

Varus Knee Osteoarthritis: Whence the Varus?



Osteoarthritis (OA) is believed to arise from the interplay of many factors, some of them mechanical (Figure 1). Typically there is deterioration or failure of the joints' cartilaginous bearing surfaces, and to a lesser extent of the collagenous supporting structures. In line with a multifactorial etiology for OA, current approaches to gonarthrosis tend to be multidisciplinary, in which the knee is viewed as an organ.

Mechanical Factors

We have focused on one aspect of the mechanical influences — the association between the disease and certain patterns of limb alignment (“static mechanical” factors). For example, it is well known that patients with knee OA are commonly bow-legged, meaning that they have a varus alignment of their limbs. In this condition (“varus OA”) there is almost always a loss of joint space (JSL) focused in the medial tibiofemoral compartment. In contrast, the less common “knock-knee” deformity (“valgus OA”) generally has the JSL focused in the lateral compartment. Both varus and valgus alignment abnormalities increase the odds of JSL occurring, and the progression of the JSL has been shown to correlate with the severity of the deformity¹.

Malalignment of the limbs is typical in OA, and JSL accounts for some but not all of this; important contributions are also made by femoral and tibial geometry².

Contributions of the tibia to varus limb alignment are well recognized in the developmental conditions of tibia vara, Blount's disease, and in adults with the so-called “malicious mal-alignment.” However, in varus knee OA the tibia appears not to be the major contributing factor. This observation arises from the studies of 2 large populations, one Caucasian and the other Middle-Eastern^{3,4}. Unexpectedly, we found that their mean tibial plateau varus angles were not significantly different from the mean values in healthy adults. (We noted tibia vara in some Middle-Eastern cases, but this was largely in advanced disease, where severe erosion of the proximal medial plateau had occurred⁴. We suspect these cases relate to the high prevalence of metabolic bone diseases (osteoporosis and osteomalacia) in the region, and possibly to lifestyles as well⁵.) What we found in both Caucasian and Middle-Eastern groups was a contribution of femoral geometry to the varus alignment in OA —

in particular a reduced valgus angle at the distal femur. This femoral abnormality was also associated with disease advancement, especially in the Middle-Eastern group, where both the abnormality and the disease itself tended to be more pronounced than in the Caucasian group.

These findings may implicate “reduced femoral valgus” as a predisposing factor for varus OA. On the other hand, it could be that the altered femoral geometry arose from significant remodeling of the femur during the disease process itself⁶. At present one cannot say which of these alternatives is the most influential in the course of OA. It is relevant, however, that normal aging may cause increased varus alignment, even in the absence of disease (i.e., no symptoms, no JSL)⁷. So if our bones tend to bow during natural aging, we may at some critical stage become vulnerable — first to onset of OA, and then to progressive joint deterioration as our bones continue to change. This needs to be considered in addition to all the many other factors that may predispose to OA.

In osteotomy the tibia is usually chosen for varus correction to unload the medial compartment. But the functional outcome and survival may be limited^{8,9} and other options have been used with benefit, including femoral and selective double osteotomy^{10,11}. These choices were based on the principle of lessening compartmental overload medially, by correction of the most deformed bone(s) identified by analysis of bone and joint loading contributions.

Directions for Future Research

Among the many questions raised from these observations are the following:

- Is “reduced femoral valgus” a developmental or a developing condition; does it predispose to varus OA? *OR*
- Does this condition arise as part of the pathogenesis of the disease? If so, how?
- In varus OA cases that lack bone abnormalities, what is it that drives the disease? To what extent are various genetic, biological, dynamic mechanical, or constitutional factors involved?
- In individual cases how does one define or differentiate between primary tissue weakness (biological) and overload (mechanical) as factors favoring the loss of cartilage?
- When surgery is required in varus knee OA, why should

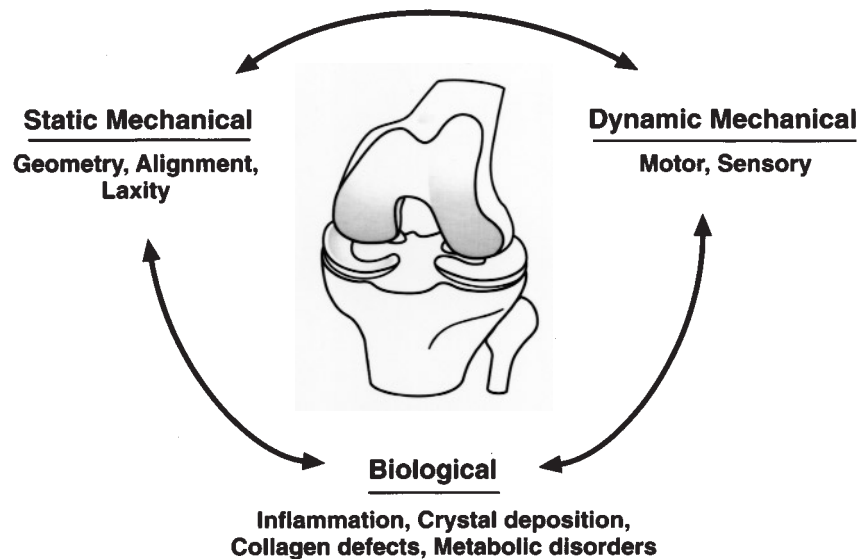


Figure 1. Mechanical and other factors that contribute to osteoarthritis.

tibial osteotomy be the logical first choice if femoral abnormalities are so frequent? *AND*

- In which circumstances would femoral realignment, if applied in the early stages of the disease, serve to ameliorate the progression?
- What nonsurgical approaches, such as bracing and selective gait training, may be effectively applied to modify the disease process?

Finally, to address some of these issues we propose the following as priorities for further evaluation:

1. Conduct longitudinal studies of limb alignment and bone geometry in populations that may be at risk, before symptoms occur
2. Develop measures or protocols assigning weight to the many risk factors for OA, e.g., bone weakness (osteoporosis and osteomalacia); mechanical overload (static, dynamic, prior trauma); genetic and racial factors; body mass; lifestyle (exercise, drinking, smoking, etc); and history of medication. These may be applicable either to study populations or individual cases, for developing or established OA
3. Establish relationships between alignment changes and biological markers of cartilage degradation, using suitable study populations
4. Re-examine studies of tibial osteotomy for varus knee OA, assessing differences in outcome in cases with mainly tibial versus femoral deformity.

We hope these remarks will provoke discussion and stimulate study in some of these areas.

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