RECURRENT DISLOCATION OF THE PATELLA
HISTOCHEMICAL AND ELECTROMYOGRAPHIC EVIDENCE OF PRIMARY MUSCLE PATHOLOGY

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The role of muscle function in the aetiology of recurrent dislocation of the patella has been examined. Eleven of the 12 patients we studied had joint hypermobility. Muscle biopsies from eight of nine patients treated by surgery had a predominance of abnormal Type 2C fibres, and three of six patients whose quadriceps muscles were studied by electromyography also had abnormal results. Our preliminary findings suggest that there may be a primary muscular defect in many cases of recurrent dislocation of the patella.

Recurrent dislocation of the patella is usually spontaneous and only rarely follows traumatic dislocation (Heywood 1961). Most commonly seen in females in late adolescence (Macnab 1952), the condition is often familial (Bowker and Thompson 1964), and about one-third are bilateral. Numerous aetiological factors have been suggested, most of which are mechanical. Dislocation is nearly always lateral, owing to the normal valgus configuration of the knee; an increased "Q angle" is an exaggeration of this configuration and may be aggravated by a laterally located tibial tubercle (Trillat, Dejour and Couette 1964) or by genu valgum (Insall 1982). Occasionally, abnormal soft tissues, either congenital or acquired, pull the patella laterally (Jeffreys 1963; Gunn 1964).

In patella alta, dislocation of the patella can occur proximal to the intercondylar patellar groove or sulcus, but when the patella is not too high dislocation is prevented by the bony ridges; the effectiveness of this bony constraint has been studied radiologically (Brattström 1964; Merchant et al. 1974; Hughston and Walsh 1979; Laurin, Dussault and Levesque 1979). Wiberg (1941) analysed the relationship between patellar shape and the lateral femoral condyle in great detail and recognised a group with a low lateral femoral condyle which was prone to dislocation. None of the features previously identified, however, are seen in every case of recurrent dislocation of the patella, nor does any of them explain the predominance of this condition in females in their late teens.

Recurrent dislocation has also been strongly correlated with ligamentous laxity (Carter and Sweetnam 1958; Heywood 1961); ligamentous laxity is a somewhat ill-defined entity but certainly exists and includes the group with Ehlers–Danlos syndrome who have an increased incidence of patellar dislocation (Beighton and Horan 1970).

In this study we examined the role of muscle itself in the aetiology of recurrent dislocation of the patella.

PATIENTS AND METHODS

Twelve patients (eight female and four male) with a history of recurrent dislocation of the patella were studied. Their ages ranged from 14 to 23 years (mean 17.7 years), and six had a positive family history of patellar instability. Patients were examined for hypermobility of joints, and had muscle biopsies, electromyography and nerve conduction studies.

Hypermobility. This was assessed in all 12 patients by using the scoring system of Carter and Wilkinson (1964) as modified by Beighton and Horan (1970).

Muscle biopsy. Nine patients were treated by operation for their dislocating patellae, and specimens from the superficial fibres of the vastus lateralis muscle were taken for histological examination and histochemical fibre typing. There were seven females, including a pair of identical twins and two males (mean age, 17.7 years).
Muscle biopsies were also taken from seven patients (five males and two females; mean age, 24 years) operated upon for acute injuries of the femur or knee; these patients acted as controls.

Blocks of muscle approximately 10 mm³ were attached to a microtome chuck and orientated vertically to produce 11 transverse serial sections 5μm thick. Sections for light microscopy were cut within two hours of collection on a Bright's cryostat set at a temperature of −20°C. A single section was also cut at 10.0μm for fat-staining.

For routine histological examination, the sections were stained with haematoxylin and eosin, Gomori's trichrome for phosphatases and lipases, periodic acid-Schiff (PAS) for glycogen and Sudan black B for lipids.

The section stained for myosin ATPase (pH 4.3) was then examined. The pale or unstained fibres which had not been labelled were identified as Type 2B, while any darkly stained fibres (which by previous analysis had proved to be Type 2) were designated as Type 2C. These were further checked in that they must have remained darkly staining in all the myosin ATPase sections and must have been confirmed as Type 2 fibres against the NADH-TR section.

Electromyography. Six patients returned for electrophysiological studies. Nerve condition velocities were measured in the common peroneal and sural nerves and quadriceps electromyography was performed on a Medelec MS 6 machine using concentric needle electrodes.

Histochemical reactions were carried out for nicotinamide-adenine dinucleotide reduced-tetrazolium reductase (NADH-TR), and adenosinetriphosphatase (ATPase).

In order to determine the proportion and distribution of the muscle fibre types in the sections, a Leitz drawing tube was attached to a Leitz dialux microscope, and an image of the section projected on to a plain piece of paper. The routine myosin ATPase section (pH 9.4) was examined first: the image of part of a section was projected and an outline of the fibres within a given field drawn. On the basis of this first histochemical reaction, fibres were designated as Type 1 or Type 2. Selecting the same field, the myosin ATPase section pre-incubated at pH 4.6 was examined next; the unstained fibres (Type 2) in this reaction were designated Type 2A.

RESULTS

Hypermobility. Eleven of the 12 patients had scores of four or more.

Muscle biopsy. On light microscopic examination all biopsies appeared normal. There was no change in the overall pattern of the muscle fascicles, and the shape and size of the muscle fibres were similar in both the patients with dislocating patellae and the controls. There were no necrotic fibres and centrally positioned nuclei were rare.

Muscle-fibre typing revealed that eight of the nine biopsy specimens from the patients with dislocating patellae contained many Type 2C fibres, the mean proportion of which was significantly greater than in the controls (p > 0.01, unpaired Student's t-test) (Figure 1). The increase in Type 2C fibres was accompanied by a significant decrease in the proportion of Type 1 fibres in

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the affected group (p > 0.05) from 45% to 31%. By contrast, the decrease in Type 2A fibres did not attain significance and the proportion of Type 2B fibres was unaffected.

Histochemical staining for NADH-TR revealed marked heterogeneity in the muscle-fibre population in the biopsy specimens of both the control and affected groups. No abnormal staining pattern was seen in the affected group. Glycogen and lipid staining were normal for all fibre types.

Electromyography. All six patients who returned for EMG studies had normal nerve conduction velocities. Three of them, however, had abnormal EMGs and two had major evidence of myopathy. In one patient, the motor part of the femoral nerve could not be stimulated at all and the EMG showed marked myopathic changes, although of an unusual pattern in that tension production was normal but units were small and polyphasic. In the other patient there was no spontaneous activity but there was a full interference pattern of small polyphasic potentials. The third abnormal EMG demonstrated more marginal pathology with distinctly large units (7.8uV) in the quadriceps, but the significance of this is unclear.

DISCUSSION

The association between hypermobility and recurrent dislocation of the patella is well known. Our results confirm this association and also emphasise the fact that bony anatomy is not the only factor in patellar instability.

Histochemical muscle-fibre typing is based on the staining intensity of certain enzymes. The NADH-TR reaction, a marker of oxidative metabolism, is localised to mitochondria and is strongly positive in the slow-twitch, fatigue-resistant red fibres. Myofibrillar ATPase activity, on the other hand, is predominant in the fast-twitching white fibres. When specimens are pre-incubated at various levels of pH the ATPase reactions vary markedly, and it is on this basis that the new standard classification of muscle-fibre types is performed. There is a close correlation between the physiological fibre typing and the histochemical classification.

In adult human muscle, the ratio of fibre types varies between different muscles: postural muscles have more Type 1 fibres while phasic muscles have more Type 2 fibres (Johnson et al. 1973). The fibre-type ratios of a muscle may also vary between individuals and between deep and superficial areas in individual muscles. The vastus lateralis muscle in an adult contains approximate-

ly equal quantities of Type 1 and Type 2 fibres, with Type 2A fibres being more predominant that Type 2B fibres (Johnson et al. 1973; Saltin et al. 1977). Type 2C fibres, however, are rare in adult muscle. In our study the distribution of the fibre types seen in the control group was similar to that found in these previous studies.

The significance of the increased proportion of Type 2C fibres is difficult to interpret. Type 2C fibres predominate in early fetal life, Type 1 fibres first appear at about 20 weeks, and Types 2A and 2B only appear at about 30 weeks (Farkas-Bargeton et al. 1977; Colling-Saltin 1978). By the age of one year, Type 2C fibres comprise only 3.5% of the total. The large number of Type 2C fibres in our patients might be due to persistence of a fetal or neonatal muscle type, and may represent a primary abnormality of muscle. In half of the patients studied, EMG results provided evidence of a primary myopathy.

Re-innervation of a fast-twitch muscle with a nerve from a slow-twitch muscle and vice versa results in a transformation of the properties of the muscles (Buller, Eccles and Eccles 1960). Type 2C fibres presumably represent transient states between fast- and slow-twitch fibres (Billette et al. 1981; Jansson, Sjödin and Tesch 1978). The large number of Type 2C fibres might possibly be due to a denervation-re-innervation process within the muscle, although this seems unlikely as there was no microscopic or electromyographic evidence of a neuropathy.

Heavy endurance training has been shown to result in a shift in fibre composition from fast to slow fibres so that both Types 2A and 2C fibres become more numerous (Janssen and Kaijser 1977; Jansson et al. 1978; Schantz and Henriksson 1983). By contrast, disuse leads to a slow-to-fast fibre-type conversion in animals (Maier et al. 1976). An unstable patella may place high demands on the quadriceps muscle so that the increased proportion of Type 2C fibres may be a training phenomenon secondary to the condition or, conversely, the 2C fibres may represent a slow-to-fast conversion of fibres due to disuse. This latter argument is supported by the finding that the proportion of Type 1 fibres in the affected muscles was significantly reduced when compared with the fibre distribution seen in controls. The large number of Type 2C fibres may even be due to a pool of unstable motor units which have greatly fluctuating levels of activity that vacillate with the condition.

That a soft-tissue and probably myopathic component exists in the aetiology of recurrent dislocations of the patella is evident. The exact nature of the deficit is, however, far from specific and further investigation is indicated.
REFERENCES


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