

ANTERIOR CRUCIATE LIGAMENT INJURY

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INTRODUCTION

Anterior cruciate ligaments (ACLs) have always been torn by sporting folk at football, rugby etc, but with the boom in the skiing industry abroad and with dry slopes sprouting everywhere at home, this knee injury is now freely available to most of the population at any age.

This "open access" has been well taken up, causing an increase in numbers which in turn is causing the injury to appear as if it were a new one, like a new disease. Such diseases generate gossip, misunderstanding and general chaos unless adequate information is available, and now that treatment is more or less standardised, this article attempts to provide that information.

WHAT DOES THE ACL DO?

It is impossible to do a total knee replacement without cutting the ACL. Next time you see someone who has a new knee, pull their tibia forward when they're sitting and relaxed: the majority will demonstrate anterior laxity, but do not complain of giving way. Why is this, when a large percentage of natural knees with torn ACLs give way? The answer is in the shape of the joints: a replacement is very congruent – a roller in a trough (see Figure 1), whereas the natural joint has a roller or wheel in a dish (medial side) and a wheel on a dome on the lateral side (Figure 2), this latter configuration having no inherent mechanical stability. The ACL maintains the lateral femoral condyle and tibial plateau in their correct

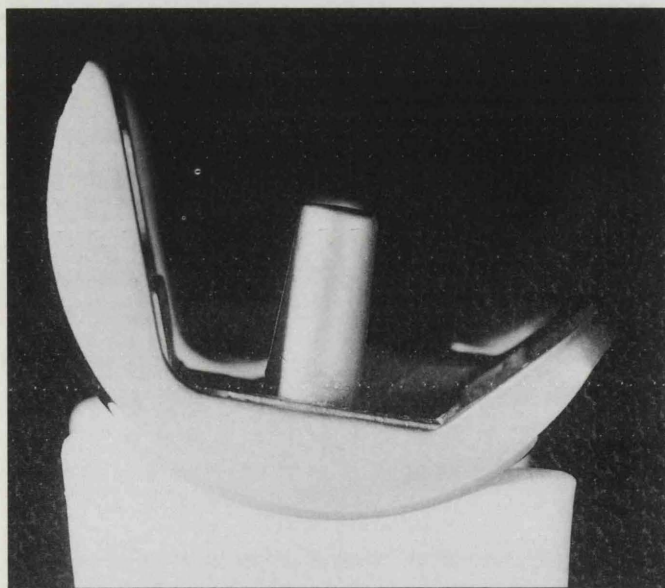


Fig. 1 Lateral view of a knee replacement, showing the "roller in a trough" configuration

relationship during flexion, such that without it the lateral tibia rotates (subluxes) forward in front of the femur under load. Understanding of the clinical condition is, therefore, helped by thinking of the ACL as limiting internal rotation of the tibia on the femur, rather than limiting anterior movement of the same, although it does the latter.

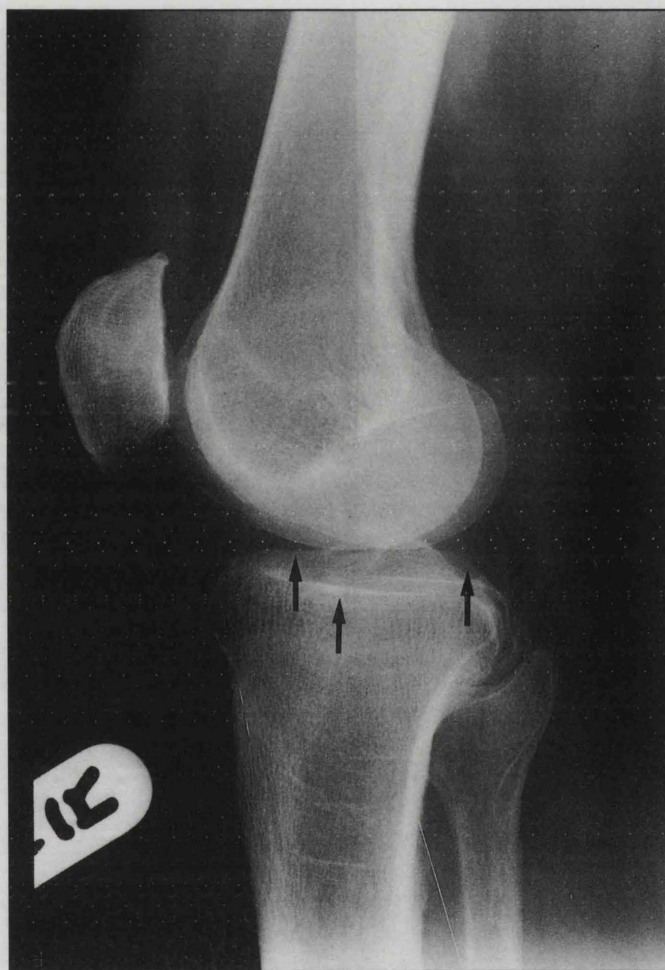


Fig. 2 Lateral XR of knee: the lower arrow points to the concave (dished) medial condyle; the upper arrows to the convex (domed) lateral condyle

WHAT HAPPENS WITHOUT IT?

The forward subluxation of the lateral tibial plateau when straightening the knee at around 30 degrees of flexion, as described above, causes a "jerk", reproduced in reverse on bending the knee when the lateral tibia reduces. This jerk, demonstrated by the test described below, is the cause of the giving way or instability (a symptom). It is also referred to as the lateral pivot shift (because American athletes say their knee "shifted when they pivoted"), and antero-lateral rotatory instability, but the term "jerk" best describes the symptom and the sign.

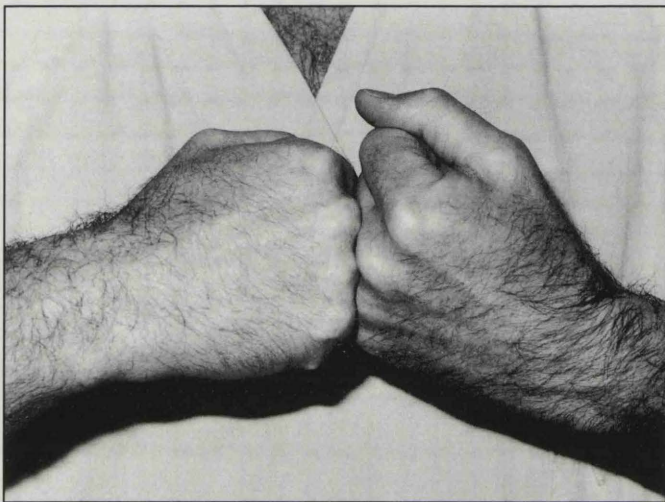
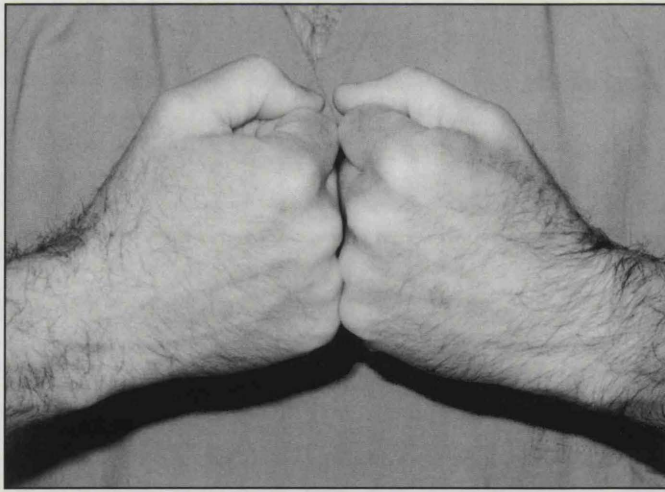


Fig. 3 & 4 The "knuckle" sign.
How some patients describe their knees giving way

Patients often, but by no means always, describe their jerk by showing their opposing knuckles twisting round each other, or can be made to by opposing their knuckles and asking them what happens when their knees give way (see Figures 3 and 4).

Not every ACL deficient knee will give way: about 75% do, this probably depending on anatomy, eg the degree of doming of the lateral plateau and its degree of backward slope, muscle strength of the quadriceps and hamstrings, and proprioceptive sense.

Remember, there are other causes of the knee giving way. In descending order of frequency they are: patello-femoral pain/maltracking; meniscal tears and loose bodies; and other ligamentous injuries. Deficiency of the ACL is the commonest cause of ligamentous instability, but still less common than the first two groups above.

THE INJURY

This is either an external rotation force to the tibia combined with a valgus force on the flexed knee, which can damage the medial collateral ligament and medial meniscus also, or an internal rotation and hyperextension force. Remember, it limits internal rotation and would therefore be tight, and the top of the intercondylar notch then "cuts" it in hyperextension.

The tear can be complete, or more commonly partial.

PRESENTATION AND DIAGNOSIS

Acutely, after an injury, with a swollen painful knee, the swelling is due to blood. The majority of knee haemarthroses (75%), have an ACL rupture (and 50% have a meniscal tear). The history may be of an injury as outlined above. The knee usually lacks full extension i.e. is "locked", and this may be due to painful spasm from the irritating blood, or genuine locking due to a torn and jammed meniscus. The tests for ACL deficiency will not be elicited in a painful swollen knee, and therefore the treatment of choice for such knees, if facilities permit, is an examination under an anaesthetic and arthroscopy with adequate lavage and suction.

Chronic cases may describe the initial injury, and may have subsequent injuries with haemarthroses each time, as a little more of the ligament is torn. Giving way, usually with change of direction or on rough uneven ground, with a jerk, and the demonstration of the knuckle sign (Figure 3) is virtually diagnostic, although not always described.

Demonstration of the jerk is the single most important examination test. This will not be elicited in a painful or unrelaxed knee, and may be minimal in an acute injury. In chronic cases, correlation of the jerk produced in the knee with the patient's "that's what happens when me knee give way" is pathognomonic. In practice, a convincing jerk sign is often only demonstrated under GA, hence a warning to trainee surgeons: the arthroscopic or MRI demonstration of an absent ACL allied with giving way does not necessarily mean this is the cause of the instability. The other causes outlined above should be considered, plus the much less common postero-lateral instability, which, when combined with an absent ACL, will abolish the jerk sign, and replacing the ACL on its own in such a patient will not be a cure.

Jerk sign The patient lies supine, head relaxed on couch, arms by side, totally relaxed. The knee is flexed to 45 degrees held by one hand on the heel internally rotating the foot, and the other on the upper lateral shin exerting a valgus force at the knee. The knee is extended. At about 30 degrees the jerk occurs; the valgus and internal rotation exaggerate the jerk⁽¹⁾. The reverse jerk is elicited on flexing the knee again (see Figures 5 and 6).

Other tests of laxity (laxity is a sign, instability a symptom). First, some more anatomy. The ACL is not one ligament taut throughout the arc of flexion – it cannot be. To act as a truly isometric structure through the arc of movement, it has to consist of several parts acting as a

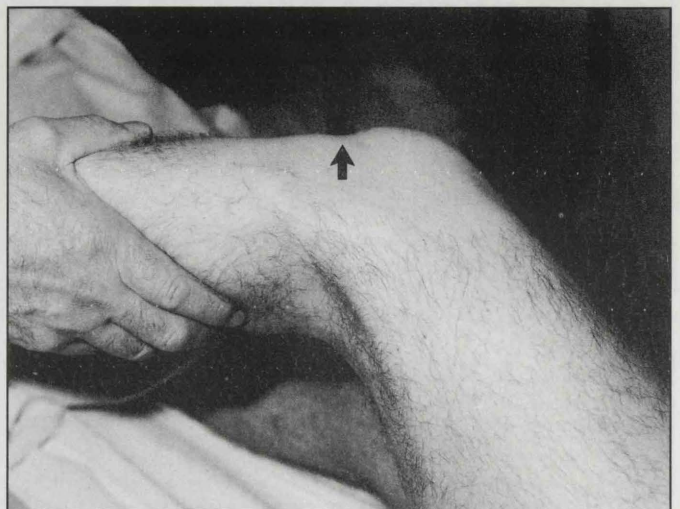


Fig. 5 The "jerk" test: the arrow shows the normal sulcus below the patella with the knee at 45 degrees of flexion

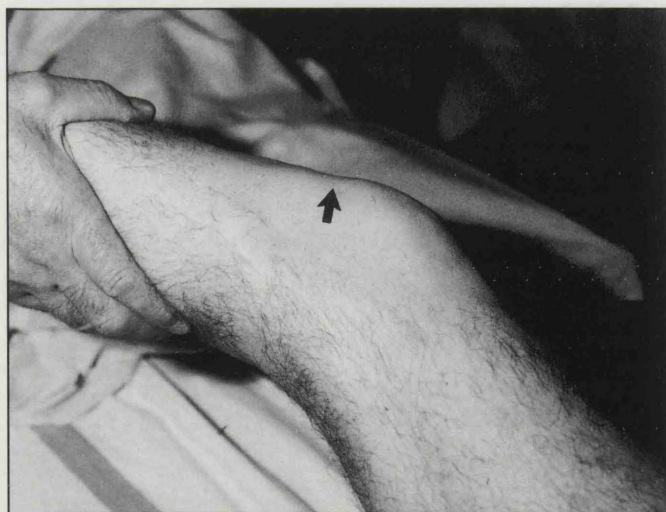


Fig. 6 The "jerk" test: the arrow shows no sulcus as the lateral tibia subluxes forward when extending the knee more than 30 degrees. Clearly this is easier to feel and see in action than to illustrate!

continuum. It has a 3cm attachment to the tibia and somewhat less to the femur. Generally speaking, the antero-medial part is taut in flexion and controls the anterior drawer sign, and the postero-lateral part is taut in extension, and is tested by the Lachman test bringing the tibia forward on the femur with the knee just off extension, ie enough to get one's hands around the bones⁽²⁾. The postero-lateral part is also said to control rotation and hence the jerk sign, explaining why the anterior drawer can be positive (antero-medial part torn) but the jerk and Lachman negative (postero-lateral part intact!), and the knee functionally stable. In the reverse situation, a functionally unstable knee can demonstrate a negative anterior drawer sign.

WHAT HAPPENS IN TIME TO THE ACL DEFICIENT KNEE

This is controversial: most studies have involved patients treated at "knee hospitals" and thus were seeking treatment and were usually sporting. A poor outcome is noted in such cases, with a tendency to tear the medial and lateral menisci, the former more than the latter, and to develop progressive arthritic symptoms and signs, these correlating with the giving way. One study showed that only 11% at five years following injury were taking part in "strenuous activity", and they were exceptional athletes⁽³⁾, but another showed that 38% at 14 years had no sporting limitations!⁽⁴⁾. Degeneration was noted in the latter study, associated with heaviness, varus deformity, and meniscectomy.

What we really need to know is what happens to the knees which are ACL deficient but don't give way, and those which do but are not functionally limiting. Patients with such knees have not presented themselves to orthopaedic clinics in the past, and hence we do not know the answer. They are presenting now, mostly because they are told that they will never ski again without an ACL. We cannot at present give them a prognosis regarding later degeneration.

WHAT DOES THE OPERATION DO?

In the rare case when bone is avulsed from the tibia, acute surgery to replace and fix the bone is successful. Otherwise, acute repairs do not work because two intra-articular "mop-ends" do not heal when sutured. Surgery after acute injury is indicated to make the diagnosis, and to rule out or deal with

associated injuries, particularly meniscal tears and other ligamentous injuries. The most common associated ligamentous tear is of the medial ligament, which can be repaired if grossly torn or avulsed, or treated by cast immobilisation if not gross. Early reconstruction of a torn ACL when there is inflammation and loss of movement of the knee gives bad results, due to fibrosis forming in the knee⁽⁵⁾.

In non-acute cases, there have been three basic types of corrective operation since the jerk sign was "rediscovered" in the early 1970s: extra-articular reconstructions using the fascia lata, to prevent internal rotation of the tibia; ligament substitution with carbon fibre, Goretex or Dacron grafts; and ligament substitution with autogenous structures, usually part of the patellar tendon.

The extra-articular procedures of MacIntosh worked in the short term, but at least a third failed after five years⁽⁶⁾. Carbon fibre worked also, but the hope that it would act as a scaffold for tissue growth was not fulfilled, and when the ligaments failed the carbon particles made a mess of the knee. Dacron grafts had fewer complications but also tended to slacken off after three years⁽⁶⁾. The central third of the patellar tendon plus a patellar bone plug was first used in the 1960s, but was not successful due to poor fixation of the bone plug, lack of understanding about isometric positioning, and lack of subsequent extension of the knee.

Current procedure

The current and widespread operation as modelled by Messrs Shearer and Gascoigne utilises a free strip of the patellar tendon, about 1cm wide, with plugs of patellar and tibial tubercle bone at either end (Figures 7 and 8). Isometric positioning is intended to reproduce the anatomy as nearly as possible⁽⁷⁾, and the fixation uses an "interference" screw jamming the bone plugs solidly in their respective femoral and tibial tunnels (Figure 9). Such fixation is planned to be absolutely rigid, and patients can therefore mobilise with full weight bearing without a brace, unless there is a hitch.

It may be best to point out here that the graft "dies" when it is taken, and loses strength to a low point at between eight and twelve weeks, and then regains strength to a maximum at around ten to eleven months. Clearly rehabilitation must be progressive, with no contact sports till ten months at the earliest, no running or twisting till about six months, and no quadriceps strengthening exercises more than lifting the weight of the leg for the last 30 degrees of extension for three months, as this is when maximum tension and stretching occurs in the ligament⁽⁸⁾.

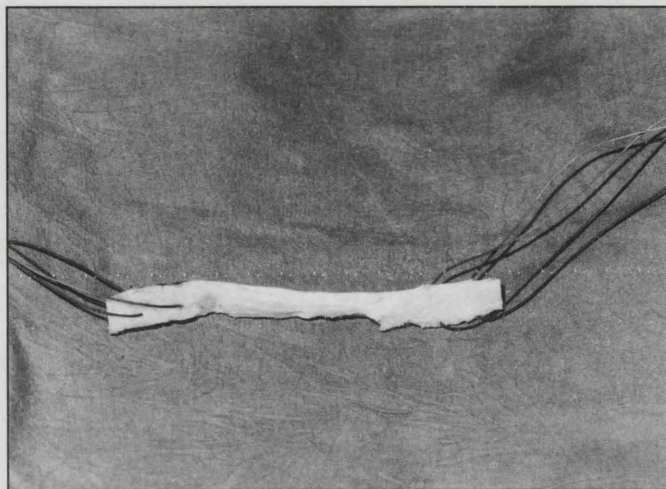


Fig. 7 The free "bone-tendon-bone" graft, with sutures through the bone plugs

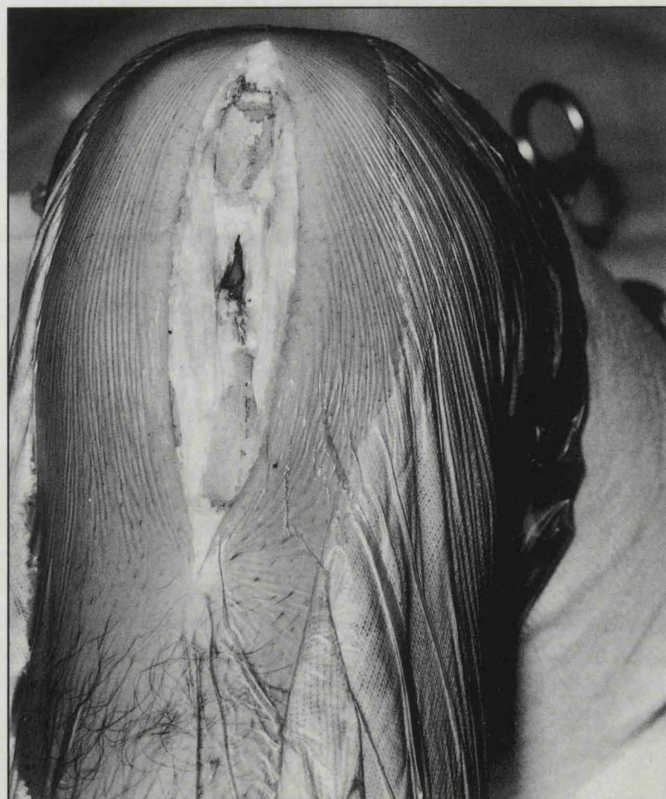


Fig. 8 The knee after graft harvesting. The patellar graft is 8mm deep, less than half the total patellar thickness

The problem is that a 1cm graft can never properly replace a structure which has a 3cm attachment. It does appear to work, as you can see on any Match of the Day, but what will Mr Gascoigne's knee be like in five or ten years' time? On reflection, perhaps we would do better to enquire this of Mr Shearer's knee. Whatever, there is only one decent study with five year follow-up, which shows that the procedure does give excellent results in that period, but that the grafts do slacken with time. One in twelve shows "rotatory instability" at five years, compared with none at two years⁽⁹⁾. As there is no absolute correlation between giving way and laxity following reconstruction, (i.e. it can work when it looks as if it hasn't), it is thought that the procedure increases proprioception in some as yet ill-understood way.

Personal experience since 1993 shows that this procedure is a winner over that short period and is now one of my top ten orthopaedic procedures. I have had one failure out of 21, and in that case the failure occurred gradually over three years. Two have returned to regular Rugby League, one to Rugby Union and football, and three to football.

More recently some specialist knee surgeons have started using multiple strands of hamstring tendons for the graft. Why? We have good five year results now for the "bone-tendon-bone" graft, as reported above, and it takes more than five years to obtain these from a new procedure! Most surgeons (Tom, Dick and Harry?) are now performing the bone-tendon-bone operation. Surely, therefore, you could not possibly think me cynical to suggest that to stay on top of the heap they have to do something different?

SUGGESTED TREATMENT PLAN

Acute tears When diagnosed clinically it is best to have examination under anaesthesia and arthroscopy to rule out or treat associated injuries as detailed above. In practice, these are often left to "settle down and see". Assisted weight-



Fig. 9 Two "interference" screws, holding the bone plugs firmly in their tunnels

bearing with crutches, and immobilising (unhinged) splints may be used for a few days only. There is no place for complicated hinged knee braces. They are expensive (over £200) and cannot really be expected to control sudden rotation of the tibia, the cause of this instability. After adequate rehabilitation (see below) patients either will or will not need an operation. A braced ACL deficient knee which does not give way is an expensive illusion causing prevarication!

Subsequently they should all have adequate physiotherapy to strengthen the quadriceps and hamstring muscles. In theory, the lateral hamstrings can prevent the subluxation of the lateral femoral condyle forwards at low speed. When muscle power is back to nearly normal, agility and neuromuscular coordination should be worked on. Clearly this depends on the patient, but if they are not prepared to work hard now, it is likely they will not behave after a reconstruction.

Having obtained good muscle power and control, or at least tried to, I tell patients to try their sport again or give it up, i.e. modify their activities. If they are still giving way, during either sport or everyday activities, on a regular basis, with a "jerk", they are advised to have a bone-tendon-bone replacement, with an excellent chance of abolishing the instability and returning to sport if that is desired. The rider is as outlined above: we do not know the ten year results.

Those with proven ACL deficiency but no giving way are not damaging their knees as far as we know, and would be better not having the operation. This probably applies to those with knees giving way infrequently and not functionally troubling them.

Acknowledgement

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