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Pressure distribution in the knee joint

Influence of flexion with and without ligament dissection

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Abstract Biomechanical factors influencing the patterns of pressure distribution at the articular surface and the subchondral bone are suggested to be most important in the pathogenesis of osteoarthritis and ostechondritis dissecans at the knee joint. Besides this, chronic joint instability is another important factor under discussion in the etiology of osteoarthritis of the knee. The patterns of pressure distribution on the femoral condyles of weight-bearing knee joints were investigated in a biostatic cadaver model. The pressure on the femoral condyles was evaluated using pressure-sensitive films with the knee in different physiological joint positions (extension, 15° and 30° flexion) with and without division of either the medial collateral ligament (MCL), the lateral collateral ligament (LCL), the MCL and the anterior cruciate ligament (ACL), or the LCL and the ACL. Results showed that the location of the contact area and peak pressure depended on the joint position and stage of ligament division. Without ligament division the maximum peak pressure was always observed on the medial condyle. Only after MCL and combined MCL + ACL division did the lateral condyle show in extension a higher peak pressure than the medial condyle. Division of the LCL and LCL + ACL resulted in an increase in peak pressure on the medial condyle, particularly in flexion. The highest peak pressure of all was measured in the 30° flexion position on the medial condyle after division of the LCL. The lowest at all was found on the lateral condyle in 15° flexion after LCL division. Additional ACL division resulted in only minor further changes. These results are important for the interpretation of clinically observed factors discussed in the etiology of secondary osteoarthritis of the knee and contribute to the theory of mechanical induction of osteoarthritis and osteochondritis dissecans.

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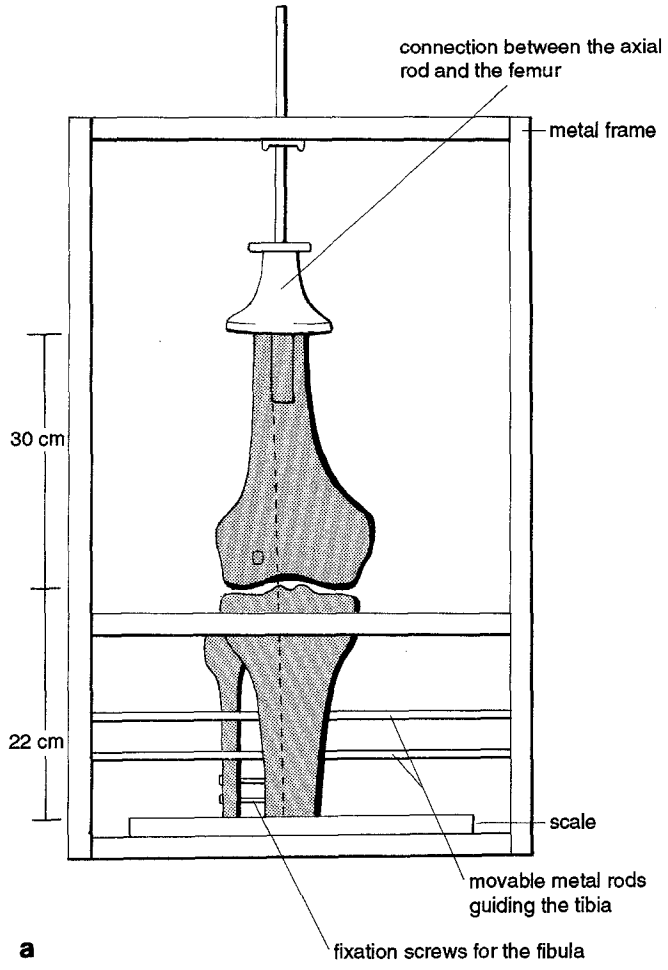
Introduction

In contrast to deviations of the axial alignment in the coronal plane, such as varus or valgus malalignments, which are themselves known as pathophysiological causes of osteoarthritis in the knee joint even without ligament lesions [18], changes in the angle of flexion and extension are physiological movements at the knee joint. Nevertheless, knowledge about the pressure distribution in different angles of flexion is important. In addition, it is likely that lesions of the collateral and cruciate ligaments alter the peak pressure in the knee joint even without malalignment in the coronal plane. It has been suggested that biomechanical factors, particularly alterations in the pressure distribution at the knee joint surface and subchondral bone, are responsible for triggering several diseases such as osteoarthritis, osteochondritis dissecans, and Ahlbäck's disease [4, 9, 29–31].

Several experiments have been performed to analyze intra-articular pressure, pressure distribution on the knee joint surfaces, and intraosseous subchondral pressure [8, 10, 13, 14, 16, 19–23, 25, 29–31, 33]. In general, pressure evaluation can be carried out using dynamic or static techniques, depending on what aspects are to be focused on; the advantages of each of these techniques are still under discussion [8]. In a continuation of our earlier study on the pressure distribution at the knee joint in dependence on varus and valgus malalignment [8], we examined the pressure distribution on the femoral condyles in dependence on different angles of flexion in comparison to extension or neutral position.

Materials and methods

In three joint positions (extension, 15° and 30° flexion) the amount of articular pressure on the femoral surface and its local distribution was examined under weight-bearing conditions with intact ligaments (division *stages D0*) and after either lateral (LCL, division *stage D1 lateral*) or medical collateral ligament division (MCL, *stage D1 medial*) and after additional division of the anterior cruciate ligament (ACL, *stage D2 medial* or *lateral*).



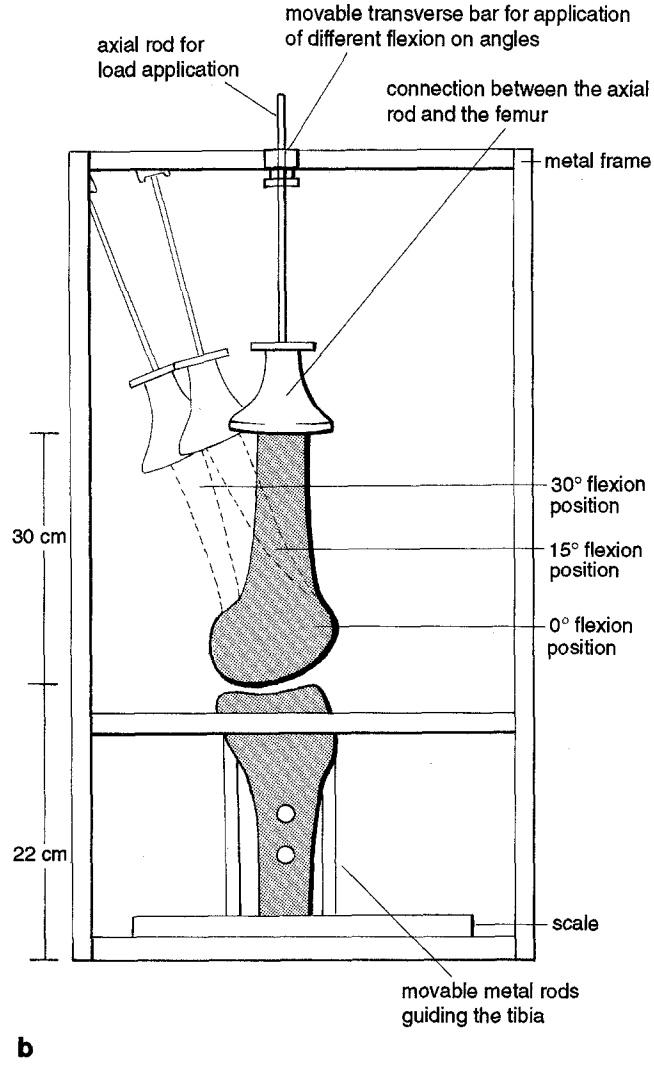


Fig. 1 a, b Experimental apparatus: **a** front view, **b** lateral view

Twelve cadaver knee joints without macroscopic signs of osteochondral lesions, osteoarthritis, ligament lesions, or axial malalignment were used. After the legs were amputated osteoligamentous preparation was performed and pressure was applied with a specially constructed apparatus as described previously [8].

For reproducible application of three different joint positions in extension and 15° and 30° flexion, the rod inserted into the proximal femur for axial load application (Fig.1) was placed in three different positions.

To prevent the tissue from drying out, preparations were kept wet by sprinkling with Ringer's solution.

For pressure measurements two pieces of a pressure-sensitive film adjusted to the size of the individual knee joint were placed in medial and lateral joint spaces (Fuji Prescale Film, pressure grade low, Fuji Photo Film Co., Ltd., lot no. 854691, range 10–80 kg/cm^2 (= 1–8 MPa). The anterior, posterior, and lateral borders of the joint surface were marked on the film.

After application of pressure with a randomly chosen axial load of 500 N for 2 min the films were removed. The pressure distribution could be immediately appreciated from the red pattern developed on the films (Figs. 2, 3). To read the color densities representing the pressure exerted on the joint surface a special densitometer (Fuji FPD-201) was used. To convert color density into pressure a pressure chart calibrated for this batch of film was used.

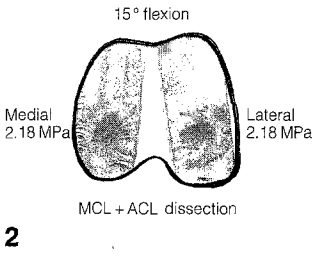


Fig. 2 Pressure distribution patterns on one pressure-sensitive film in the 15° flexion position following division of the medial collateral (MCL) and anterior cruciate (ACL) ligaments

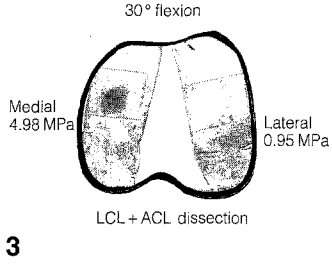


Fig. 3 Pressure distribution patterns on one pressure-sensitive film in the 30° flexion position following lateral collateral ligament (LCL) and ACL division

In all specimens the pressure distribution in the three joint positions with intact ligaments (stage D0) was examined. Then, the MCL in six specimens and the LCL in six specimens were divided (stage D1), followed by a new pressure evaluation. After this second evaluation, the ACL in all specimens was divided (stage D2) and the pressure evaluated again.

Statistical analyses were done using the Wilcoxon test.

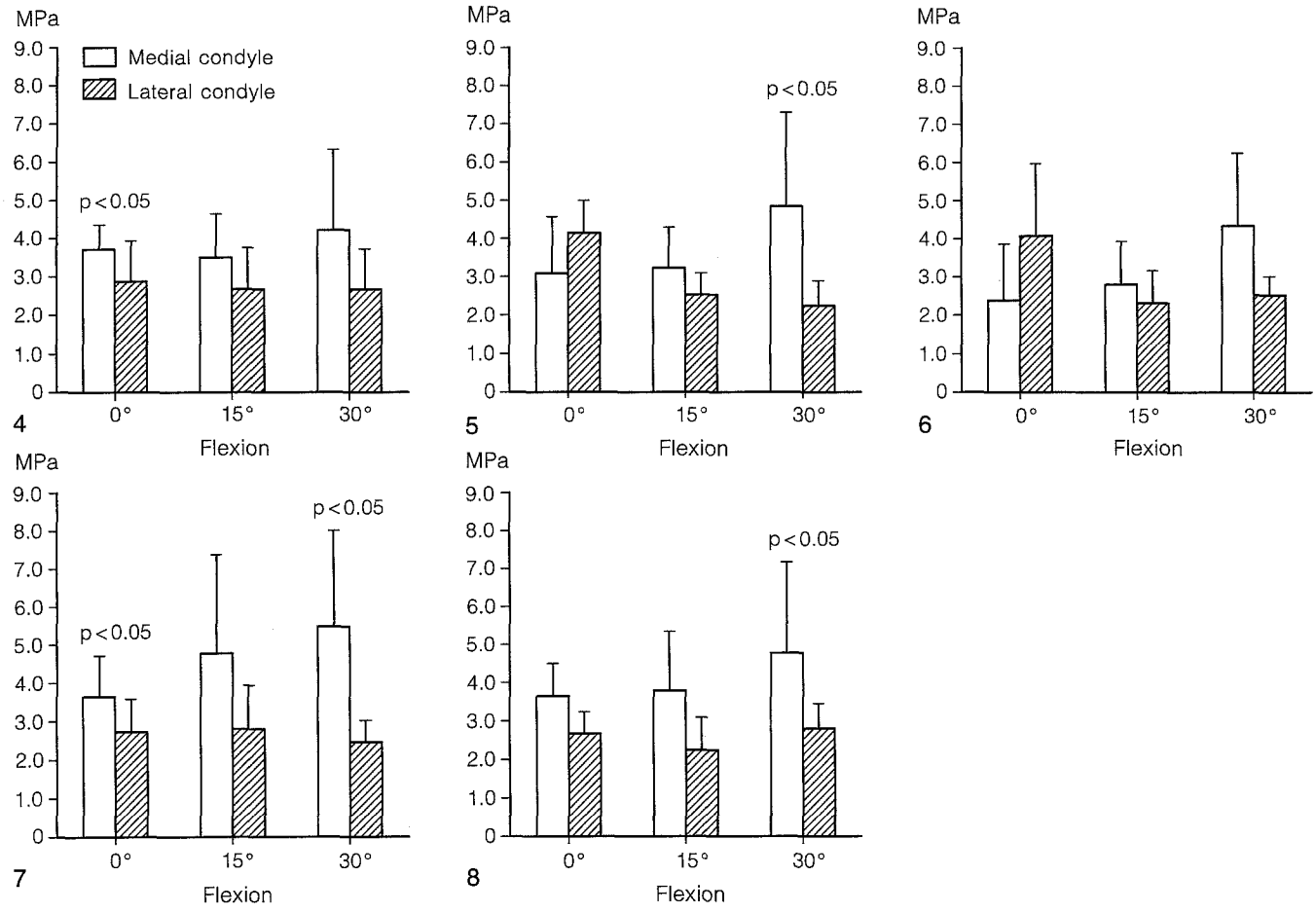


Fig. 4 Mean peak pressure values in extension and 15° and 30° flexion without any ligament division

Fig. 5 Mean peak pressure values in extension and 15° and 30° flexion after MCL division

Fig. 6 Mean peak pressure values in extension and 15° and 30° flexion after MCL + ACL division

Fig. 7 Mean peak pressure values in extension and 15° and 30° flexion after LCL division

Fig. 8 Mean peak pressure values in extension and 15° and 30° flexion after LCL + ACL division

Results

Peak pressure depended on the joint position and stage of ligament division.

Extension

Before ligament division (stage D0) the medial condyle showed a higher peak pressure (P_{max} = 3.68 MPa) than the lateral (P_{max} = 2.90 MPa; $P < 0.05$; Fig.4).

After MCL division (stage D1 medial) the medial-to-lateral difference decreased and the peak pressure on the lateral condyle (P_{max} = 3.99 MPa) was higher than that on the medial condyle (P_{max} = 3.10 MPa), but the difference was without significance ($P > 0.05$; Fig.5).

Additional ACL division (stage D2 medial) resulted in an increase of the peak pressure on the lateral condyle (P_{max} = 4.12 MPa) and a decrease of the peak pressure on the medial condyle (P_{max} = 2.43 MPa), but without a significant difference ($P > 0.05$; Fig.6).

LCL division (stage D1 lateral) resulted in a higher peak pressure (P_{max} = 3.63 MPa; $P < 0.05$) on the medial condyle than on the lateral (P_{max} = 2.68 MPa), the difference being significant (Fig. 7).

Additional ACL division (stage D2 lateral) did not change the values significantly, but the significant difference between the lateral (P_{max} = 2.67 MPa) and the medial condyle (P_{max} = 3.58 MPa) was lost ($P > 0.05$; Fig. 8).

In extension the *maximum mean peak pressure* was found on the lateral condyle after MCL and ACL division (Fig. 6). The *minimum mean peak pressure* was observed on the medial condyle after MCL and ACL division (Fig. 6).

15° Flexion

With intact ligaments (stage D0) a higher pressure was found on the medial condyle (P_{max} = 3.47 MPa) than on the lateral condyle (P_{max} = 2.72 MPa; Fig. 4), but the difference was not significant ($P > 0.05$).

After MCL division (stage D1 medial) the peak pressure values on both medial (3.16 MPa) and lateral condyles (2.47 MPa) had decreased compared with stage D0; no significant difference was observed between medial and lateral (Fig. 5).

Additional ACL division (stage D2 medial) resulted in a further reduction of the peak pressure on both condyles medial 2.77 MPa; lateral 2.35 MPa); there was no significance difference ($P > 0.05$; Fig. 6).

With LCL division (stage D1 lateral) the mean peak pressure on the medial condyle (P_{max} = 4.60 MPa) was higher than at stage D0 (P_{max} = 3.47 MPa), but not significantly different ($P > 0.05$) to that on the lateral condyle (P_{max} = 2.81 MPa; Fig. 7).

Additional ACL division (stage D2 lateral) resulted in a similar reduction of the peak pressure on both condyles, with a higher pressure on the medial condyle (P_{max} = 3.82 MPa) than on the lateral condyle (P_{max} = 2.35 MPa; $P < 0.05$; Fig. 8).

The *maximum pressure* in the 15° flexion position was found on the medial condyle after LCL division (stage D1 lateral; P_{max} = 4.69 MPa). The lateral condyle showed maximum pressure after LCL division (stage D1 lateral; P_{max} = 2.81 MPa; Fig. 7). The *minimum pressure* in the 15° flexion position was found on the lateral condyle after LCL + ACL division (stage D2 lateral; P_{max} = 1.15 MPa; Fig. 8).

30° Flexion

With intact ligaments (stage D0) the mean peak pressure was higher on the medial condyle (P_{max} = 4.7 MPa; $P > 0.05$) than on the lateral condyle (P_{max} = 2.73 MPa; Fig. 4).

After MCL division (stage D1 medial) the pressure on the medial condyle had increased (P_{max} = 4.77 MPa) in comparison to the lateral condyle (P_{max} = 2.17 MPa); the difference was significant ($P < 0.05$; Fig. 5).

Additional ACL division (stage D2 medial) led to a reduction of peak pressure on the medial condyle; no marked changes were seen in the lateral condyle (P_{max} = 2.53 MPa), and the medial-to-lateral difference was nonsignificant ($P > 0.05$; Fig. 6).

After LCL division (stage D1 lateral) the pressure on the medial condyle (P_{max} = 5.40 MPa) was higher than the peak pressure on the lateral condyle (P_{max} = 2.41 MPa), the difference being significant ($P < 0.05$; Fig. 7).

Additional ACL division (stage D2 lateral) resulted in a further decrease in the peak pressure on the medial condyle (P_{max} = 4.79 MPa), with a significant difference to the lateral condyle (P_{max} = 2.17 MPa; Fig. 8).

The *maximum peak pressure* in the 30°-flexion position was found on the medial condyle (P_{max} = 5.40 MPa); the *minimum peak pressure* was observed on the lateral condyle after MCL division and after LCL + ACL division (P_{max} = 2.17 MPa in both cases).

Overall maxima and minima

The *maximum mean peak pressure* overall on the *medial* condyle was in the 30° flexion after LCL division (P_{max} = 5.40 MPa; Fig. 7). The *minimum* at the medial condyle was found in extension after MCL + ACL division (P_{max} = 2.43 MPa; Fig. 6).

The *lateral* condyle showed *maximum peak pressure* in extension after MCL + ACL division (P_{max} = 4.12 MPa; Fig. 6); the *minimum* (P_{max} = 2.17) was found in the 30°-flexion position after MCL division and after LCA + MCL division

Discussion

As discussed previously [8], dynamic gait analyses allow examination of normal and pathological dynamic conditions in vivo including normal and pathological muscular and/or neurological functions. However, these techniques do not allow direct measurement of the intra-articular pressure on the articular surfaces, and the pressure can only be evaluated by mathematical estimation [3, 6, 7, 13–15, 17, 20–23, 25, 33]. In contrast to that, static techniques allow exact evaluation of the pressure distribution on the joint surface under defined conditions, which necessarily exclude physiological and pathological elements such as muscular strength and neurological disease.

These differences are important for the interpretation of results obtained with this particular experimental design, in which changes in the distribution of pressure in the knee joint were analyzed in dependence on flexion-extension movements. In addition, in regard to physiological conditions in flexion, in this experiment the function of patellar and quadriceps strength, flexion moments due to the action of hamstrings, biceps femoris and the gastrocnemius muscles, and the antagonistic effects of the patellar-quadriceps complex on the ACL, particularly after ACL division, could only be roughly estimated.

Nevertheless, our results clearly demonstrate that under almost all experimental conditions the medial condyle is exposed to a higher peak pressure than the lateral condyle. Only after MCL and combined MCL ACL division did the lateral condyle in extension exhibit a higher peak pressure than the medial condyle.

In contrast to that, a higher peak pressure on the medial condyle was always observed when the knee was in 15° and 30° flexion. This was even observed after MCL and combined MCL + ACL division. The values were similar to those reported by Ahmed and Burke [1], Baratz et al. [5], Brown and Shaw [6], Fukubayashi and Kurosawa [10], McKellop et al. [19], and Walker and Hajek [33], and corroborated analyses of mathematical analyses and biomechanical considerations by Maquet [18] and Pauwels [26].

To our knowledge, only a few studies have been reported that examine biostatically the influence of "physiological" flexion of the knee on the pressure patterns at the femoral condyle. However, our results are indirectly in

agreement with results reported by Kettelkamp and Jacobs [16] and Walker and Hajek [33], both of whom found reduction of the tibiofemoral contact area with increasing angles of flexion.

Harrington [13, 14] and Morrison [21–23] stated that the center of joint pressure was located in the medial compartment, that almost no force was exerted on the MCL during weight bearing, but that the LCL, on the other hand, transmitted load for a major portion of the stance phase; they calculated a 140-lb load on the LCL for most of the stance phase, i.e., in even slight degrees of flexion.

Corresponding to the particular conditions during the stance phase, our data allow the deduction that the same is true in flexion, and confirm that the LCL is an important passive stabilizer of the knee joint, compensating the pressure imbalance not only in extension but also in different angles of flexion. Collateral ligament division resulted in specific changes, and isolated division of the collateral ligaments affected the pressure distribution more than combined division of the collateral and anterior cruciate ligaments. This has been observed with varus-valgus malalignment [8], and has now been demonstrated here in "physiological" flexion.

Furthermore, division of the LCL increased medial-to-lateral differences, and even MCL division led to a higher peak pressure on the medial condyle than on the lateral, except in extension. This agrees with results reported by Grood et al. [12]. Comparing different restraint moments, these authors found that the LCL and MCL have the most important restraining function, and that the cruciate ligament complex has a minor restraining function.

The balancing out of loading conditions or compensation of the static imbalance with accentuated pressure on the medial condyle is provided by passive stabilizers such as the ligamentous strain forces of the LCL and the restraining forces of the muscles [12] such as the biceps femoris and tensor fasciae latae. This balancing-out function of particular muscles is corroborated by Andriacchi et al. [3]. They studied muscular activities under application of different flexion, extension, adduction, and abduction moments and found a distinct increase of EMG-registered activities of muscles with abduction function (particularly the biceps femoris, lateral gastrocnemius, and tensor fasciae latae), especially in slight flexion when adduction moments were applied additionally. Similar results were reported by Olmstead et al. [24] and Gollehon et al. [11]. Olmstead et al. [24] suggested that preventing opening of the lateral joint space under varus loading caused greater varus stability. Gollehon et al. [11] demonstrated the stabilizing effect of the posterolateral ligament complex.

The interaction between active and passive knee stabilizers during level walking was examined by Schipplein and Andriacchi [32]. They found that patients with lateral laxity tended to compensate for a high mid-stance phase adducting moment by walking with a style of gait that demanded more muscle force, with greater flexion-extension moments. In addition to that, dynamic examination of lower-limb mechanics during stair climbing (with knee flexion) resulted in distinctly increased flexion-extension moments and joint forces [2, 20].

Conclusions: Clinical relevance

In regard to the etiology of osteoarthritis of the knee, the present results clearly demonstrate the accentuated function of the medial condyle even in knee flexion and underlines how every manipulation of the knee joint that changes the balance of forces towards a medial overload is an important predisposing factor for arthrosis. Further, these data contribute (in combination with results reported in part I of the study [8]) to an understanding of the biomechanical etiology of osteochondritis dissecans, at least at the medial condyle [4, 9]. For the management of this particular disease, this means that factors that may be disturbing the balance of forces at the knee joint need to be considered in treatment.

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