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Defining sagittal knee phenotypes via monopedal static anterior tibial translation. Part 1: Translating weight-bearing sagittal position into clinical risk profiles

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Abstract

Static anterior tibial translation (sATT) is a reproducible monopodal weight-bearing radiographic parameter that reflects the resting sagittal position of the tibiofemoral joint. Distinct from manual laxity tests that quantify passive displacement limits, sATT captures the functional equilibrium of the tibia under physiological load, representing the cumulative interplay of bony morphology, neuromuscular control, meniscal containment and time-dependent adaptations. Current literature frequently conflates passive laxity with weight-bearing sagittal position, leading to an incomplete understanding of why isolated soft-tissue reconstructions fail in biomechanically hostile environments. Elevated sATT manifests in three clinically distinct sagittal phenotypes with specific pathomechanisms: *osseous anteriorization* primarily related to increased posterior tibial slope and loss of the anterior cruciate ligament; *acquired soft-tissue anteriorization* resulting from sagittal decompensation due to chronic injury and secondary stabilizer, particularly meniscal deficiency; and *inherent soft-tissue anteriorization* characteristic of generalized hyperlaxity. This paper, Part 1 of a two-part series, establishes the biomechanical 'set-point' theory, highlights the necessity of strict monopedal imaging to unmask instability and defines how distinct sagittal phenotypes may converge, creating cumulative or synergistic risk patterns that increase the mechanical burden on the central pivot.

Level of Evidence: Level V.

KEYWORDS

anterior cruciate ligament reconstruction, knee instability, posterior tibial slope, slope-reducing osteotomy, static anterior tibial translation

Abbreviations: ACL, anterior cruciate ligament; ACLD, anterior cruciate ligament deficiency; ACL-R, anterior cruciate ligament reconstruction; ATS, anterior tibial subluxation; ATT, anterior tibial translation; DSX, dynamic stereo x-ray; GJH, generalized joint hypermobility; KT-1000, KT-1000 arthrometer; MRI, magnetic resonance imaging; PTS, posterior tibial slope; sATT, static anterior tibial translation; sLATT, static lateral anterior tibial translation; SRO, slope-reducing osteotomy; ΔsATT, side-to-side difference in static anterior tibial translation.

INTRODUCTION: FROM PASSIVE LAXITY TO WEIGHT-BEARING SAGITTAL POSITION

Anterior cruciate ligament (ACL) deficiency increases anterior tibial translation (ATT), yet objectively quantifying this phenomenon remains challenging in clinical practice. Consequently, the field moved towards quantification, initially relying on dynamic stress tests such as the Lachman test and its instrumented or stress-based laxity assessments, such as the KT-1000 arthrometer (KT-1000; MEDmetric Corp.), Rolimeter arthrometer (Aircast Europa) and Telos stress device (Telos GmbH) [9, 15, 17, 22]. Radiographic extensions of these concepts were also introduced, such as the radiographic Lachman test, of which two different techniques were available [12, 34] and the quadriceps-contraction technique [13], aiming to improve objectivity and reproducibility by visualizing tibial displacement under controlled conditions. While these techniques enhanced diagnostic accuracy for ACL insufficiency [34], they primarily assessed the limits of motion within the 'joint play envelope' rather than the sagittal kinematic pattern of the knee. Accordingly, a key unmet need remains: an objective parameter that captures the tibia's resting sagittal position under physiological load rather than its passive displacement limits. Monopedal weight-bearing static ATT (sATT) addresses this by quantifying the equilibrium position of the tibia under axial load, approximating the early-stance kinematics of the knee.

STATIC JOINT KINEMATICS UNDER PHYSIOLOGIC LOAD

Measured on strict monopedal weight-bearing lateral radiographs (Figure 1), sATT represents static joint kinematics under physiologic load, providing a more functional assessment than its historical 'static' label might suggest [12]. Unlike passive tests, sATT is obtained during active weight-bearing, integrating the forces of body weight, joint compression, muscle tone and limb geometry. It captures the sagittal equilibrium position, the functional 'set-point' of the tibia relative to the femur, at a specific point in the stance phase.

The 'envelope versus set-point' theory

Conceptually, instrumented laxity defines the size of the *motion envelope* [23], whereas sATT identifies the *functional set-point* within that envelope under load. This distinction explains why knees with similar instrumented laxity may demonstrate markedly different sATT values and divergent clinical behaviour [45].

FIGURE 1 Schematic illustration of the setup for obtaining strict monopedal weight-bearing lateral radiographs. This technique is used to measure static anterior tibial translation, capturing a static state of the knee under physiologic loading that integrates active forces, including body weight, joint compression and muscle tone, to determine the functional sagittal equilibrium 'set-point' of the knee at a specific point in the stance phase.

When abnormal, sATT indicates a shifted resting position, an altered balance between joint geometry, ligamentous restraint and muscular forces that may persist even after technically adequate ligament reconstruction. Previous studies showed ACL reconstruction (ACL-R) did not decrease monopedal weight-bearing based on sATT [11, 35, 36]. This concept is further supported by radiographic and magnetic resonance imaging (MRI)-based evidence demonstrating that clinically successful ACL-R can normalize total anteroposterior laxity as measured by instrumented laxity tests such as the KT-1000 (MEDmetric Corp.), Telos stress device (Telos GmbH) or Rolimeter arthrometer (Aircast Europa) [11, 22, 37], while leaving the tibia in a persistently anteriorly subluxated position [1, 2].

The nomenclature trap: Distinguishing monopedal sATT from passive measures

With growing recognition of its clinical relevance, sATT has recently been incorporated as an adjunct parameter in decision-making for tibial slope-reducing osteotomies (SROs) in ACL-deficient and revision settings [21, 33, 49]. However, this renewed attention has introduced significant heterogeneity in terminology.

A recent consensus statement on posterior tibial slope (PTS) [49] conflated multiple distinct measurements under the single umbrella of 'sATT.' Notably, it grouped:

1. *True sATT*: Measured on monopedal weight-bearing radiographs at 20–30° of flexion [12].

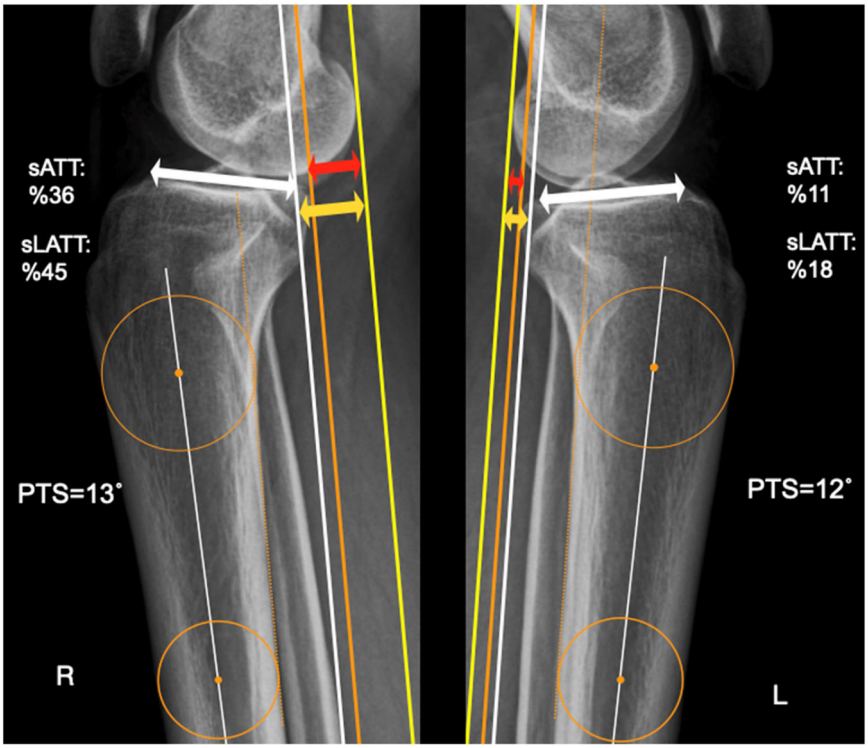

FIGURE 2 Comparison of medial versus lateral static anterior tibial translation (sATT) on bilateral monopedal weight-bearing radiographs. The white double-headed arrows indicate the sagittal width of the medial tibial plateau. The red arrows represent medial-based sATT, capturing the primary sagittal translation of the medial pivot. The yellow arrows denote lateral-based sATT (sLATT), which inherently encompasses the medial translation distance plus an additional anatomical offset dictated by the lateral compartment's geometry. In this example, the healthy left knee (L) demonstrates a medial sATT of 11% and an sLATT of 18%. On the injured right knee (R), medial sATT increases to 36% and sLATT to 45%. When evaluating side-to-side differences, the medial measurement demonstrates a 3.3-fold relative increase (36% vs. 11%), whereas the lateral measurement yields a smaller 2.5-fold increase (45% vs. 18%). Because the constitutive baseline of the lateral measurement is inherently higher, it dilutes the relative effect size of the pathological translation. This visualizes why referencing the medial compartment provides a more sensitive, high-fidelity marker of sagittal decompensation.

2. Passive anterior tibial subluxation (ATS)/MRI-based static ATT: Derived from non-weight-bearing MRI [43] or fluoroscopy in full extension [32].

Why this distinction matters

These modalities interrogate fundamentally different biomechanical states. ATS reflects a passive (gravity with the MRI coil), unloaded position, whereas sATT reflects the knee's behaviour under axial load. Furthermore, measuring sATT on full-length tibial radiographs may be technically challenging, as accurate assessment requires appropriate centreing of the x-ray beam on the knee joint to optimize femoral condyle superimposition. This geometry may be difficult to achieve consistently on long-leg films centred on the tibial shaft.

Therefore, we propose that whenever 'static' ATT is reported, a clear distinction must be made regarding the acquisition method: specifically, whether the measure represents a *monopedal weight-bearing radiographic* assessment or a *non-weight-bearing MRI or fluoroscopic* finding. While these metrics are related, they represent distinct biomechanical states and are *not necessarily interchangeable*.

Why the medial compartment?

The decision to reference sATT to the medial tibial plateau rather than the lateral compartment is grounded in the distinct anatomical and kinematic profiles of the two compartments. The medial compartment serves as the primary pivot for sagittal stability, often described as having a 'ball-and-socket' congruence compared to the more mobile, convex lateral compartment. Consequently, previous studies indicate that ACL insufficiency produces a greater and more consistent ATT on the medial side [12]. Crucially, because a lateral-based measurement inherently includes an additional anatomical offset dictated by the lateral compartment's

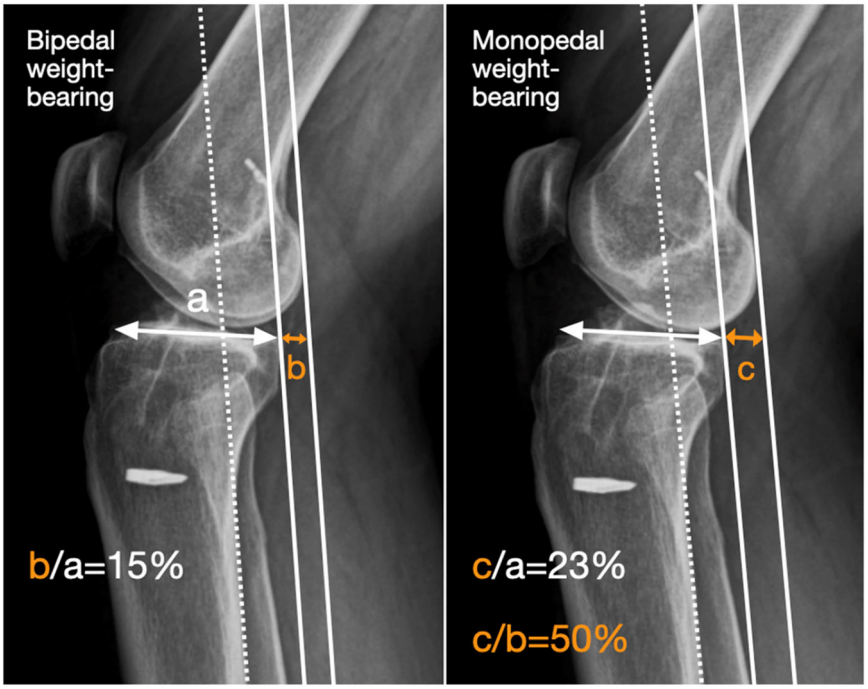

FIGURE 3 Illustration of sagittal anteriorization under monopedal loading in the setting of Anterior cruciate ligament reconstruction (ACL-R) failure. Bipedal (left) and monopedal (right) weight-bearing lateral radiographs of the same right knee in a 36-year-old male presenting with ACL-R failure. Reference line a denotes the sagittal width of the medial tibial plateau. Under a bipedal stance, partial load sharing between the limbs may reduce the apparent degree of sATT, yielding an sATT value of 15% (Line b). Under strict monopedal loading, full weight acceptance on the affected limb demonstrates a greater anterior tibial position, with an sATT value of 23% (Line c). The relative increase in ATT (c/b) by 50% shown in the figure represents a case-specific descriptive comparison between these two measurements and should not be interpreted as a validated threshold or generalizable estimate. This example illustrates how monopedal imaging may reveal sagittal anteriorization that is less apparent during bipedal weight-bearing assessment. sATT, static anterior tibial translation.

geometry, its larger constitutive baseline mathematically dilutes the relative magnitude of any pathological shift. This results in a larger effect size and lower measurement variability for medial-sided sATT, making it a significantly more sensitive marker of sagittal instability than lateral-based measurements [12] (Figure 2).

This medial predominance is also corroborated by in vivo kinematic data. At 36 months after ACL-R, tibiofemoral contact location has been shown to shift significantly more anteriorly in the medial compartment compared to the contralateral knee, whereas no significant anterior–posterior changes were observed on the lateral side [24]. These findings further validate the medial-based assessment strategy originally proposed by Dejour and Bonnin [12].

In contrast, recent studies exploring lateral ATT measurements [47, 48] or non-weight-bearing MRI-based assessments [31] highlight the ongoing challenges in achieving measurement reproducibility. Across the current literature, substantial heterogeneity exists regarding patient positioning (single-leg [48] vs. bilateral stance [47]), knee flexion angles (20° vs. 30°) and terminology, limiting direct comparability between cohorts. Furthermore, as this is a rapidly evolving field, it is important to contextualize recent reports of postoperative ATT changes following SROs [47] alongside similar radiographic frameworks established in earlier literature [7]. Synthesizing these parallel findings will be critical to addressing current discrepancies and achieving consensus regarding compartment-specific sagittal kinematics.

The necessity of monopedal loading: Unmasking the instability

The biomechanical rationale for *monopedal weight-bearing* is the necessity of 'stressing' the joint to reveal its functional limits while also applying an axial load in a walking scenario. Bipedal stance allows patients to subconsciously offload the injured limb, masking the true extent of sagittal decompensation.

This 'unmasking effect' is illustrated in Figure 3, which compares bipedal and monopedal lateral radiographs obtained from the same ACL-deficient knee. Under otherwise identical imaging conditions, monopedal loading results in a visibly greater anterior tibial position. As demonstrated in Figure 4, this example

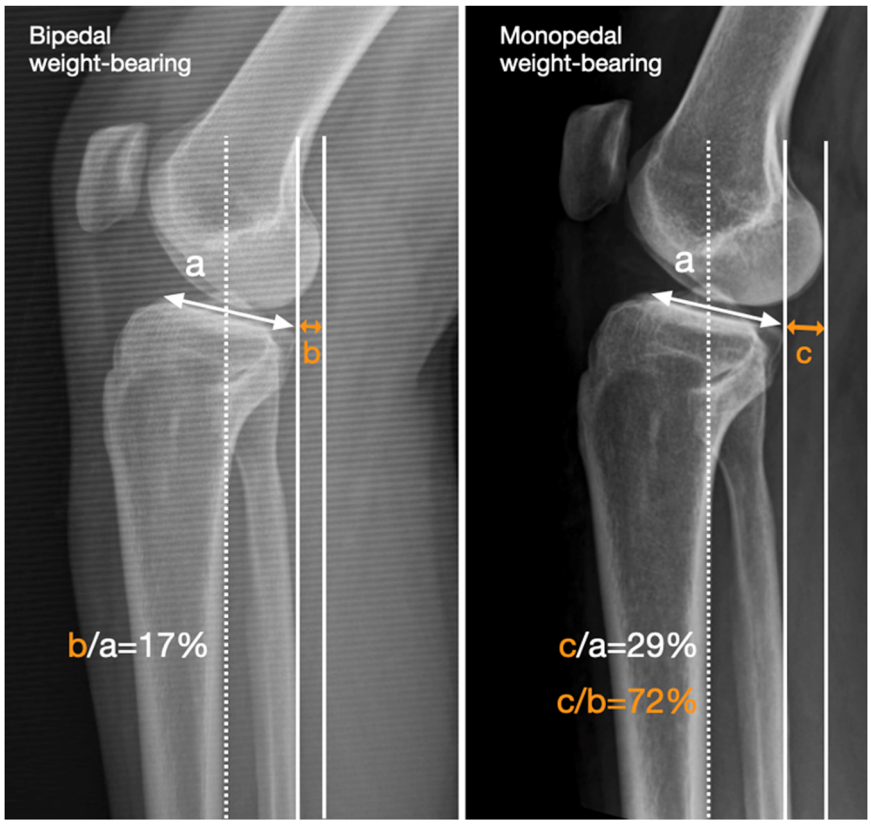

FIGURE 4 Illustration of the effect of strict monopedal weight-bearing on measured sagittal anteriorization. Bipedal (left) and monopedal (right) weight-bearing lateral radiographs of the same right knee in a 34-year-old female with anterior cruciate ligament (ACL) deficiency. Reference line a denotes the sagittal width of the medial tibial plateau. Under a bipedal stance, partial load sharing between the limbs may reduce the apparent degree of static anterior tibial translation (sATT), yielding an sATT value of 17% (Line b). Under strict monopedal loading, full weight acceptance on the affected limb demonstrates a greater anterior tibial position, with an sATT value of 29% (Line c). The relative increase in ATT (c/b) by 72% shown in the figure represents a case-specific descriptive comparison between these two measurements and should not be interpreted as a validated threshold or generalizable estimate. This illustrative example suggests that monopedal imaging may reveal sagittal anteriorization that is less apparent during bipedal imaging.

suggests that monopedal sATT may reveal clinically relevant sagittal anteriorization that is underestimated during bipedal loading.

This discrepancy highlights that sagittal decompensation is a patient-specific, load-dependent phenomenon. Consequently, non-weight-bearing imaging (such as with MRI) and even bipedal weight-bearing radiographs may significantly underestimate functionally relevant anteriorization and cannot be considered interchangeable with strict monopedal assessments.

DISSOCIATION OF INSTRUMENTED LAXITY AND WEIGHT-BEARING SAGITTAL POSITION

Historically, the primary aim of ACL-R was to restore restraint against both anterior tibial translation and rotatory instability, traditionally quantified by instrumented laxity tests and the clinical pivot-shift assessment [42]. While ACL-R consistently normalizes these passive displacement limits compared with the ACL-deficient state [42], accumulating evidence suggests that restoring static, unloaded laxity does not necessarily restore a normal tibiofemoral relationship under load.

This dissociation is most clearly demonstrated by in vivo kinematics. Dynamic stereo x-ray (DSX) analysis, considered the gold standard for in vivo precision, reveals that standard clinical laxity measurements are poor predictors of functional knee behaviour. Although KT-1000 anterior laxity may remain largely unchanged between 5 and 12 months after ACL-R, DSX analyses show progressive increases in functional graft elongation and anterior tibial translation during gait and running over the same period [45]. Importantly, no meaningful correlation exists between KT-1000 values and dynamic graft length, highlighting a fundamental distinction: static tests quantify passive motion limits within the joint play envelope, whereas DSX captures the functional motion path under muscle-driven loading [45]. These findings are corroborated

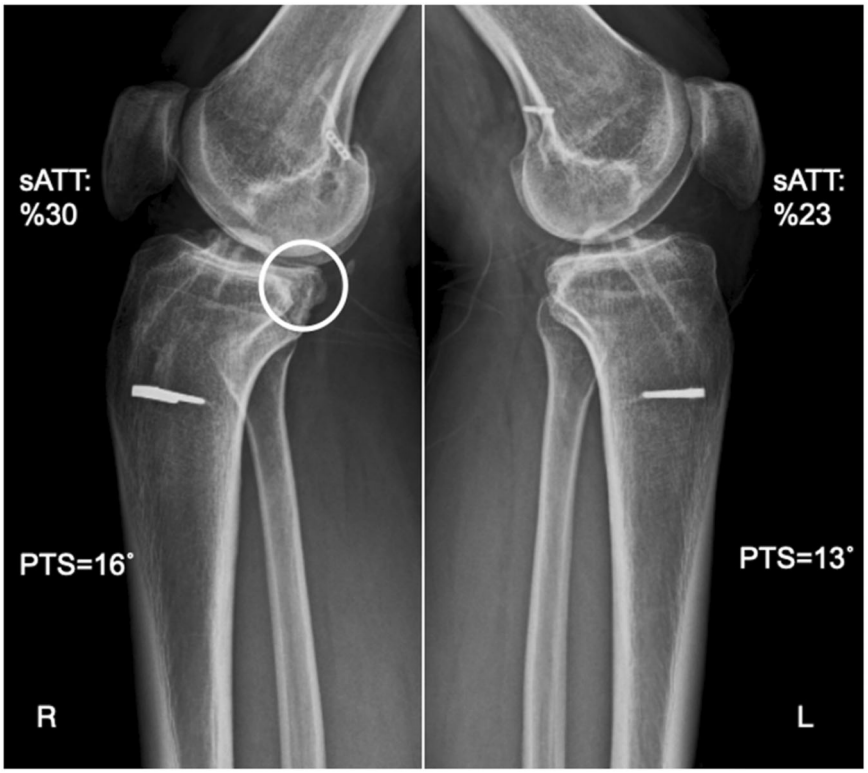

FIGURE 5 Long-term arthrokinematic consequences of chronically elevated static anterior tibial translation (sATT). Bilateral monopedal weight-bearing lateral radiographs of a 35-year old male patient who underwent anterior cruciate ligament reconstruction (ACL-R) 16 years prior on the right knee (R) and 7 years prior on the left knee (L). The complaint-free right knee demonstrates a steep posterior tibial slope (PTS) of 16° and a highly elevated sATT of 30%. The persistently anteriorized resting position of the tibia has led to chronic posterior loading by the femoral condyle, resulting in visible posteromedial compartment wear and prominent posterior osteophyte formation (white circle). In contrast, the left knee exhibits a PTS of 13° and an sATT of 23%, with correspondingly less advanced degenerative changes despite the 7-year post-reconstruction interval. This case visually underscores how an uncorrected, pathological sagittal set-point acts as a mechanical driver for compartment-specific post-traumatic osteoarthritis.

by longer-term in vivo studies (12–36 months post-reconstruction), which consistently report progressive increases in anterior translation despite stable clinical laxity [5, 24, 46].

Same-patient evidence: Set-point versus envelope

Direct evidence distinguishing the weight-bearing sagittal position from passive excursion comes from studies evaluating both parameters in the same patients. Dejour et al. demonstrated that while ACL-R successfully reduced stress-induced anterior tibial translation (from ~7–8 to 3–4 mm), monopedal sATT remained unchanged [11]. These findings confirm that non-weight-bearing stress measurements quantify the joint's global laxity limits, whereas monopedal sATT captures the functional equilibrium set-point under load. Consequently, ACL-R may restore passive restraint without necessarily recentering the tibia to a normal resting position.

In vivo evidence for the 'anterior tibial set-point'

High-resolution motion analysis further supports the concept of an altered anterior tibial set-point in ACL-deficient knees. During stance tasks and quasi-static lunge positions closely resembling monopedal weight-bearing, ACL-deficient knees exhibit a significant anterior shift (~3 mm) of the tibia compared to the contralateral healthy side at low flexion angles [10]. In contrast, healthy control cohorts demonstrate minimal physiological asymmetry, with side-to-side differences averaging ≤1.3 mm in translation across the gait cycle [14].

THE CLINICAL CONSEQUENCE: WHY THE 'RESTING POSITION' MATTERS

Why does a shifted tibial resting position matter if the knee is not subjectively unstable? A key answer lies in the characteristic pattern of post-traumatic osteoarthritis. Although the causal pathway is multifactorial, the

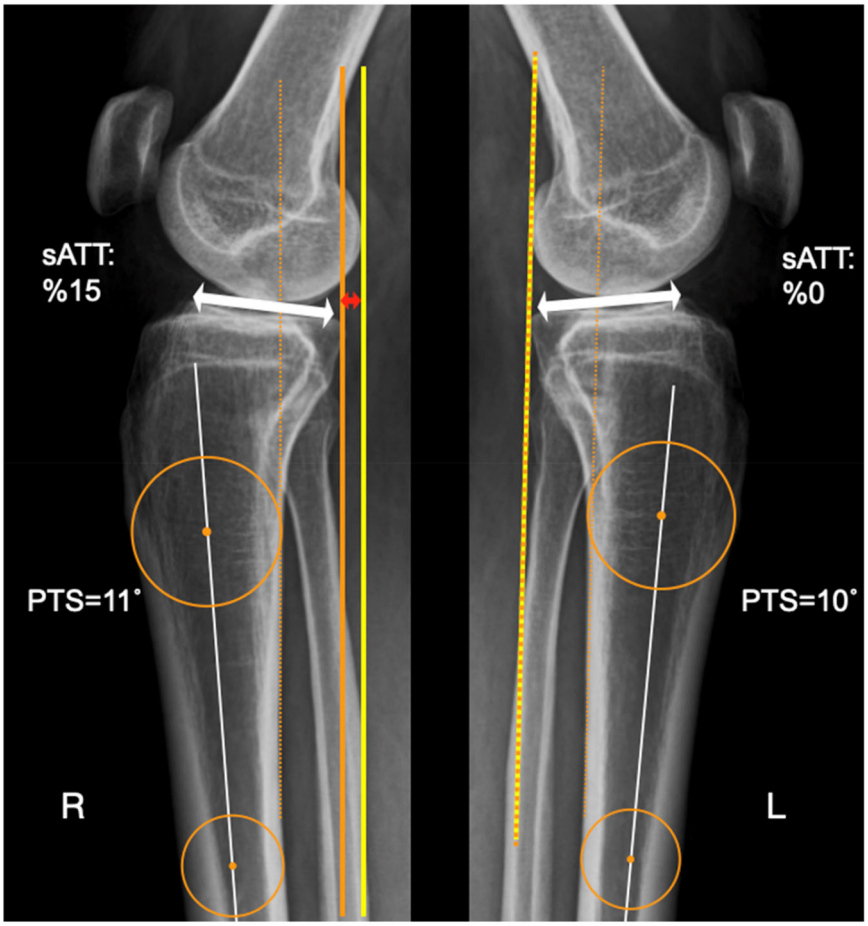

FIGURE 6 The 'PTS effect' unmasked by anterior cruciate ligament (ACL) deficiency. Bilateral monopedal weight-bearing lateral radiographs of a 34-year old male demonstrating the ACL's critical role as the primary restraint against PTS-induced anterior tibial translation (ATT). The healthy left knee (L) establishes the patient's structural baseline; despite a PTS of 10°, the static anterior tibial translation (sATT) remains fully restrained at 0%, as the intact ligament effectively neutralizes the anterior shear forces. Conversely, the right knee (R) sustained an ACL rupture with a concomitant lateral meniscal root-equivalent tear and a medial meniscal body tear. Despite possessing a nearly identical osseous morphology (PTS = 11°), the loss of the ACL 'brake' unleashes the geometric influence of the PTS, resulting in an uncompensated sATT of 15%. This illustrates how similar anatomical PTS yield drastically divergent sagittal position states depending on the integrity of the soft-tissue envelope. PTS, posterior tibial slope.

literature consistently associates ACL deficiency with posteromedial compartment wear [28, 29]. When the tibia rests in a chronically anteriorly subluxated position (elevated sATT), the femoral condyle loads the posterior region of the medial tibial plateau rather than the central concavity. Consistent with this mechanical theory, cartilage mapping studies in early post-traumatic osteoarthritis demonstrate degeneration concentrated in the posterior tibial plateau and the corresponding anterior loading region of the femoral condyle [52] (Figure 5).

Thus, sATT should not be viewed merely as an isolated numerical measurement but as a potential radiographic marker of altered sagittal tibiofemoral alignment under load. Although direct evidence linking increased sATT to long-term joint degeneration remains limited, anterior tibial shift may be associated with altered contact mechanics and meniscal or cartilage loading. Even complaint-free ACL-deficient patients may demonstrate subtle anterior tibial shift and altered contact patterns, suggesting that functional stability does not necessarily imply restoration of normal sagittal alignment [51].

The role of active stabilizers

Dynamic stability ultimately reflects the interaction between passive restraints (graft, meniscus and bony geometry including PTS) and active stabilizers. Electromyographic and kinematic studies indicate that the quadriceps and gastrocnemius act synergistically during stance to increase joint compression; however, they may also generate an anterior shear component that tends to drive the tibia forward [23, 42].

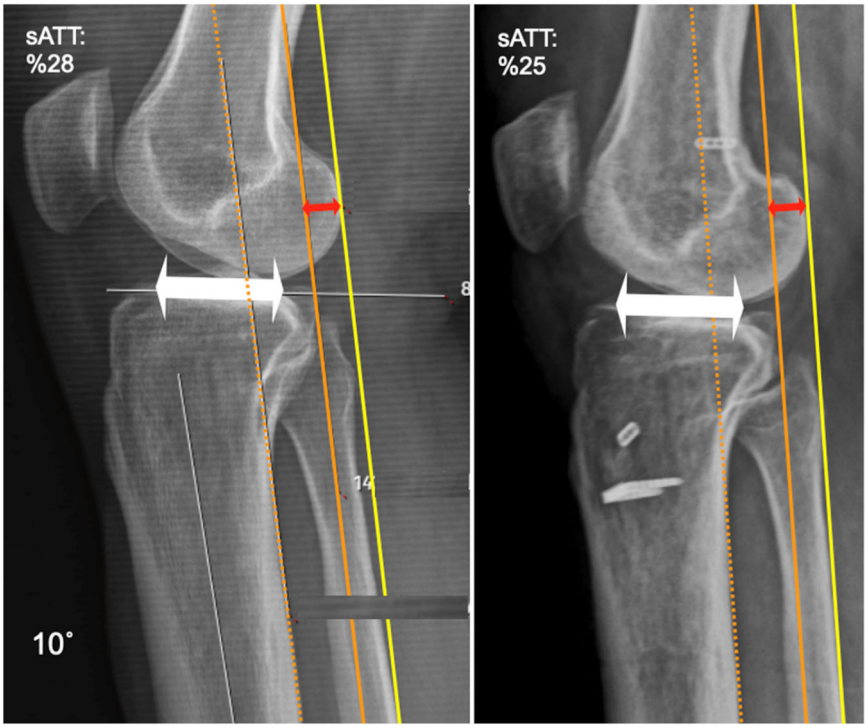

FIGURE 7 Failure of soft-tissue reconstruction to recenter the tibia in a severely decompensated knee. Preoperative (left) and postoperative (right) monopedal weight-bearing lateral radiographs of a 26-year-old male (Beighton score 2 and a posterior tibial slope [PTS] of 10°). The preoperative imaging (left) demonstrates a profoundly anteriorized resting position with a static anterior tibial translation (sATT) of 28%. This extreme acquired soft-tissue anteriorization represents an acute-on-chronic decompensation: the patient sustained an acute medial meniscal root tear 1 month prior, superimposed on a 1-year-old chronic anterior cruciate ligament (ACL) rupture. The loss of the primary ligamentous restraint, compounded by the 'meniscal multiplier' effect of the root tear, completely destabilizes the sagittal equilibrium. Following isolated soft-tissue reconstruction with ACL-R and transtibial root repair (right), the postoperative sATT remains persistently elevated at 25% under monopedal axial load. This vividly illustrates that in the setting of chronic deficiency and secondary stabilizer failure, restoring the passive motion envelope via an anatomical graft is insufficient to normalize the functional sagittal set-point, leaving the joint chronically subluxated.

While the hamstrings are theoretically capable of counteracting this translation, their ability to function as a primary restraint during early weight acceptance appears limited [23]. Instead, excessive co-contraction may produce a 'stiffening strategy,' in which the knee is stabilized but remains locked in an anteriorly subluxated position [42].

CLASSIFYING SAGITTAL KNEE PHENOTYPES

Elevated sATT should not be interpreted as a single entity or as a direct surrogate for perceived instability. Rather, it represents a final common radiographic phenotype arising from three distinct but clinically convergent mechanisms.

OSSEOUS ANTERIORIZATION (PTS-DRIVEN)

Osseous anteriorization is driven by bony morphology, specifically an increased PTS, which generates an anterior shear component under axial load and physically biases the tibia towards an anteriorly translated equilibrium position [19, 21]. Biomechanical studies consistently demonstrate that increased PTS increases ATT [16]. In ACL-deficient knees, PTS appears to be the dominant structural driver of resting sagittal position and influences whether neuromuscular compensation can meaningfully recenter the tibia, with steeper PTS producing greater anteriorization that cannot be fully counteracted [25].

Quantifying the 'PTS effect'

Clinical studies using monopedal lateral knee radiographs demonstrate that the association between PTS and sATT is substantially stronger in ACL-deficient knees than in ACL-intact controls [4, 8]. Quantitative analyses indicate that a 10° increase in PTS corresponds to an approximate 5–6 mm increase in sATT in ACL-deficient knees [12], whereas comparative cohorts report more modest PTS-related increases in ACL-intact knees (e.g., ~3.6 mm vs. ~5.0 mm per 10° when contrasting intact and deficient states) [5]. When normalized to medial plateau distance, a 10° increase in PTS produces a 9.7% increase in sATT in ACL-

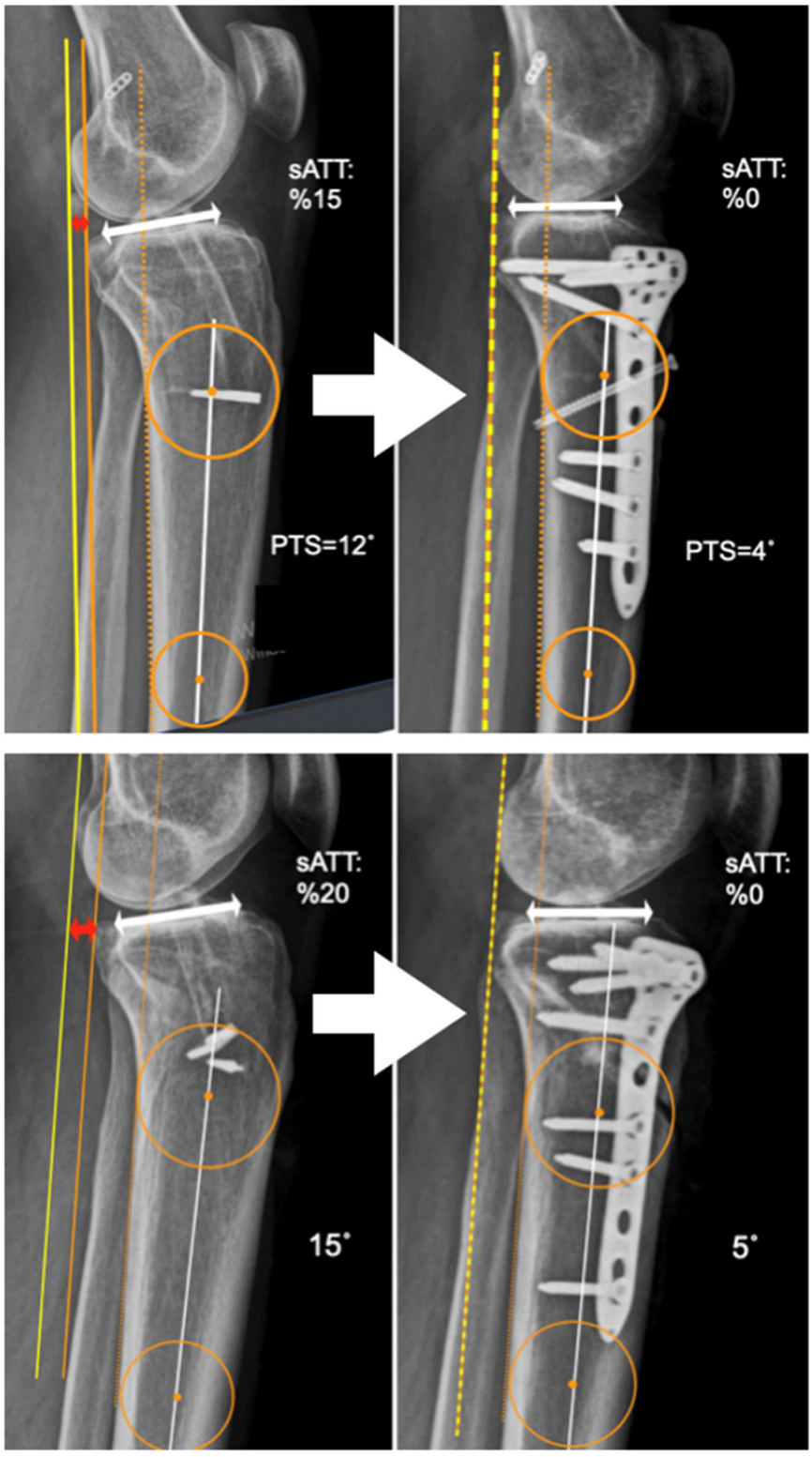

FIGURE 8 The power of osseous correction to restore the sagittal set-point. Preoperative (left) and postoperative (right) monopedal weight-bearing lateral radiographs demonstrating the definitive normalization of static anterior tibial translation (sATT) following slope-reducing osteotomy (SRO). (Top) A 37-year-old male presenting with chronic sagittal decompensation, demonstrating a preoperative sATT of 15% driven by a posterior tibial slope (PTS) of 12°. Following combined SRO + revision ACL-R, the osseous shear vector is neutralized (PTS reduced to 4°), allowing the joint to auto-recenter to a physiological sATT of 0% under axial load. (Bottom) A 33-year-old male presenting with a highly hostile structural phenotype (preoperative sATT of 20% and an extreme PTS of 15°). Following isolated SRO, surgically flattening the medial tibial plateau (PTS reduced to 5°) eliminates the anterior gravitational vector, completely restoring the sagittal equilibrium (sATT = 0%). These cases provide biomechanical proof that when functional resting subluxation is driven by underlying osseous geometry, modifying the bone is the definitive mechanism for recentering the tibia. ACL-R, anterior cruciate ligament reconstruction.

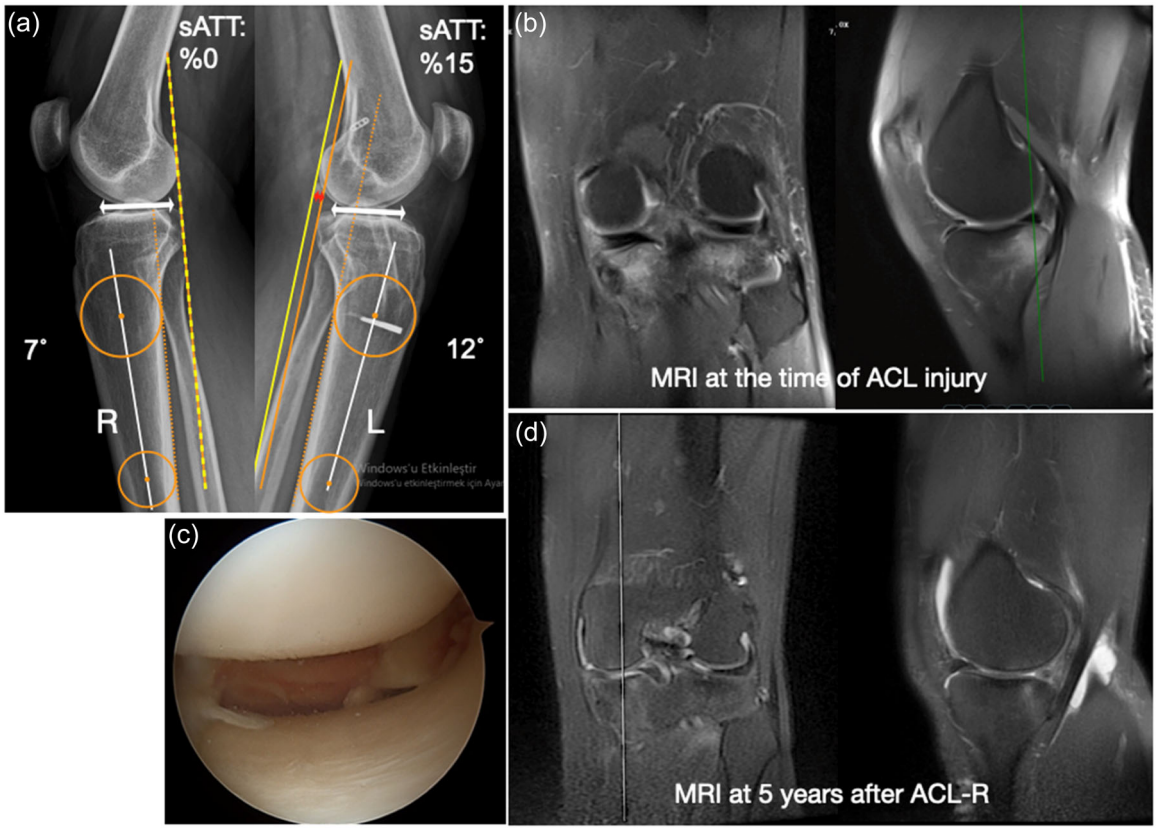

FIGURE 9 The synergistic role of meniscal loss and chronicity on sagittal decompensation. A 37-year-old male patient presenting with an anterior cruciate ligament reconstruction (ACL-R) failure. (a) Bilateral monopedal lateral knee radiographs demonstrate marked asymmetric sagittal position. The healthy right knee shows a posterior tibial slope (PTS) of 7° with 0% static anterior tibial translation (sATT), whereas the left knee demonstrates an increased PTS of 12° with an elevated sATT of 15%. (b) Magnetic resonance imaging (MRI) obtained approximately 5 years prior, immediately following the index ACL injury, confirms intact posterior horns of both menisci prior to the initial surgical intervention. The patient subsequently underwent ACL-R, which failed after 4.5 years. (c) Arthroscopic image obtained during revision surgery demonstrates a complete radial tear with a significant tissue defect of the medial meniscus posterior horn. (d) Corresponding MRI obtained 5 years after the index reconstruction confirms the chronic radial tear and meniscal tissue loss, highlighting how progressive secondary stabilizer failure amplifies slope-driven anterior translation over time.

deficient knees, compared with only a 0.8% increase in ACL-intact controls [8]. By utilizing the paired data from this recent percentage-based study, wherein a 1 mm absolute translation mathematically equates to an approximate 2.3% relative translation, historical findings reported exclusively in millimetres can be conceptually translated into percentage equivalents (e.g., a 5 mm absolute shift translates to roughly an 11.5% sATT%). This conversion ratio provides a useful bridge between legacy millimetre data and current percentage-based methods. However, definitively establishing physiological baselines and surgical thresholds will require future multi-centre studies that utilize direct, side-to-side comparisons within the same patients.

Despite variations in historical measurement modalities, the unifying mechanical conclusion remains clear: the ACL functions as a 'brake' on PTS-induced translation; once that brake is removed, the geometric influence of PTS is unleashed (Figure 6).

Clinical implication: Why soft-tissue reconstruction may not recenter the tibia

In the setting of high PTS, ACL-R can restore stress-based laxity limits while failing to normalize the tibial resting position under load [11, 35, 36] (Figure 7). An anatomical graft may reduce anterior excursion yet remain chronically exposed to the PTS-driven anterior vector. This distinction provides a biomechanically plausible explanation for why stress-based laxity measurements may not fully reflect the sagittal tibial resting position under weight-bearing conditions. This biomechanical vulnerability is mirrored in recent clinical findings demonstrating that while a steep PTS does not necessarily impair subjective functional outcomes after primary ACL-R, it significantly increases the risk of ultimate graft failure [39]. This dissociation highlights the inherent danger of relying on patient-reported functional scores to gauge true joint stability in structurally hostile environments.

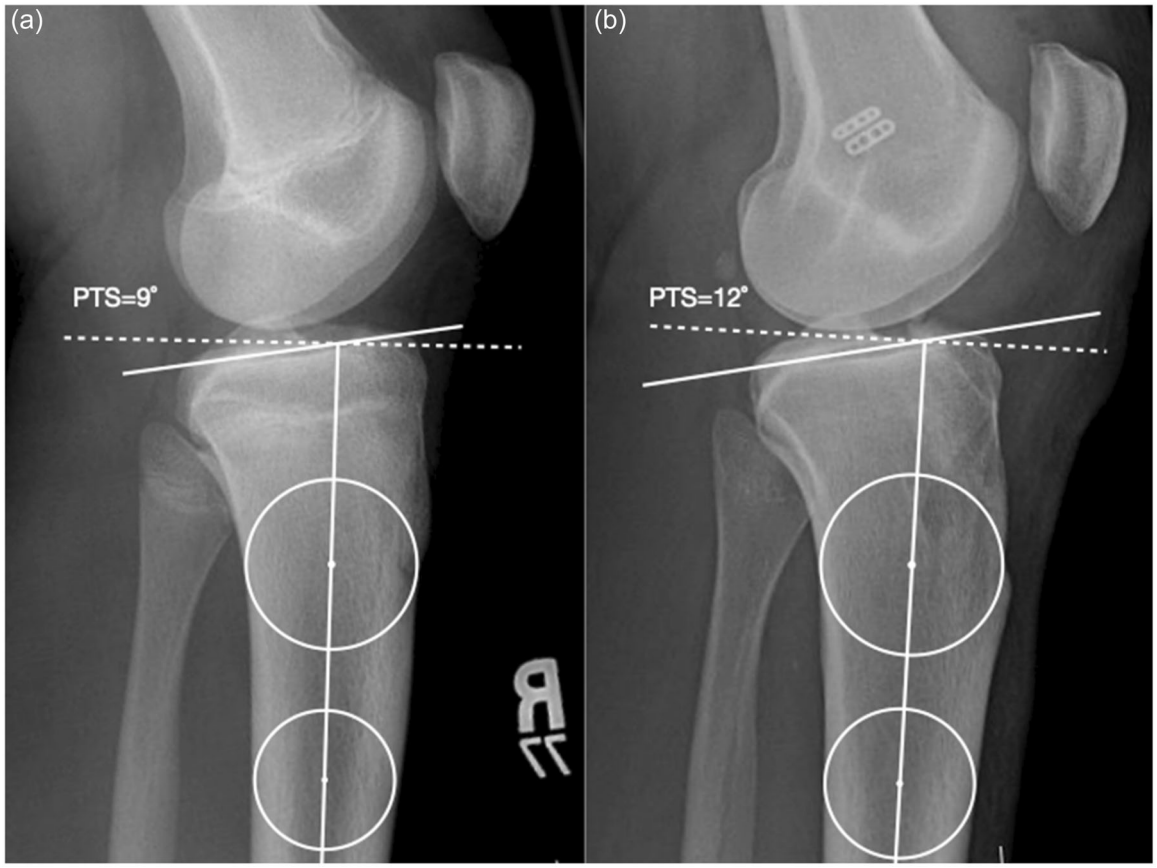

FIGURE 10 Longitudinal structural remodelling driven by chronic sagittal decompensation. Bipedal lateral radiographs of the index knee in a female patient, obtained 11 years apart. (a) The initial radiograph, taken at age 15 following an anterior cruciate ligament injury and concomitant medial meniscal tear, demonstrates a structurally neutral baseline posterior tibial slope (PTS) of 9°. (b) The 11-year follow-up radiograph (demonstrating hardware from two consequent reconstructions) reveals a progressive steepening of the tibial plateau, with the PTS increasing to 12°. This documented 3° increase over a decade visually confirms the phenomenon of acquired structural anteriorization: persistent altered kinematics and the chronic loss of the secondary meniscal wedge induce long-term osseous remodelling, creating a vicious cycle that further exacerbates the biomechanical hostility of the joint.

The power of osseous correction: Re-establishing the set-point

Conversely, directly addressing the underlying structural deformity can definitively restore the sagittal set-point. In severely decompensated knees with high PTS, combining a tibial SRO with revision ACL-R neutralizes the osseous shear vector while restoring the soft-tissue tether, reliably returning the sATT to a physiological 0%. However, the mechanical dominance of PTS is most profoundly illustrated by patients who undergo *isolated* SRO in the setting of chronic ACL graft failure. By surgically flattening the medial tibial plateau without revising the incompetent soft-tissue graft, the anterior gravitational vector is eliminated and the joint can auto-recenter to a 0% sATT under axial load (Figure 8). This phenomenon provides definitive in-vivo proof of the Structural Phenotype: when the resting subluxation is driven by osseous geometry, modifying the bone alone is sufficient to restore a stable functional equilibrium.

ACQUIRED SOFT-TISSUE ANTERIORIZATION (SAGITTAL DECOMPENSATION AND SECONDARY STABILIZER FAILURE)

Acquired soft-tissue decompensation following ACL injury is a long-recognized clinical phenomenon. As early as the 1980s, radiographic laxity studies demonstrated that anterior tibial translation increases with the duration of ACL deficiency, indicating a time-dependent loss of sagittal restraint [34]. However, isolating the independent effect of injury chronicity from progressive damage to secondary stabilizers, particularly the menisci, remains challenging.

Even in the absence of overt meniscal pathology, time alone contributes to sagittal decompensation. MRI-based studies evaluating ATS in full extension have demonstrated progressive anterior tibial positioning as early as [6–12] months following injury, likely

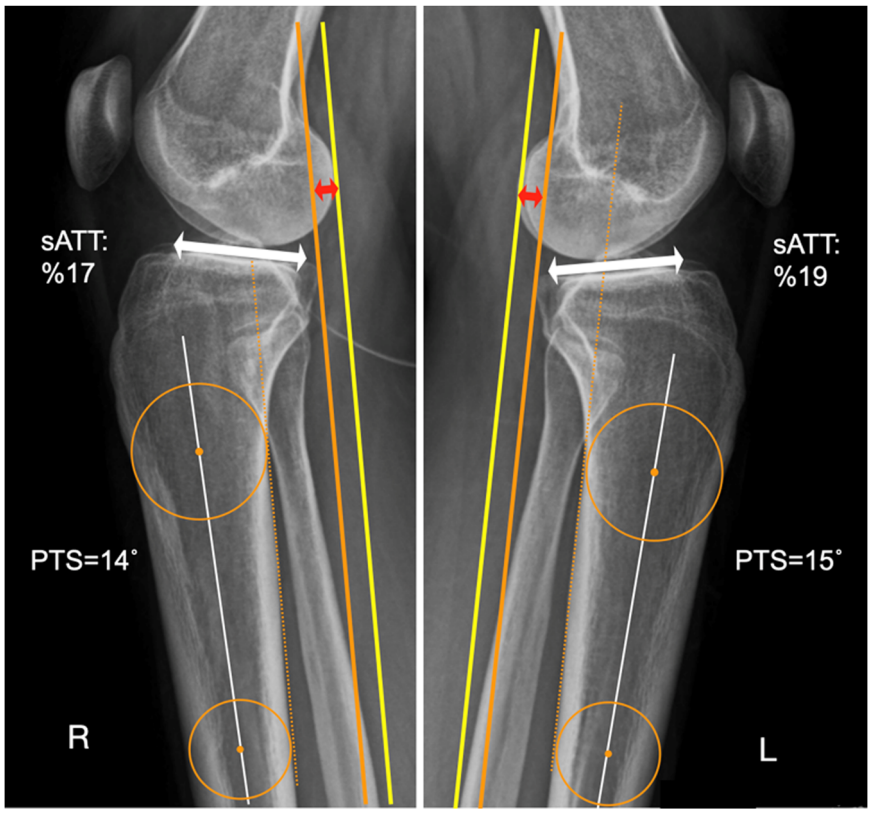

FIGURE 11 Symmetric constitutional anteriorization driven by generalized joint hypermobility. Bilateral monopedal weight-bearing lateral radiographs of a 27-year-old female with generalized hyperlaxity (Beighton score 5) and magnetic resonance imaging (MRI) confirming intact anterior cruciate ligaments (ACL) and menisci bilaterally. The dominant right knee (R) exhibits a posterior tibial slope (PTS) of 14° and a static anterior tibial translation (sATT) of 17%. The non-dominant left knee (L) demonstrates a similar osseous profile with a PTS of 15° and a corresponding sATT of 19%. Despite the absolute sATT values being elevated compared to normomobile populations, the side-to-side difference (ΔsATT) is minimal (2%). Within this framework, this symmetry confirms a benign, patient-specific constitutional resting sagittal position dictated by baseline soft-tissue compliance, rather than a unilateral pathological decompensation.

reflecting gradual capsuloligamentous creep and increasing compliance of the secondary soft-tissue envelope [32]. Cross-sectional analyses further identify injury chronicity, baseline anterior laxity and knee hyperextension as independent determinants of pre-operative ATS, with more pronounced effects observed beyond 2 years of ACL deficiency [18, 44].

Similar findings have been reported using force-controlled laxity testing, in which patients evaluated 1–20 years after ACL injury exhibited sustained sagittal instability during the transition from non-weightbearing to weightbearing compared with both contralateral knees and healthy controls [3].

The meniscal multiplier effect

Disentangling the effects of time and meniscal injury is often difficult, as they are inherently linked: most chronically ACL-deficient knees ultimately sustain meniscal damage. Nevertheless, evidence supports a synergistic relationship in which meniscal injury acts as an accelerant of sagittal decompensation (Figure 9).

Using monopedal weight-bearing radiography, Macchiarola et al. demonstrated that side-to-side differences in static anterior tibial translation (ΔsATT) progressively increase with time from injury, reaching approximately 4.2 mm in knees deficient for more than four years compared with 2.0 mm within the first year [27]. Crucially, this time-dependent increase was markedly amplified in meniscal-deficient knees, whereas knees with intact menisci maintained substantially better sagittal position.

Further evidence for this 'meniscal multiplier' effect comes from postoperative settings. Dejour et al. reported that medial meniscal deficiency significantly influences knee laxity following ACL reconstruction [11]. In multivariable regression analysis, partial medial meniscectomy was independently associated with an increase of approximately 2 mm in postoperative sATT.

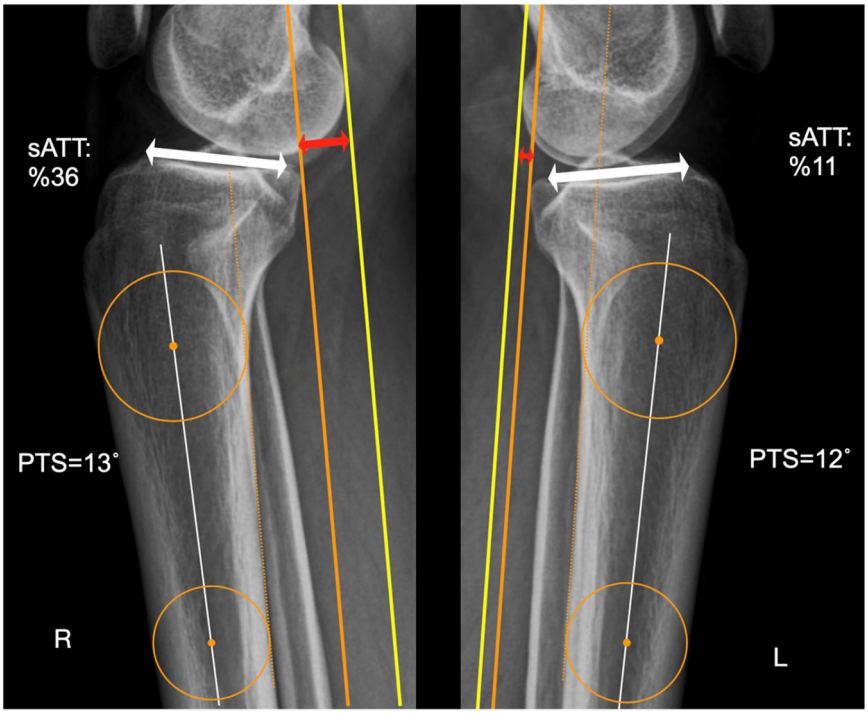

FIGURE 12 Acute pathological anteriorization superimposed on a hyperlax constitutional baseline. Bilateral monopedal weight-bearing lateral radiographs of a male patient with generalized joint hypermobility (Beighton score 5). The uninjured left knee (L) establishes the patient's constitutional and structural baseline, demonstrating a posterior tibial slope (PTS) of 12° and a static anterior tibial translation (sATT) of 11%. The right knee (R) sustained an acute (<1 month) anterior cruciate ligament (ACL) rupture with a concomitant lateral meniscal root tear. The affected side exhibits a slightly steeper PTS of 13°. Despite the acute timeframe precluding chronic tissue creep, the combined loss of the primary ligamentous restraint and a secondary meniscal stabilizer, superimposed on a highly compliant joint and borderline steep morphology, unleashes an extreme sATT of 36%. This profound pathological shift (ΔATT = 25%) illustrates how constitutional hyperlaxity and increased PTS act synergistically to lower the threshold for severe sagittal decompensation following acute structural injury.

Structural adaptation and the vicious cycle of sagittal decompensation

Longitudinal observations suggest that persistent anterior tibial positioning may induce structural remodelling. Martin et al. reported a progressive increase in lateral PTS on MRI of approximately 2° over nine years in untreated ACL-deficient knees [27]. In a skeletally mature cohort, Kayaalp et al. demonstrated an increase exceeding 2° in PTS in the same knees of patients undergoing revision ACL-R at long-term follow-up, particularly in the presence of medial meniscal deficiency [20]. These findings suggest a vicious cycle in which acquired soft-tissue anteriorization promotes bony remodelling, which in turn further amplifies sagittal decompensation (Figure 10).

Defining chronicity: A continuous risk spectrum

The definition of 'chronic' ACL deficiency varies widely in the literature (ranging from >6 weeks to >2 years), which obscures clinical decision-making [12, 26, 48]. However, the biological reality is a continuous accumulation of risk:

- *>3 months*: risk of postoperative residual anteriorization and/or instability increases [1].
- *>6 months*: risk of irreparable medial meniscal tears and abnormal pre-reconstruction sagittal position rises significantly [6].
- *>12 months*: high incidence of medial meniscal and chondral injury [50], often marking the transition to established sagittal decompensation.

Pronounced alterations in sagittal weight-bearing position, including increased ATS and side-to-side differences, are most evident in knees with ACL deficiency exceeding 2 years [44].

INHERENT SOFT-TISSUE ANTERIORIZATION

Generalized ligamentous laxity represents an inherent soft-tissue modifier of sagittal knee behaviour that operates independently of bony morphology or injury chronicity. Increased compliance of the posterior capsule, meniscocapsular junction and secondary soft-tissue restraints reduces passive resistance

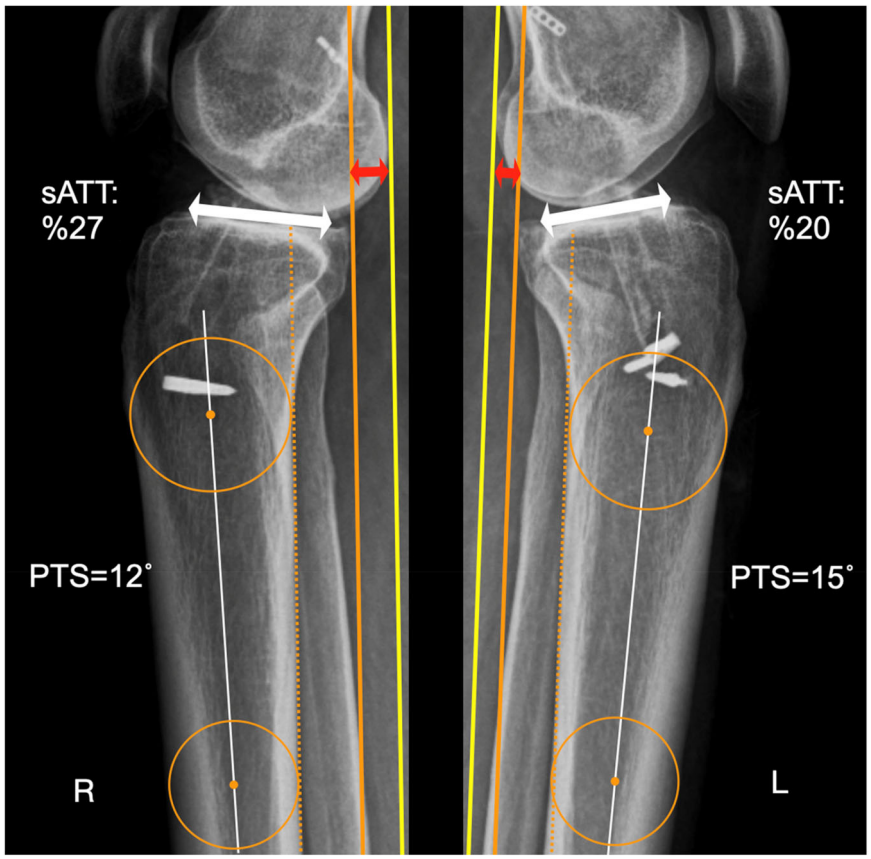

FIGURE 13 The 'double hit' phenomenon: Synergistic compounding of structural and functional anteriorization. Bilateral monopedal weight-bearing lateral radiographs of a 34-year-old male (Beighton score 0) with bilateral chronic anterior cruciate ligament reconstruction (ACL-R) failures. Because hyperlaxity is absent, the elevated resting sagittal positions are driven entirely by osseous and acquired soft-tissue double hits. The symptomatic left knee (L) demonstrates a structurally steep posterior tibial slope (PTS) of 15°, compounded by a recent ACL-R failure and a horizontal medial meniscal tear, resulting in a static anterior tibial translation (sATT) of 20%. Conversely, the right knee (R) illustrates a more extreme functional decompensation: despite a less severe PTS of 12°, a 7-year chronic ACL-R failure combined with a complete radial tear and absent posterior medial meniscal tissue unleashes a profound pathological resting position, with the sATT surging to 27%. This vividly confirms that the complete loss of the secondary meniscal wedge can synergistically compound with even moderate osseous geometry, driving the joint into severe sagittal decompensation.

to anterior tibial positioning, predisposing hyperlax knees to a more anterior resting position under physiological load. This effect is typically symmetric and bilateral, reflecting a systemic connective tissue phenotype rather than a unilateral pathological process (Figure 11).

Direct data quantifying sATT in generalized joint hypermobility (GJH) populations are limited. However, dynamic in vivo data suggest that a hypermobile phenotype directly influences weight-bearing kinematics. Zeng et al. demonstrated increased ATT during gait in GJH subjects compared with normomobile controls, even in the absence of structural injury [53]. This implies that inherent soft-tissue compliance likely exacerbates the shifted resting position seen in ACL-deficient knees.

Clinically, GJH has been associated with increased ACL injury risk and greater postoperative laxity after ACL-R [30, 41]. Moreover, Sundemo et al. demonstrated increased rotatory laxity in contralateral, uninjured knees of hyperlax patients, supporting a bilateral baseline effect [40]. Constitutional anteriorization should therefore be regarded as a background modifier that lowers the threshold for clinically relevant anteriorization when combined with adverse geometry, meniscal deficiency or ligament injury (Figure 12).

Importantly, increased ATT may be a patient-specific trait rather than purely an injury outcome. Kvist demonstrated that 'non-copers' exhibited anterior tibial positions on their *uninjured* side comparable to the *injured* side of 'copers' [23]. This suggests a constitutionally anterior tibial resting position, likely driven by PTS, laxity, or both and may predispose patients to poorer functional adaptation even before injury occurs. Higher preoperative ATT has similarly been associated with inferior postoperative results [38], reinforcing that constitutional anteriorization is a risk factor that may persist despite reconstruction.

In this context, bilateral imaging and side-to-side comparison remain informative, not to define

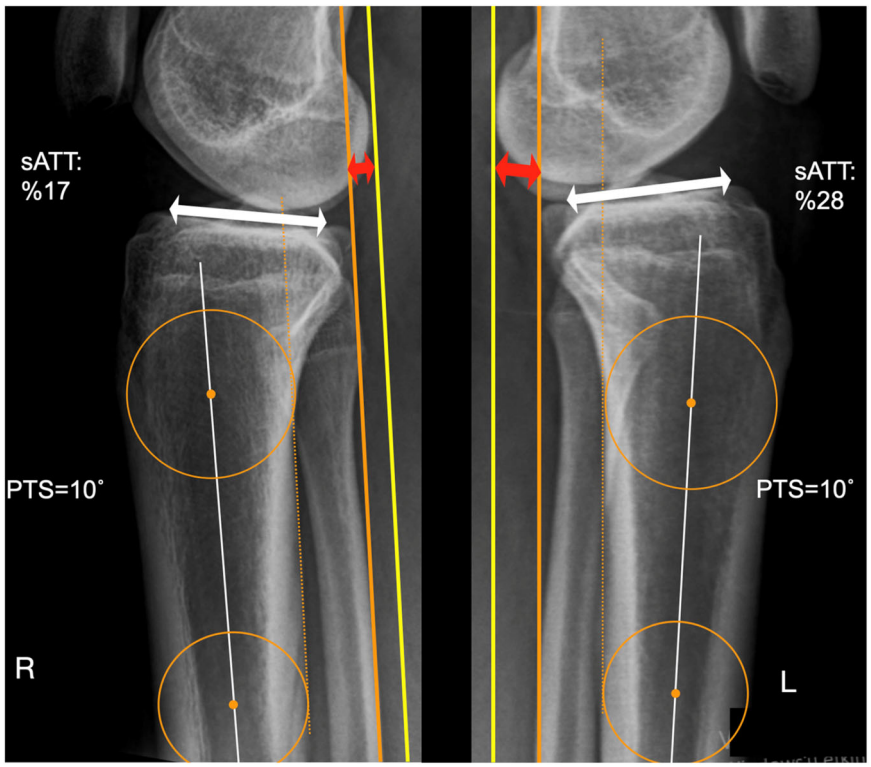

FIGURE 14 The 'double hit' of acute structural failure superimposed on an inherent soft-tissue hyperlax baseline. Bilateral monopedal weight-bearing lateral radiographs of a 17-year-old male presenting with a 1-month-old acute non-contact injury to the left knee. The patient demonstrates systemic hyperlaxity (Beighton score 5) and bilateral knee recurvatum. The uninjured right knee (R) establishes the patient's constitutional baseline, showing a normal posterior tibial slope (PTS) of 10° but an elevated static anterior tibial translation (sATT) of 17%, driven by physiological soft-tissue compliance. The injured left knee (L) sustained an anterior cruciate ligament (ACL) rupture with a concurrent lateral posterior horn tear expanding into the meniscal root. Despite the protective, flat osseous geometry (10° PTS) and the acute timeframe precluding chronic tissue creep, the sudden loss of the primary ligamentous and secondary meniscal restraints triggers a severe 'double hit' decompensation. The sATT on the affected side surges to 28%, creating a marked side-to-side discrepancy (ΔsATT = 11%). This vividly illustrates how an inherent soft-tissue laxity drastically lowers the threshold for extreme anteriorization following acute injury, underscoring the potential need for augmented surgical stabilization even in the absence of steep structural geometry.

'normality,' but to identify superimposed asymmetry beyond a patient-specific hyperlax baseline.

THE 'DOUBLE HIT' PHENOMENON: SYNERGISTIC COMPOUNDING OF RISK

While osseous, acquired soft-tissue-based and inherent soft-tissue-based anteriorization can independently drive elevated sATT, they most frequently present in tandem. A 'double hit' occurs when two distinct mechanisms converge, synergistically increasing the mechanical burden on the central pivot. The most common clinical manifestation is the combination of osseous and acquired soft-tissue-based anteriorization: for example, a patient with a steep PTS who sustains a meniscal tear or undergoes chronic tissue creep (Figure 13). In this scenario, the osseous geometry provides the continuous anterior shear vector, while the loss of the posterior meniscal wedge removes the critical secondary restraint, unleashing the PTS's full mechanical influence.

Alternatively, an inherent soft-tissue anteriorization (generalized hyperlaxity) combined with an acute structural failure (ACL and meniscal injury) can produce a profound decompensatory effect, even without severe chronicity or extreme PTS. These compounding 'double hits' represent the exact clinical threshold where isolated soft-tissue reconstruction becomes biomechanically insufficient. In these highly hostile environments, an SRO may be necessary to permanently alter the bony morphology and restore sagittal equilibrium (Figure 14). Concurrently or alternatively, lateral extra-articular procedures may be indicated to act as a protective tether to protect the ACL graft; recent dynamic in vivo data suggest these procedures also provide a temporary restriction of sagittal translation during the vulnerable early postoperative healing phase [5].

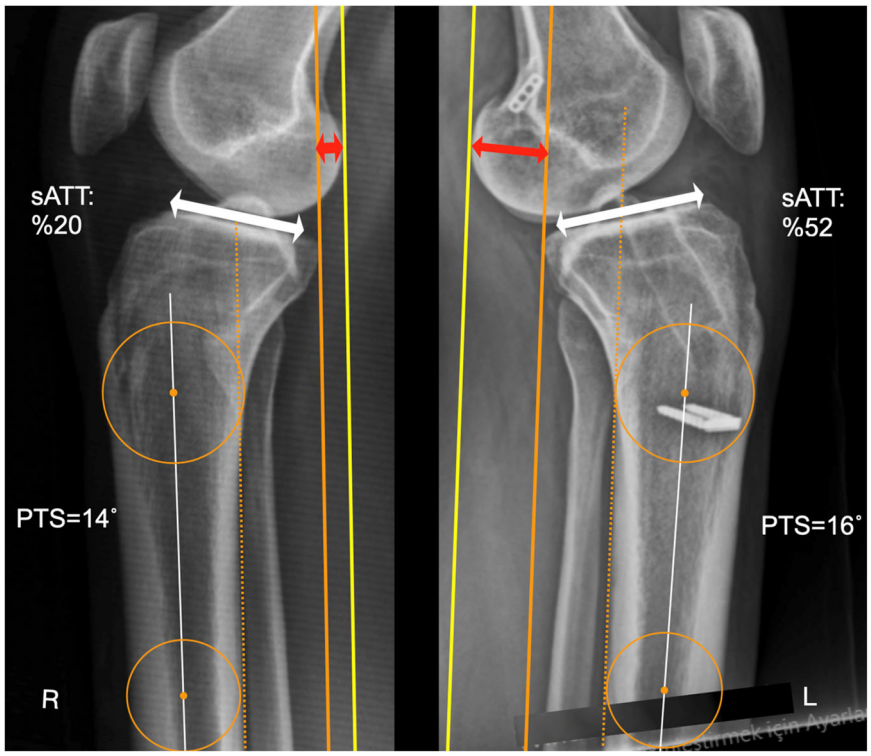

FIGURE 15 The 'triple hit' phenomenon resulting in extreme sagittal decompensation and atraumatic graft failure. Bilateral monopedal weight-bearing lateral radiographs of a 23-year-old female presenting with an atraumatic anterior cruciate ligament reconstruction (ACL-R) failure on the left knee (L). This case illustrates the compounding effect of concurrent isolated confounders. The uninjured right knee (R) demonstrates an osseously and inherently elevated baseline static anterior tibial translation (sATT) of 20%, driven by generalized hyperlaxity (Beighton score = 5) and a posterior tibial slope (PTS) of 14°. On the affected left knee, the convergence of a steeper osseous morphology (PTS = 16°), inherent hyperlaxity and severe injury chronicity (index ACL-R performed 7 years post-injury, allowing for catastrophic acquired secondary stabilizer creep) culminates in an extreme sATT of 52%. This near-subluxation state highlights how the 'triple hit' exponentially deteriorates the sagittal set-point, creating a hostile biomechanical environment that dooms isolated soft-tissue reconstruction.

THE 'TRIPLE HIT' PHENOMENON: SYNERGISTIC EFFECTS OF CONCURRENT CONFOUNDERS

When a patient presents with multiple concurrent risk factors, the resulting sATT can reach extreme values that cannot be attributed to a single variable, such as isolated increased PTS. This convergence of osseous, acquired soft-tissue-based and inherent soft-tissue-based anteriorization constitutes the 'triple hit' phenomenon, a worst-case biomechanical scenario representing a phenotype at extreme risk for primary and recurrent ACL graft failure.

A classic presentation involves a patient with generalized hyperlaxity (inherent soft-tissue anteriorization), an increased PTS (osseous anteriorization) and a chronic ACL tear with concomitant meniscal deficiency (acquired soft-tissue anteriorization). Because of their hyperlaxity, these patients already operate with an elevated baseline sATT. This fragile equilibrium is severely disrupted upon ACL injury, as the steep osseous geometry is unleashed. Over time, the chronicity of the injury and the progressive failure of secondary meniscal stabilizers further degrade sagittal containment, culminating in catastrophic instability and near-subluxation sATT values (Figure 15).

Clinically, this phenotype may help explain selected cases of early or recurrent ACL-R failure in which multiple sagittal risk factors coexist. However, direct evidence defining this presentation as a distinct clinical entity remains limited. Therefore, these patients should be evaluated using a comprehensive, kinematics-focused approach that considers PTS, sATT, generalized laxity, injury chronicity and secondary stabilizer deficiency. In selected high-risk cases, particularly when increased PTS is accompanied by persistent sagittal anteriorization, SRO osteotomy may be considered as an adjunct to ligamentous revision rather than relying on isolated soft-tissue reconstruction alone.

CONCLUSION

The functional resting position of the knee is a complex interplay of osseous geometry, soft-tissue compliance and time. Recognizing these distinct

phenotypes—osseous, acquired soft-tissue and inherent soft-tissue—is the first step towards moving beyond passive laxity limits. Part 2 of this series will build on this phenotypic framework by introducing the assessment-led personalization system, a clinical algorithm designed to measure, interpret and surgically address these specific sagittal phenotypes.

AUTHOR CONTRIBUTIONS

All listed authors contributed substantially to this work. All authors read and approved the final manuscript for submission and publication. *Writing—original draft, writing—review and editing*: Mahmut Enes Kayaalp. *Writing—review and editing*: Hamit Caglayan Kahraman, Tunay Erden, Christoph Lutter, Sven Scheffler, Omer Taser, Roland Becker, Michael T. Hirschmann and David H. Dejour.

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DATA AVAILABILITY STATEMENT

Available upon request.

ETHICS STATEMENT

The authors have nothing to report.

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