The risk of anterior cruciate ligament rupture with generalised joint laxity

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We assessed hyperextension of the knee and joint laxity in 169 consecutive patients who underwent an anterior cruciate ligament reconstruction between 2000 and 2002 and correlated this with a selected number of age- and gender-matched controls. In addition, the mechanism of injury in the majority of patients was documented. Joint laxity was present in 42.6% (72 of 169) of the patients and hyperextension of the knee in 78.7% (133 of 169). All patients with joint laxity had hyperextension of their knee. In the control group only 21.5% (14 of 65) had joint laxity and 37% (24 of 65) had hyperextension of the knee. Statistical analysis showed a significant correlation for these associations. We conclude that anterior cruciate ligament injury is more common in those with joint laxity and particularly so for those with hyperextension of the knee.

Many factors predispose to rupture of the anterior cruciate ligament (ACL). These may be intrinsic non-changeable factors such as physiological joint laxity, female gender, or size of the femoral notch and extrinsic, potentially changeable factors such as the type of footwear, playing surface and inherent conditioning skills and co-ordination.\(^1\) The exact mechanism of this injury is still debated\(^1\) with as many as 70% of ACL injuries being caused by a non-contact event.\(^1\)

The evidence supporting joint laxity and knee hyperextension as predisposing factors is conflicting. After following up 139 professional football players, Nicholas\(^4\) concluded that laxity of the joint predisposes to ligament injuries. Godshall\(^5\) disagreed, in a series of ‘growing’ athletes, and Moretz, Walters and Smith\(^6\) concluded, in a series of 155 football players, that laxity did not predispose to ligament injuries. However, Boden et al\(^1\) showed a strong correlation between hamstring flexibility and ACL rupture after analysing data from 100 ruptures. At the Hunt Valley consensus conference for the prevention of non-contact ACL injury, it was judged that the relationship between ACL ruptures and joint laxity remained unresolved.\(^2\)

We have observed that a high proportion of patients with ACL injuries have excessive joint laxity and hyperextension of the knee. We therefore assessed the degree of hyperextension in the uninjured knee in 169 patients before they underwent ACL reconstruction. We then correlated these findings with a selected group of age- and gender-matched control subjects who did not have an ACL injury and discuss the implications of our findings.

Patients and Methods
We performed 356 ACL reconstructions between 1996 and 2002. Since making the observation of joint laxity and knee hyperextension, we decided to prospectively assess the degree of joint laxity and knee hyperextension in all patients with a symptomatic ACL injury before they underwent reconstruction.

Between 2000 and 2002, 169 patients underwent an ACL reconstruction for isolated ACL injury. Our tertiary referral practice meant that by the time the referral was made, the patients had already been through a failed rehabilitation programme and were still symptomatic. There were 137 men and 32 women, whose ages ranged between 18 and 34 years. All had their laxity scored by Beighton, Solomon and Sokoline’s\(^7\) method while their knee hyperextension was measured at the time they were listed for surgery. In addition, the mechanism of ACL injury, whether by a contact or non-contact event, was also recorded in the majority of cases.

The Beighton score ranges from 0 to 9 and is derived by assigning one point each for: 1) hyperextension of the metacarpophalangeal joint of each little finger beyond 90°; 2) ability to touch the volar surface of each forearm with...
the thumb; 3) hyperextension of each elbow; 4) hyperextension of each knee and 5) the ability to place the palm of both hands flat on the ground by forward flexion with knees straight. A score of greater than 6 would indicate hypermobility and increased joint laxity.\textsuperscript{7,9}

Our control group comprised 65 individuals matched for age and gender with no known ACL-related problems or those attending shoulder clinics, or athletes who underwent medical screening at a local football club but who had no knee symptoms. The degree of hyperextension of the knee and the laxity scores were determined in these individuals.

The degree of hyperextension in the contralateral, uninjured knee was assessed using a goniometer. For this, the patient was supine on the examination couch and one limb of the goniometer was applied to the lateral thigh in line with the femur. The other limb of the goniometer was applied to the leg with the centre of the device over the knee joint. The proximal hand which held the goniometer firmly held the thigh down to the bed so that the popliteal fossa was in contact with the couch, while the distal hand hyperextended the knee by lifting the leg from the heel. Values of more than 10° were taken to represent hyperextension.

Heel clearance, as assessed by placing the fingers underneath the heel in question, allowed us to develop a quick screening method. With the patient lying supine and the thigh and popliteal fossa in contact with the couch, the number of fingers which could be placed under the heel was assessed. If the mean width of a digit is 20 mm, and if one can insert two digits or less, the angle at the knee joint should be less than 10° for most adults. It was important for the foot to be in a neutral or dorsiflexed position during this assessment. After development of this technique, we only occasionally needed to use a goniometer in order to assess the degree of hyperextension (Fig. 1).

Table I. Number of injured and control patients with and without joint laxity

<table>
<thead>
<tr>
<th></th>
<th>Laxity</th>
<th></th>
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</thead>
<tbody>
<tr>
<td></td>
<td>Absent</td>
<td>Present</td>
<td>Total</td>
</tr>
<tr>
<td>ACL* injury</td>
<td>97</td>
<td>72</td>
<td>169</td>
</tr>
<tr>
<td>Control</td>
<td>51</td>
<td>14</td>
<td>65</td>
</tr>
<tr>
<td>Total</td>
<td>148</td>
<td>86</td>
<td>234</td>
</tr>
</tbody>
</table>

* ACL, anterior cruciate ligament

Table II. Number of injured and control patients with and without hyperextension of the knee

<table>
<thead>
<tr>
<th></th>
<th>Hyperextension</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Absent</td>
<td>Present</td>
<td>Total</td>
</tr>
<tr>
<td>ACL* injury</td>
<td>36</td>
<td>133</td>
<td>169</td>
</tr>
<tr>
<td>Control</td>
<td>41</td>
<td>24</td>
<td>65</td>
</tr>
<tr>
<td>Total</td>
<td>77</td>
<td>157</td>
<td>234</td>
</tr>
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</table>

* ACL, anterior cruciate ligament

Statistical analysis was performed using the chi-squared test with values for $p < 0.05$ being regarded as significant.

Results

The prevalence of generalised joint laxity in those who presented for an ACL reconstruction was 42.6% (72 of 169). However, the prevalence of generalised joint laxity in the control group was 21.5% (14 of 65). This difference was statistically significant ($p < 0.01$) (Table I).

The prevalence of hyperextension of the knee in those with rupture of the ACL was 78.7% (133 of 169). However, the prevalence of hyperextension of the knee in the control group was only 37% (24 of 65). This difference was significant ($p < 0.001$) (Table II).

The mechanism of injury was only documented for 130 of the 169 patients. For the remaining 39, although there
was information about the injury, the actual events leading to the rupture, whether by a contact or non-contact mechanism, was not clearly recorded. This was mostly in longstanding failed rehabilitators and tertiary referral patients. We noted that 75.4% (98 of 130) of the injuries happened as a result of a non-contact mechanism and that there was no difference between those who had hyperextension of the knee and those without laxity. It therefore appeared that the non-contact mechanism of injury was more common in both groups (Table III).

### Discussion

The ACL can rupture as a result of either a contact or a non-contact injury. The latter is more common, a trend which is reflected in our series. Rupture of an ACL in this way is characterised by an absence of collision, but an awkward, single-leg landing or stopping, or rapid changes in direction, especially lateral movements.

In a contact injury, however, there is a collision with another person, leading to valgus-varus stress and pivoting. Numerous factors can predispose to rupture of the ACL. After the Hunt Valley consensus conference held in Maryland, in 1999, a prevention booklet was published in which factors contributing to non-contact ACL injuries were highlighted. Some factors thought to contribute to ACL injury are shown in Table IV.

Hyperextension of the knee and physiological joint laxity have been highlighted as intrinsic factors contributing to these injuries. However, it has also been judged that the relationship between hypermobility and knee ligament injuries is still unresolved. The literature is divided with regards to the association between joint laxity and ligament injuries, partly because of the inconsistencies in assessing laxity.

The Nicholas technique of grading joint laxity has not been validated while Godshall’s results may be biased, as many of his cohort were skeletally immature, something which is acknowledged in his paper. He commented that his subjects became ‘tighter’ with time. The strongest evidence available is that of Boden et al, who correlated hamstring flexibility and genu recurvatum with an ACL rupture. Their technique of assessing hamstring flexibility and hyperextension of the knee could be laborious in a clinic setting. We used Beighton’s method as it is quick to perform and has been well established in the literature.

In our study, we have provided further, statistically significant evidence to support the association between laxity and hyperextension of the knee and rupture of the ACL, by having a larger sample group. In our series, 78.7% had hyperextension as compared with 37% of controls, who were also at risk of developing an ACL rupture.

We have found sufficient evidence in the literature to suggest that the final pathway of a non-contact ACL rupture could be hyperextension of the knee. We base this hypothesis on the videotaped evidence of athletes who have sustained such an injury. Ireland described a ‘position of no return’ after which the ACL potentially ruptures in about 70 milliseconds. In this position, the back is straight without a lumbar lordosis, the trunk leans backwards with the hip and knee slightly flexed and the foot lands flat on the ground with the centre of gravity behind the knee. This pre-positioning of the trunk initiates events that may lead to an inevitable ligament injury. When there is a change in the sequence of movements in order to regain control, there is an excessive, eccentric contraction of the quadriceps. These events lead to hyperextension of the knee with increased anterior translation of the tibia.

Borsa et al showed that this anterior translation force is greatest when the quadriceps is activated at higher acceleration and with small knee flexion angles. Therefore, when there is pre-existing excessive hyperextension, the knee moves through further in its final arc and generates greater anterior transitory forces. This continued momentum locks the knee into hyperextension and allows the ACL to hit the notch and guillotine itself. Different notch shapes, especially the stenosed A-shaped notch can influence the nature of the ACL injury. Hyperextension of the knee can, therefore, lead to an increasing frequency of ACL rupture by this mechanism. We recognise that there is multifactorial interplay before this final event, which may include the degree of tibial rotation, the actual landing technique, and the ability to initiate hamstring activity.

There is ample evidence to show the intricate relationship between proprioception, increased laxity and joint injury. Loudon, Goist and Loudon showed that this anterior translation force is greatest when the quadriceps is activated at higher acceleration and with small knee flexion angles. Therefore, when there is pre-existing excessive hyperextension, the knee moves through further in its final arc and generates greater anterior transitory forces. This continued momentum locks the knee into hyperextension and allows the ACL to hit the notch and guillotine itself. Different notch shapes, especially the stenosed A-shaped notch can influence the nature of the ACL injury. Hyperextension of the knee can, therefore, lead to an increasing frequency of ACL rupture by this mechanism. We recognise that there is multifactorial interplay before this final event, which may include the degree of tibial rotation, the actual landing technique, and the ability to initiate hamstring activity.

There is ample evidence to show the intricate relationship between proprioception, increased laxity and joint injury. Loudon, Goist and Loudon showed that a person with genu recurvatum has poor proprioceptive control at the terminal degrees of extension. Roberts et al showed that the proprioceptive threshold was elevated, even in an uninjured limb, compared with controls and concluded that there was deficient proprioception in both injured and

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**Table III. Mechanism of ACL rupture for those with and without joint laxity, by number and percentage**

<table>
<thead>
<tr>
<th>Overall</th>
<th>Laxity present (n = 100)</th>
<th>Laxity absent (n = 30)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Non-contact</td>
<td>Contact</td>
<td>Non-contact</td>
</tr>
<tr>
<td>98 (75.4%)</td>
<td>32 (24.6%)</td>
<td>74 (74%)</td>
</tr>
</tbody>
</table>

*ACL, anterior cruciate ligament
† information not available for 6 patients
‡ information not available for 33 patients

**Table IV. Factors thought to predispose to an ACL rupture**

<table>
<thead>
<tr>
<th>Extrinsic (potentially changeable)</th>
<th>Intrinsic (non-changeable)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Strength</td>
<td>Limb alignment</td>
</tr>
<tr>
<td>Conditioning</td>
<td>Physiological laxity</td>
</tr>
<tr>
<td>Shoes</td>
<td>Hamstring flexibility</td>
</tr>
<tr>
<td>Playing surface</td>
<td>Hyperextension of the knee</td>
</tr>
<tr>
<td>Proprioception</td>
<td>ACL size/thickness</td>
</tr>
<tr>
<td>Neuromuscular rhythm</td>
<td>Size and shape of femoral notch</td>
</tr>
<tr>
<td>Acquired skill and co-ordination</td>
<td>Female gender</td>
</tr>
<tr>
<td>Landing techniques, etc.</td>
<td>Hormonal influence</td>
</tr>
</tbody>
</table>

*ACL, anterior cruciate ligament

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uninjured knees. It is, therefore, possible that patients with hyperextension of the knee may have a poor proprioception feedback loop. The poor proprioceptive feedback seen in both hyperextension and increased joint laxity can affect both limbs and reduce the ability to initiate protective reflexes.

By virtue of their joint laxity, individuals may steer themselves towards a high level of competitive sport.24,27,28 The knowledge of an association between hyperextension of the knee and ACL rupture would prove valuable in the prevention of ACL injuries by identifying the at-risk population. A simple clinical test to identify those with hyperextension would allow specific proprioception-oriented retraining exercises. Observation and training by an informed coach would allow specific proprioception-orientated retraining simple clinical test to identify those with hyperextension of ACL injuries by identifying the at-risk population. A knee and ACL rupture would prove valuable in the prevention of ACL rupture and various prevention strategies have been recommended.2,3,29 Those with hyperextension of the knee may be selectively targeted for these retraining exercises as a knee which hyperextends stands a high risk of rupturing its ACL.

No benefits in any form have been received or will be received from a commercial party related directly or indirectly to the subject of this article.

References