then an in vivo laxity under large activity forces can be expected. Whether this represents a functional disability or not will be discussed. The results of the clinical laxity tests after rupture of the primary restraint depend on the integrity of the secondary restraints, which, in fact, contribute little to long-term stability. As Figure 2 indicates, if the secondary restraints are intact, the clinical test, which is normally performed under low forces, misleadingly shows only a slight laxity even though the primary restraint is gone. The test is, therefore, an inadequate predictor of future joint stability. If the secondary restraints stretch with time or are initially torn, the clinical laxity tests will show greater laxity.

The concepts of primary and secondary restraints apply to all of the various clinical laxity tests.[5,6] For example, the posterior cruciate ligament is the primary restraint for posterior drawer stability and provides 95 percent of the restraining force. For the medial side of the knee, the medial collateral ligament provides about 78 percent of the restraint to medial opening at 25 degrees of knee flexion. For the lateral side, the lateral collateral ligament provides 69 percent of the restraint to lateral opening at 25 degrees of knee flexion. With knee extension, the posteromedial and posterolateral parts of the capsule tighten and provide a greater restraining force, although the collaterals are still the primary restraints. The anterior and posterior cruciate ligaments are initially secondary restraints working together, resisting medial and lateral joint opening. However, after the collateral and capsular structures are torn, full reliance for medial or lateral stability is placed on the cruciate ligaments. If there is gross laxity, particularly in extension, then they are also damaged.

Isolated ligament ruptures seldom, if ever, occur for reasons already discussed.[5–7] Therefore, a certain amount of disruption in the secondary restraints can be expected any time the primary restraint is ruptured. After ligament rupture and healing, if the primary restraint is not restored, then progressive stretching and increasing laxity can be expected in the secondary restraint. The clinical laxity examination of the knee in the initial months after injury and surgical repair is an inadequate predictor of future joint stability. Even if the primary restraint were nonfunctional, the secondary capsular structures and scar tissue could easily block any laxity in the clinical stability tests. However, with activity, the integrity of the primary restraint will be required to prevent laxity, and, in the face of inadequate healing, the weaker secondary restraints (whether healed or not) will be subjected to deleterious forces.

Joint surfaces may also be affected following ligament injuries. Joint arthritis may be a silent event, with the symptoms of pain, swelling, and disability occurring in some cases only after it is too late to limit activity and protect the joint. Functional stability of the knee is a primary goal after ligament injury. However, functional stability is often unpredictable by the clinical laxity examination, for reasons already discussed. Also, functional stability may be short term, relying on superb muscle control, which some individuals have but others do not. Age and loss of conditioning may lead to deterioration in adaptive muscular control and, therefore, loss of joint stability. Any quick or sudden activity may subject the joint to displacements too fast for the muscles to counteract and purposely control. For these reasons it appears that joint deterioration can occur if there is any abnormal laxity on clinical examination; that is, if there is any indication that the passive ligamentous system is compromised. Rehabilitation should be directed towards improving strength and endurance and, perhaps primarily, to increasing neuromuscular coordination and agility. Even so, joint deterioration may still occur despite complete rehabilitation.

An inadequate predictor of the success of a surgical procedure or recovery from a prior significant injury (too often applied to the athlete) is his ability to "return to play." We must be aware of early joint

arthritis that will progress through the years. In certain cases, we have performed arthroscopy one to two years after major ligamentous injury or surgical repair in the active or athletic individual. We have been impressed with the frequent finding of joint surface deterioration in certain individuals despite lack of any initial symptoms. In these knees, although functional stability exists and the athlete can perform adequately, joint wear still occurred. In such cases the athlete should be warned that the joint is at risk for further deterioration even though one cannot predict how long it will take. Serious discussion is required to permit each individual to decide the extent of his future athletic activities.

SUMMARY

In summary, newer biomechanical concepts clarify human function, the clinical laxity tests, and functional stability of the joint. Biomechanical studies show that one or two ligaments provide the primary passive restraint for each plane of knee stability, with the remaining ligaments having a secondary, helping role. Correct interpretation of clinical laxity tests and surgical treatment of instability require understanding of this differentiation. Knowledge of the interaction between the primary and secondary restraints during the clinical laxity tests allows for more accurate interpretation of the extent of ligament injury. Weak secondary restraints may initially allow little laxity to be demonstrated in the clinical laxity tests. However, the secondary restraints will eventually stretch out and cause a greater laxity. In an acute knee injury, "a little laxity is a lot" and should be considered as serious. In knee injuries, an exact diagnosis of injury is required. This often requires examination under anesthesia and arthroscopy to define the extent of ligament damage. Functional stability of the knee is a primary treatment goal after ligament injury but the stability may be short term if it relies on muscle control alone without the fine-tuning action of the ligamentous system. Abnormal laxity on clinical examination means increased risk for joint wear, cartilage deterioration, and arthritis on a long-term basis. Close follow-up after ligament injuries, adequate rehabilitation, and correct advice on allowable activities are important treatment concepts after any serious ligament injury.

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