Editorial

Acute patellar dislocation—closed or operative treatment?

Acute patellar dislocation (APD) is typically sustained in sports activities and it is—after anterior cruciate ligament injury—the second most common cause of traumatic hemarthrosis of the knee (Harilainen et al. 1988). APD is a serious knee injury, which often leads to considerable subjective complaints (pain, snapping, feeling of giving way), recurrence (dislocation or subluxation) and patellofemoral arthrosis.

Although APD may occur after direct trauma in a patient with normal patellar alignment, it is generally agreed that most patellar dislocations take place in patients with habitual patellar subluxation (Fulkerson and Shea 1990, Jackson 1992). Hypoplasia of the musculus vastus medialis obliquus, increased laxity of the medial retinaculum or increased tightness of the lateral retinaculum, patella alta, flat femoral trochlea, dysplastic patella, lateralization of the tibial tuberosity, increased femoral anteversion with external tibial rotation and genu valgum, and generalized ligamentous laxity are among the numerous other factors known to predispose to the dislocation of the patella.

Operative treatment of APD began in 1888, when Roux fixed a transposed patellar tendon with a screw. Since the Roux's procedure, over 130 different operative techniques have been described for APD, but no single operation has gained widespread approval among physicians. Current surgical techniques can be classified as: 1) proximal realignment procedures between patella and femur (fascial-, tendon- and muscleplasties), 2) static distal realignment procedures between patella and tibia (tendonplasties and bone reconstructions), and 3) a combination of these techniques (proximal and distal realignment procedures).

Half a century after Roux, Hauser (1938) described his classic procedure, in which the patellar tendon insertion is transferred distally and medially with release of the lateral retinaculum and duplication of the medial retinaculum. Despite its initial popularity, this procedure is, due to the wide variety of associated complaints only rarely used nowadays (anterior knee pain, patellar arthrosis and patellar instability, especially in children) (Barbari et al. 1990), (and it should never be used in patients with an open proximal tibial epiphysis). A popular and simple operation which includes medial retinacular duplication and lateral retinacular release at arthrotomy was described by Helfet (1982). This procedure provided good short-term results (Harilainen and Myllynen 1988, Vainionpää et al. 1990) but, in the longrun, it did not seem to result in much better improvement than the previously described techniques (Harilainen and Sandelin 1993). Tendonplasty, described by Avikainen et al. (1993), is one of the latest in a long list of new surgical approaches. In this operation, the usually elongated or ruptured medial patellofemoral ligament is reinserted in the medial femoral epicondyle and augmented by the harvested adductor magnus tendon. In the light of preliminary results, this technique seems promising, but proper long-term follow-up is still lacking. Arthroscopic repair of the medial retinaculum, release of the lateral retinaculum and reduction or extraction of osteochondral fragments have also been very successful in several studies, all of which unfortunately have included very few patients (Dainer et al. 1988).

The outcome of the treatment of APD has generally been determined by simple categorical scores (excellent, good, fair and poor) and the frequency of redislocation. Various studies have shown that operative treatment results in redislocation in 0–17% and excellent or good outcome in 50–100% of the patients, respectively (Chen and Ramanathan 1984, Hawkins et al. 1986, Cash and Hughston 1988, Dainer et al. 1988, Harilainen and Myllynen 1988, Savarase and Lunghi 1990, Vainionpää et al. 1990, Avikainen et al. 1993, Harilainen and Sandelin 1993). Osteochondral fractures or loose fragments were found peroperatively in 22–100% and predisposing factors in 20–80% of the cases. The initial surgical complications included lesions of the nervus infrapatellaris, deep and superficial wound infections, deep venous thrombosis, and flexion and extension restrictions (Vainionpää et al. 1990, Nikku et al. pp 419–423).

Although there are many studies on surgical treatment, the current medical literature has fewer than 15 studies on the closed treatment. Most of them are retrospective and short-term. The protocols for closed treatment have been very inconsistent, generally comprising aspiration of hemarthrosis, immobilization of

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the knee in varying degrees and for various durations, and some kind of postimmobilization physical therapy. The redislocation rate has varied from 13% to 52% in these studies and the outcome has been excellent or good in 47% to 85% of the patients (Larsen and Lauridsen 1982, Hawkins et al. 1986, Cash and Hughston 1988, Deline et al. 1989, Savarase and Lunghi 1990, Nietosvaara 1996). Osteochondral fractures and loose fragments were found radiographically in 9–40% of the cases, which is, of course, lower than in the surgical reports, while the reported incidence in the patients with clear predisposing factors was very similar to that in the papers on surgical treatment.

In consequence of this lack of adequately designed and performed studies assessing the primary treatment of APD, the recommendations presented by various authors vary accordingly. Larsen and Lauridsen (1982) favored universal closed treatment, but Savarase and Lunghi (1990) advocated immediate operative treatment. Others have suggested primarily closed treatment combined with surgery on specific indications—e.g., when signs of certain predisposing factors, large osteochondral fragments or large palpable defects in the vastus medialis obliquus muscle are found, as well as in elite athletes (Hawkins et al. 1986, Cash and Hughston 1988, Garth et al. 1996). Some have also subjected all patients to routine arthroscopy before closed treatment, since arthroscopy, in their opinion, is the only means by which a sufficiently comprehensive view of the joint status can be established. Closed and operative treatment have also been compared in a closely related clinical problem, a recurrent dislocation of the patella (Arnbjörnsson et al. 1992). In their elegantly designed study, a unilateral operation was performed on 29 patients with bilateral recurrent dislocations. Although no definitive conclusions regarding the treatment of APD can be based on the study, it has to be noted that surgical treatment, which may bring some short-term benefits, did not seem to provide any advantage over closed treatment in the long term. It seems evident that strong controversy still exists on the optimal treatment of APD.

In this issue of *Acta Orthop Scand*, Nikku et al. (pp 419–423) present the results of their prospective, randomized study comparing operative and closed treatment of APD in 125 patients. This series is one of the largest follow-ups published on the issue and, as a properly designed (prospective, randomized) study, it provides important new information on which to base one's treatment decisions. The authors conclude that surgical treatment is not better than nonoperative treatment as regards the clinical outcome in the patients or the frequency of redislocation/recurrent subluxation in the 2-year follow-up. These results are in line with a recently published prospective randomized 2-year follow-up comparing closed and operative treatment in children (age under 16 years) with APD showing no significant difference in patient satisfaction, rate of redislocation, or the functional and clinical outcomes between treatment groups (Nietosvaara 1996).

Although Nikku et al. must be commended for their study, there are some points that need consideration. First, the follow-up period (2 years) of Nikku et al. is undoubtedly too short to justify any conclusions regarding the success of treatment in terms of redislocations after APD. The need for a substantially longer follow-up is convincingly pointed out by Mäenpää et al. (in this issue of *Acta Orthop Scand*, pp 424–426). In their 6–24-year follow-up of 75 patients, the cumulative redislocation percentage was 50, of which more than half (22/37) occurred 2 years or more after the primary dislocation. This study confirms the preliminary finding by Harilainen and Sandelin (1993), who reported a late increase in the rate of redislocations (from 2% 1 year postoperatively to 17% 6.5 years postoperatively, respectively) after operative treatment of APD. In this light, I would like to urge Nikku and his colleagues to re-examine their patients in the years to come.

Secondly, the great variation in both their surgical (numerous different operative procedures) and closed (either a cylinder cast or a plastic splint for the first 3 weeks postoperatively) treatment policies, inevitably makes the interpretation of their results difficult. The use of rigid immobilization for the first 3 weeks is not, in general, considered to be an optimal treatment of soft tissue injuries, although the "caution" of the authors is understandable, as the success rate of more active closed treatment has been reported to be poor. However, most of the previously published closed treatment protocols have been carried out using patellar bandages/braces inferior to today's products and presumably they have not given enough support to the injured tissues around the knee. Experimental and clinical studies have shown that even short periods of rigid immobilization are harmful to bone, cartilage, ligaments, tendons and muscles and cause substantial atrophy of the affected tissues and even development of permanent degenerative changes (Kannus et al. 1992). Further, Woo et al. (1997) have demonstrated that early controlled loading and motion of the healing medial collateral ligament (MCL) stimulates collagen synthesis, increases strength and helps to align the repair cells and collagen fibrils, whereas lack of tension leaves repair tissue cells and matrix disorient-

ed. The medial retinaculum with adjacent soft tissues are the most commonly injured structures in APD and, as structurally analogous to the MCL, could like that MCL be successfully treated with early controlled motion. However, Woo et al. (1997) emphasized that the positive effect of early motion on the healing soft tissue is achieved only in a completely stable joint. In view of this a theoretically optimal rehabilitation of APD would require the use of a patella-supporting brace that dissipates patellar lateral force, maintains alignment and improves patellar tracking and simultaneously allows free range of knee motion. It should probably be used for 5–6 weeks to ensure that the injured soft tissues achieve sufficient stability. Today, we may already have such devices, what we lack is a prospective randomized study that confirms/refutes this idea! Active controlled mobilization is currently regarded as the treatment-of-choice in complete tears of the lateral ligaments of the ankle and medial collateral ligament of the knee. Why could not APD also obey this rule?

Thirdly, as also pointed out by Nikku et al. (pp 419–423), we should finally believe that APD is only the clinical manifestation (outcome) of various chains of events, of which the underlying pathology can range from a simple trauma of a previously healthy ("normal") knee to complicated structural and functional pathology of the knee extensor apparatus and adjacent tissues. Therefore the etiology of APD must be most carefully assessed.

The papers by Mäenpää et al. and Nikku et al. (pp 419–423) strengthen my belief that our current treatment policies for APD are far from optimal. There is definitely a subset of APD patients who benefit from surgery, but equally a subset who do not!

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References

Arnbjörnsson A, Egund N, Rydling O, Stockerup R, Ryd L. The natural history of recurrent dislocation of the patella. J Bone Joint Surg (Br) 1992; 74: 140-2.

Avikainen V, Nikku R K, Seppänen-Lehmonen T K. Adductor magnus tenodesis for patellar dislocation: technique and preliminary results. Clin Orthop 1993; 297:12-6.

Barbari S, Raugstad TS, Lichtenberg N, Refvem D. The Hauser operation for patellar dislocation. 3-32-year results in 63 knees. Acta Orthop Scand 1990; 61: 32-5.

Cash J D, Hughston J C. Treatment of acute patellar dislocation. Am J Sport Med 1988;16: 244-9.

Chen S C, Ramanathan E B S. The treatment of patellar instability by lateral release. J Bone Joint Surg (Br) 1984; 66: 344-8.

Dainer R D, Barrack RL, Buckley S L, Alexander A H. Arthroscopic treatment of acute patellar dislocation. Arthroscopy 1988; 4: 267-71.

Deline P, Hardy D, Lafontaine M, Simons M. Initial dislocation of the patella: which treatment? Acta Orthop Belg 1989; 55: 411-27.

Fulkerson J P, Shea K P. Disorders of patellofemoral alignment. Current concepts review. J Bone Joint Surg (Am) 1990; 72: 1424-9.

Garth W P, Pompherey M, Merrill K. Functional treatment of patellar dislocation in an athletic population. Am J Sports Med 1996; 24: 785-91.

Harilainen A, Myllynen P. Operative treatment in acute patellar dislocation: Radiological predisposing factors, diagnosis and results. Am J Knee Surg 1988; 1: 178-85.

Harilainen A, Sandelin J. Prospective long-term results of operative treatment in primary dislocation of the patella. Knee Surg, Sports Traumatol, Arthroscopy 1993; 1: 100-3.

Harilainen A, Myllynen P, Antila H, Seitsalo S. The significance of arthroscopy and examination under anesthesia in the diagnosis of fresh-injury hemarthrosis of the knee joint. Injury 1988; 19: 21-4.

Hauser E D W. Total tendon transplant for slipping patella: new operation for recurrent dislocation of the patella. Surg Gynecol Obstet 1938; 66: 199-214.

Hawkins R J, Bell R H, Anisatte G. Acute patellar dislocations. The natural history. Am J Sports Med 1986; 14: 111-20.

Helfet A J. Disorders of the knee. J B Lippincott Company, Philadelphia, Toronto 1982.

Jackson A M. Recurrent dislocation of the patella. J Bone Joint Surg (Br) 1992; 74: 2-4.

Kannus P, Jozsa L, Renström P, Järvinen M, Kvist M, Lehto M, Oja P, Vuori I. The effects of training, immobilization, and remobilization on musculoskeletal tissues. Part I. Training and immobilization. Scand J Med Sci Sports 1992; 2: 100-18.

Larsen E, Lauridsen F. Conservative treatment of patellar dislocation. Influence of evident factors on the tendency to redislocation and the therapeutic results. Clin Orthop 1982; 171: 131-6.

Mäenpää H, Huhtala H, Lehto M U K. Recurrence after patellar dislocation. Redislocation in 37/75 patients followed for 6-24 years. Acta Orthop Scand 1997; 68: 424-426.

Nikku R, Nietosvaara Y, Kallio P E, Aalto K, Michelsson J-E. Operative versus closed treatment of primary dislocation of the patella. Similar 2-year results in 125 randomized patients. Acta Orthop Scand 1997; 68: 419-423.

Nietosvaara Y. Acute patellar dislocation in children and adolescents. Academic dissertation. University of Helsinki, Finland. 1996: 1-57.

Savarase A, Lunghi E. Traumatic dislocations of the patella: problems related to treatment. Chi Organi Mov 1990; 75: 51-7.

Vainionpää S, Laasonen E, Silvennoinen T, Vasenius J, Rokkanen P. Acute dislocation of patella. A prospective review of operative treatment. J Bone Joint Surg (Br) 1990; 72: 366-9.

Woo S L-Y, Hildebrand K A. Healing of ligament injuries: From basic science to clinical practise. In: Soft tissue injuries in sports. Baillière's Clinical Orthopaedics (Ed. M. Järvinen). Baillière Tindall, London 1997; 1: 63-79.