PERSISTENT JOINT LAXITY AND CONGENITAL DISLOCATION OF THE HIP

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This paper reports an investigation of the incidence of persistent generalised joint laxity in patients with congenital dislocation of the hip and in normal school children.

Capsular laxity has attracted much attention in studies of the etiology of congenital dislocation of the hip joint. At first it was thought to be an isolated result of mechanical stretching, but it is now usually considered to be part of a generalised joint laxity affecting both axial and limb joints. It seems probable that there are two forms of generalised joint laxity which develop during pre-natal life, one being temporary and disappearing during the neo-natal period, the other persisting into adult life. The temporary laxity is confined to girls and is believed to have a hormonal origin, whereas the persistent laxity may occur in either sex and usually has a familial incidence.

Temporary hormonal laxity—Most interest has been shown in temporary hormonal laxity. Heusner (1902) was the first to discover a greater degree of capsular laxity in the hip joints of female than of male stillborn infants. He did not appreciate its hormonal origin, and Hisaw (1926) first demonstrated a pelvic laxity which was dependent on the female hormones. In immature and in mature guinea-pigs he produced joint laxity by injection of oestrogen and then progesterone. He believed that the responsible agent was a hormone, relaxin, which was produced in the uterus. Recently, Wilkinson (1963) showed that, in immature rabbits, the...
same hormonal laxity also affects the ligaments of the hip and knee joints, indicating a generalised distribution. Such laxity greatly facilitated the experimental production of atraumatic dislocations in young female rabbits. Chapple and Davidson (1941) developed the hypothesis that foetal laxity was the result of maternal hormones passing through the placenta to produce a greater effect on the female foetus. Andrén and Borglin (1961) suggested that such laxity might be intensified when the foetal liver has a reduced capacity to conjugate oestrogen. Hormonal laxity disappears soon after birth when the maternal and placental hormones are removed.

Persistent laxity—Lorenz (1920) described persistent generalised joint laxity in children with congenital dislocation of the hip, but he considered it to be a rare association. Massie and
Howorth (1951) attached greater etiological significance to this form of joint laxity and noted that it affected many joints, particularly those of the feet. Such generalised joint laxity is a feature of certain rare, genetically determined connective-tissue disorders, such as Ehlers-Danlos syndrome (McKusick 1956), but it is more often an isolated finding. It commonly affects more than one member of the family (Finkelstein 1916, Key 1927, Carter and Sweetnam 1958) and its association with recurrent dislocation of the patella and recurrent dislocation of the shoulder has been previously reported (Carter and Sweetnam 1960).

**METHOD**

In this research persistent generalised joint laxity was diagnosed when more than three of the following tests were positive, both upper and lower limbs being involved: 1) passive apposition of the thumb to the flexor aspect of the forearm (Fig. 1); 2) passive hyperextension of the fingers so that they lie parallel with the extensor aspect of the forearm (Fig. 2); 3) ability to hyperextend the elbow more than 10 degrees (Fig. 3); 4) ability to hyperextend the knee more than 10 degrees (Fig. 4); 5) an excess range of passive dorsiflexion of the ankle and eversion of the foot (Fig. 5).

Each child was examined by both authors. The control group consisted of 145 schoolboys and 140 schoolgirls, aged six to eleven years, in two primary schools in north-west Kent. The patients consisted of a random series of fifty-eight girls and fourteen boys with congenital dislocation of the hip, aged five to fourteen years, who had attended The Hospital for Sick Children, and of nineteen boys from the Royal National Orthopaedic Hospital, who were added to increase the number of male patients. Five of the girls and four of the boys had a first degree relative (parent or sibling) affected; these are considered separately and we have added to them a girl and two boys, who also had a first degree relative affected, but were not in the random sample.

**RESULTS**

**Normal controls**—The total prevalence of joint laxity in the control children was nineteen in 285 (7 per cent). There was no significant difference between the two sexes, nor was there any clear indication of any change in prevalence between the younger and older children. In normal adults the prevalence of joint laxity on these standards is less, due to normal loss of joint mobility with increasing age. In over half the control children all five tests were negative and three-quarters had not more than one joint affected. The findings are summarised in Tables I and II.

**Patients with no first degree relative affected**—Of girls with congenital dislocation of the hip, just under a third (fourteen out of forty-eight) showed joint laxity. This is five times the proportion in the controls. Of the boys, over three-quarters (nineteen out of twenty-six) showed laxity. This is not only much higher than the proportion of controls, but significantly higher than the proportion of female patients who were lax. The findings are summarised in the upper part of Table III.

**Patients with a first degree relative affected**—The findings in the fourteen cases with a parent or sibling affected are shown in the lower part of Table III. In the boys the proportion with joint laxity (five out of seven) was high, as it was in the non-familial cases. In the case of the girls, however, the proportion with laxity (four out of seven) suggests—though the series is small—that laxity is commoner than in the non-familial cases.

**Patients' parents**—In eighteen cases in which a patient with congenital dislocation of the hip had generalised joint laxity the parents were also examined. In five of nine male cases one or other parent also had obvious joint laxity: in three instances it was the father and in two
the mother who showed laxity. In all nine female cases one or other parent had obvious laxity: in four instances the mother was affected and in four the father; in one instance both parents were affected.

In certain individual families the interaction of familial generalised joint laxity and other predisposing factors, acetabular dysplasia and breech malposition, is clear. Four such families are illustrated in Figure 6. In family A the father was dead but the mother gave a clear

**TABLE I**

<table>
<thead>
<tr>
<th>Age (years)</th>
<th>Number of joints lax</th>
</tr>
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<tbody>
<tr>
<td></td>
<td>0</td>
</tr>
<tr>
<td>6-7</td>
<td>24</td>
</tr>
<tr>
<td>8-9</td>
<td>14</td>
</tr>
<tr>
<td>10-11</td>
<td>37</td>
</tr>
<tr>
<td>Total</td>
<td>75</td>
</tr>
</tbody>
</table>

**TABLE II**

<table>
<thead>
<tr>
<th>Age (years)</th>
<th>Number of joints lax</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>0</td>
</tr>
<tr>
<td>6-7</td>
<td>15</td>
</tr>
<tr>
<td>8-9</td>
<td>39</td>
</tr>
<tr>
<td>10-11</td>
<td>25</td>
</tr>
<tr>
<td>Total</td>
<td>79</td>
</tr>
</tbody>
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**TABLE III**

<table>
<thead>
<tr>
<th></th>
<th>Number with three or more joints lax</th>
<th>Number examined</th>
</tr>
</thead>
<tbody>
<tr>
<td>Non-familial cases</td>
<td>Boys: 19</td>
<td>26</td>
</tr>
<tr>
<td></td>
<td>Girls: 14</td>
<td>48</td>
</tr>
<tr>
<td>Familial cases (parent or sibling affected)</td>
<td>Boys: 5</td>
<td>7</td>
</tr>
<tr>
<td></td>
<td>Girls: 4</td>
<td>7</td>
</tr>
</tbody>
</table>

description of his generalised joint laxity, which was also present in his three sons, the first and second of whom had congenital hip dislocation. In family B the joint laxity which was present in the index patient and her brother was also present in the mother and the mother's mother. In family C the index patient, a girl, did not show joint laxity, but the affected boy did and so did his mother. In family D generalised joint laxity was present in association with congenital dislocation of the hip in the patient, her mother and her mother's mother. The presence of the other predisposing factors is shown in the figures.

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CONCLUSIONS

It is apparent from this investigation that generalised joint laxity, which is often familial, plays a part in the etiology of most cases of congenital dislocation of the hip in boys. Even in the nine male patients in this series who did not fulfil our criteria for generalised joint laxity, there was some evidence of joint laxity in six.

![Diagram](image)

Families containing two or more patients with congenital dislocation of the hip—showing the interaction of joint laxity, acetabular dysplasia and breech malposition.

Generalised joint laxity plays a part in the etiology of a smaller, but still significant, proportion of girls with congenital dislocation of the hip. In girls, as noted above, there is an alternative or additional hormonal mechanism by which generalised joint laxity might be induced in utero. Although we have not been able to demonstrate clinically such sex-limited laxity conclusively during the neo-natal period, Andrén (1962) has demonstrated it radiologically.

Idiopathic generalised joint laxity is probably an important factor in the familial cases of congenital hip dislocation in both sexes.

We have to stress that joint laxity, whether idiopathic or hormonal, is only a predisposing factor in the pathogenesis of dislocation, except perhaps when it is extreme (as in Case 2 of Carter and Sweetnam 1960). Primary acetabular dysplasia is also a predisposing factor in many cases, as indicated by the presence of a shallow acetabulum on the unaffected side in about 40 per cent of unilateral cases of congenital hip dislocation (Wilkinson and Carter 1960). However, it is our belief that the primary factor in congenital dislocation of the hip is mechanical and often, if not always, consequent on breech malposition (Wilkinson 1963).
SUMMARY

1. General joint laxity affecting more than three joints was found in 7 per cent of normal schoolchildren. Similar laxity was found in fourteen of a random series of forty-eight girls, and in nineteen of twenty-six boys, with non-familial congenital dislocation of the hip. Such laxity was also found in four of seven girls and five of seven boys with familial (first degree relative affected) congenital dislocation of the hip.

2. It is concluded that persistent generalised joint laxity, which is often familial, is an important predisposing factor to congenital dislocation of the hip in boys. It is less important in girls, except perhaps in familial cases, as in girls there is an alternative temporary hormonal cause of joint laxity.

We wish to thank the surgeons at The Hospital for Sick Children for access to their patients and, in particular, Mr G. C. Lloyd-Roberts for his interest and encouragement. We are indebted to Mrs K. A. Evans for arranging the visits to these patients. Mr H. J. Seddon and Mr David Trevor kindly allowed us to see their male patients at The Royal National Orthopaedic Hospital and we also thank Mrs M. A. Glen Haig of the Records Department for arranging appointments. We are indebted to the Department of Medical Illustration at The Hospital for Sick Children for the Figures. We wish to thank Dr Peter Currie and the Heads of the two schools concerned for arranging the examination of the schoolchildren.

REFERENCES