

Multimodal assessment of compensatory strategies during sit-to-stand in knee osteoarthritis

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ABSTRACT

Sit-to-stand (STS) is one of the most frequently performed daily tasks and a key functional test to evaluate mobility and neuromuscular performance in individuals with knee osteoarthritis (KOA).

While clinical STS tests mainly quantify performance through time or repetition counts, these metrics do not capture how the task is executed or the biomechanical and neuromuscular adaptations that sustain task completion. This review summarizes the main compensatory mechanisms observed during STS in KOA and discusses how they can be quantified as biomarkers to support functional assessment and clinically relevant prediction.

Across the STS phases, KOA is commonly associated with increased anterior trunk flexion to redistribute mechanical demand toward the hip, interlimb loading asymmetry with reduced contribution of the affected limb and greater reliance on the contralateral side, and neuromuscular adaptations such as delayed quadriceps activation and increased antagonist co-contraction that may enhance perceived stability but increase compressive loading. Upper limb assistance is also frequently adopted, providing additional impulse and reducing lower limb joint moments. Quantitative assessment using kinematics, kinetics, and surface electromyography, potentially integrated through multimodal approaches, offers a pathway to characterize heterogeneous compensatory profiles and track change over time.

KEYWORDS

Biomechanics, sit-to-stand, osteoarthritis, compensatory strategies, knee.

Introduction

Knee osteoarthritis (KOA) causes pain, stiffness, and muscle weakness that compromise key functional tasks such as walking, stair climbing, and sit-to-stand (STS)^[1-3].

STS is among the most frequently performed daily tasks and, given its biomechanical complexity, serves as one of the primary functional tests to evaluate neuromuscular performance and mobility limitations^[4-6]. The task requires coordinated control of the trunk and lower limb segments under rapidly changing stability conditions, and this control is further challenged by pain, reduced quadriceps strength, and joint stiffness, which collectively constrain the feasible set of movement solutions and promote compensatory strategies^[7-10].

In clinical practice, STS performance is primarily quantified through timing metrics, such as the 30 Second Chair Stand Test and the Five Times Sit to Stand Test, which are useful for monitoring functional impairment^[11]. However, beyond timing, comprehensive biomechanical evaluation is essential to capture compensatory strategies and underlying neuromuscular alterations^[11,12-14].

During STS, people with KOA often use compensatory movement patterns to minimize pain and perceived instability. These may include altered trunk and lower limb kinematics, interlimb loading asymmetries characterized by reduced

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contribution of the more affected limb and greater reliance on the contralateral side during seat-off and early extension, and neuromuscular adaptations such as modified timing of extensor activation and increased antagonist co-contraction, potentially improving perceived stability but increasing joint compressive loading^[13-15]. Upper limb support is also frequently used, providing additional impulse and reducing lower limb joint moments.

Collectively, these compensatory mechanisms indicate that KOA affects the entire kinetic chain, resulting in a global reorganization of movement strategies during STS^[16]. Quantifying these adaptations is therefore essential for accurate functional assessment and for designing targeted interventions aligned with each patient's compensatory profile^[17].

Rapid advances in motion analysis technologies, wearable sensors, and machine learning enable the extraction of STS-derived digital biomarkers and the development of models for disease stratification, prognostication, and prediction of treatment response^[11,16].

This review provides a synthesis of compensatory biomechanical mechanisms during STS in KOA and discusses how these adaptations can be quantified and leveraged as digital biomarkers to support functional assessment and prediction of clinically relevant outcomes.

Biomechanical framework of sit-to-stand

Task phases and mechanical demands

STS can be described through three sequential phases, each associated with distinct kinematic, kinetic, and neuromuscular requirements [16,18–20]. During Phase 1, the flexion momentum phase, the trunk and pelvis rotate forward to translate the whole-body center of mass anteriorly toward the feet, generating forward momentum and preparing the system for seat-off [20]. The timing and magnitude of this forward momentum influence subsequent stability demands and joint loading distribution. Phase 2, the momentum transfer phase, begins at seat-off, when the base of support is reduced and mechanical demands peak [20,21]. Ground reaction forces rise rapidly, and coordinated hip and knee extensor activation is required to redirect the center of mass upward while controlling balance. Phase 3, the extension and stabilization phase, involves progressive extension of the hip, knee, and trunk toward upright standing, with postural control mechanisms attenuating residual oscillations and ensuring steady stance [20,21]. Deficits in force generation or balance regulation typically manifest as prolonged phase durations, altered joint loading distribution, and increased reliance on compensatory strategies (Figure 1).

Biomechanical outcomes and assessment method

Clinical STS tests primarily quantify performance using task time or repetition counts, but a biomechanical assessment is required to identify compensatory strategies, distinguish dif-

ferent motor solutions, and derive digital biomarkers. In this context, kinematic analyses are commonly used to quantify sagittal plane ranges of motion of the trunk, hip, knee, and ankle across the different phases of the task, together with trunk flexion or lateral lean, which may indicate strategies adopted to reduce knee loading [3,15]. Complementary kinetic measures such as ground reaction forces and external knee flexion torque can further discriminate individuals with KOA, who typically exhibit lower peak values than healthy controls [3,9].

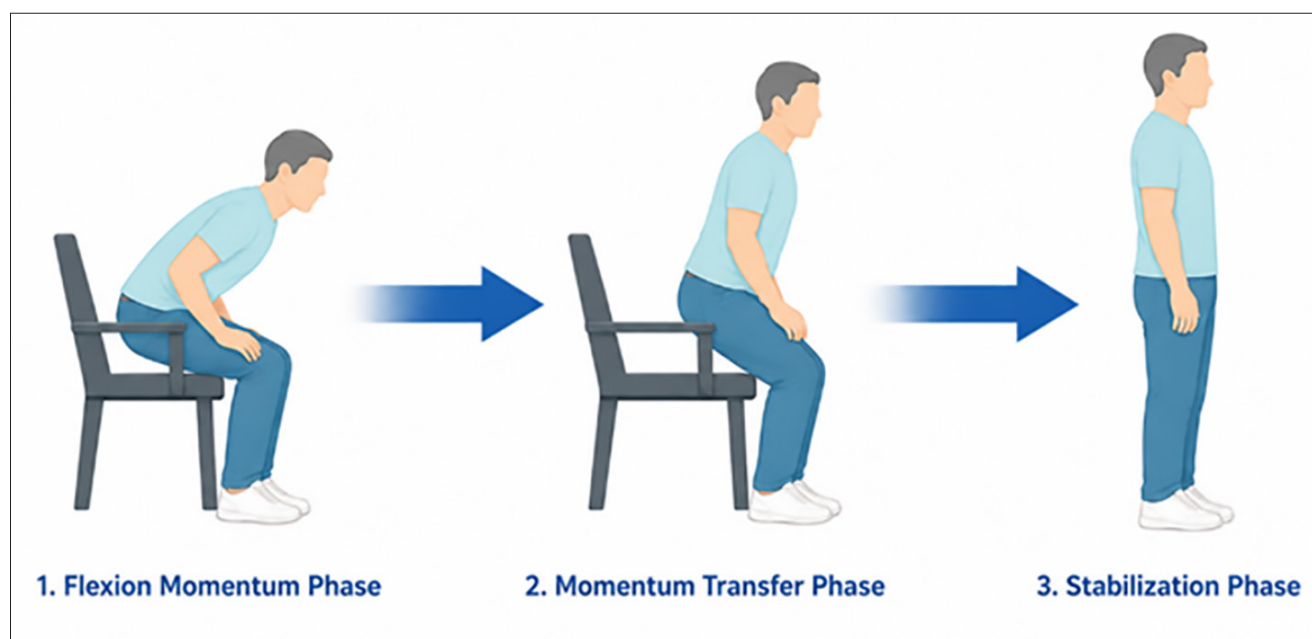
Neuromuscular control can be characterized using surface electromyography (EMG) by extracting features that capture both the magnitude and the organization of muscle activity [15,22]. Common metrics include the co-contraction index, which reflects the degree of simultaneous activation of antagonist muscle groups and is often elevated when compensatory stabilization strategies are adopted, as well as amplitude-based descriptors such as the root mean square and frequency domain features that provide additional insight into changes in activation profiles [15,22].

Finally, dynamic balance can be quantified using center of mass and center of pressure trajectories and their time derivatives around seat-off, providing insight into how momentum is generated and regulated even when overall task duration appears unchanged.

Biomechanical alterations during STS in KOA

KOA alters the biomechanical demands of STS through multiple mechanisms, with interacting impairments becoming most evident around seat-off, when stability demands increase and extensor demands peak [3,11]. Pain, joint stiffness, and quadriceps weakness constrain knee excursion and reduce the ability to generate the knee extensor torque needed for an effi-

Figure 1 STS phases.



cient rise, thereby shaping the selection of movement solutions^[3]. In parallel, impaired sensorimotor control may disrupt the timing and scaling of muscle activation, promoting increased co-contraction and altering joint loading^[14,23]. Together, these factors increase task difficulty and bias motor behavior toward strategies that favor pain avoidance and perceived stability rather than mechanical efficiency^[3,24].

A frequent adaptation is increased anterior trunk flexion, particularly during the flexion momentum phase and around seat-off. By shifting the whole-body center of mass anteriorly and increasing hip extensor leverage, greater trunk flexion facilitates a proximal redistribution of demand, reducing the relative reliance on knee extensor contribution while maintaining the ability to complete the task^[6].

Beyond kinematics, KOA is also associated with systematic changes in neuromuscular control^[15,22]. Surface EMG studies commonly report delayed quadriceps activation and prolonged activity patterns during the momentum transfer phase, consistent with an attempt to control stability under peak demands. Increased co-contraction between knee extensors and flexors, and in some cases ankle stabilizers, further stiffens the limb and may improve perceived joint stability^[23,25]. However, this stabilizing strategy can increase tibiofemoral compressive loading and energetic cost^[23]. Muscle synergy analyses indicate a relative shift from extensor-dominant synergies toward synergies primarily involved in stabilization, consistent with a more robust but less efficient motor control strategy. For unilateral KOA, a prominent compensatory feature is interlimb asymmetry, with reduced contribution of the affected limb and greater reliance on the contralateral side during seat-off and early extension^[15].

This pattern is reflected by lower vertical ground reaction forces of the affected limb and reduced symmetry indices compared with healthy controls, and it is often accompanied by lateral adjustments that facilitate weight transfer and increase

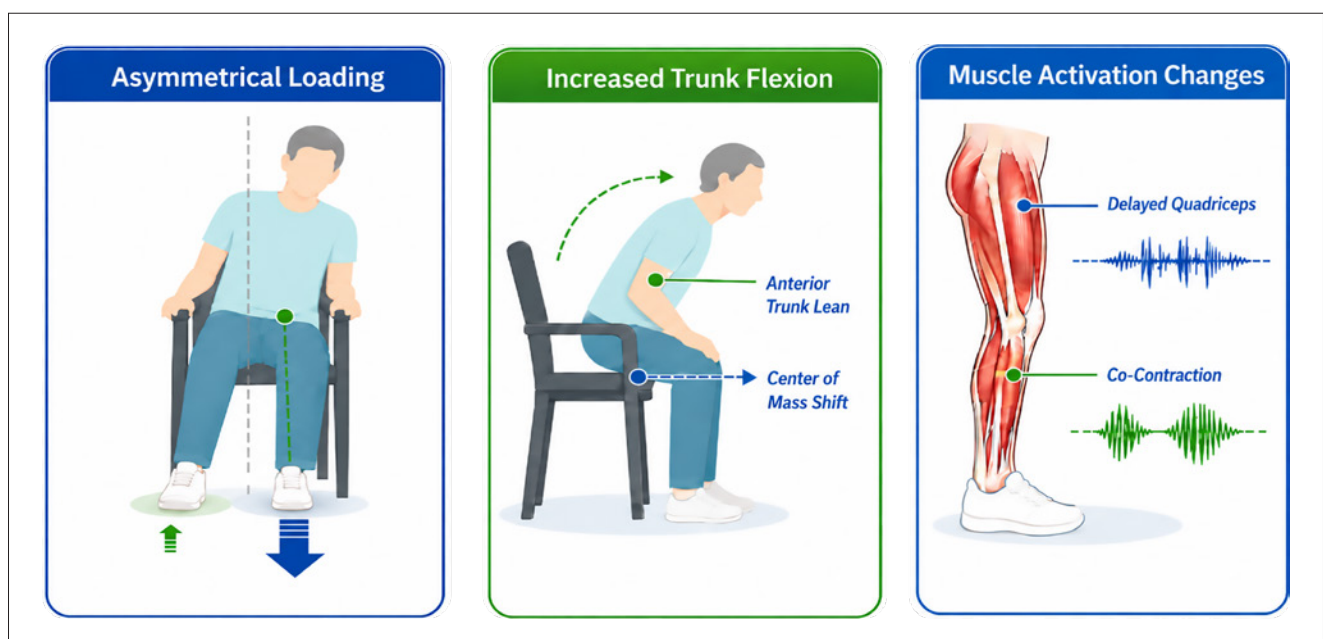
contralateral joint moments^[26]. From a clinical perspective, persistent contralateral overloading is relevant because it may contribute to degenerative changes in the less affected limb^[15]. Force platform-derived measures, including symmetry metrics and center of pressure trajectories that shift toward the contralateral limb, provide sensitive descriptors of these compensatory behaviors.

Compensatory strategies during STS should also be interpreted in relation to compartment involvement, as different KOA phenotypes may impose distinct mechanical constraints. Medial and lateral compartment OA may be associated with different weight shift, frontal plane, and trunk adaptations aimed at redistributing tibiofemoral loading. Therefore, stratifying patients according to compartment involvement may improve the clinical interpretation of STS-derived biomarkers and support more individualized rehabilitation planning (Figure 2).

Upper limb assistance represents an additional strategy frequently adopted in KOA^[27]. Arm support can contribute substantially to the vertical impulse during the transition, reducing lower limb joint moment requirements, particularly at the knee, and facilitating task completion in individuals with limited extensor capacity or high pain levels^[8,27]. This strategy is most evident around seat-off, when mechanical demands peak and balance demands increase. However, upper limb assistance also modifies overall movement coordination and can alter trunk kinematics and the center of mass trajectory, with potential implications for postural control during the extension and stabilization phase^[8,28]. While occasional arm use may represent an adaptive safety strategy, a strong dependence on upper limb support is typically interpreted as a marker of more advanced functional limitation and reduced lower limb capacity.

The identification of these compensatory profiles may help clinicians define patient-specific rehabilitation targets, monitor changes over time, and evaluate whether an intervention modifies not only task performance but also movement quality.

Figure 2 Schematic overview of representative compensatory mechanisms during STS in knee osteoarthritis.



Multimodal fusion and functional phenotyping

The assessment of compensatory strategies during STS can provide clinically relevant information on movement quality, joint loading, postural control, and neuromuscular adaptations. However, these dimensions cannot be fully characterized by a single assessment modality, as each method provides information on a specific component of the task. Integrating kinematics, kinetics, and neuromuscular signals supports a more complete representation of these strategies during STS and may better characterize heterogeneous movement solutions in KOA. Multimodal fusion can be achieved by combining descriptors extracted from different signals or by integrating predictions generated by modality-specific models ^[1,29].

Multimodal approaches are especially relevant for functional phenotyping, where the goal is to identify distinct compensatory profiles that may share similar task duration yet differ substantially in loading asymmetry, kinematic redistribution, and neuromuscular stabilization. In this context, features that relate to functional limitation and pain, such as asymmetry indices, trunk angular velocity, and altered EMG timing, represent candidate digital biomarkers for stratifying severity, guiding targeted rehabilitation strategies, and monitoring change over time, including outside the laboratory when the use of wearable sensors is feasible ^[11,29].

Future directions

Future research should aim to further develop STS-based digital biomarkers and predictive models in KOA, transforming these promising laboratory measures into reliable, generalizable, and clinically useful tools. Protocol heterogeneity remains a major barrier, since chair height, foot placement, movement speed, and allowance of upper limb support can substantially alter mechanics and reduce cross-study comparability, in turn weakening model portability. Sample size and cohort diversity are also critical, as models trained on small or homogeneous datasets risk poor generalization to broader clinical populations. Finally, multimodal integration introduces additional challenges related to synchronization, alignment, and missing data handling when kinetics, kinematics, and EMG are acquired with different systems.

Future work should prioritize standardized acquisition protocols where feasible, shared reporting of key task constraints, and multimodal datasets with synchronized signals and longitudinal follow-up. These steps are central to building validated models that are robust, clinically interpretable, and ready for deployment as digital biomarkers for stratification and monitoring in KOA.

Conclusion

Objective multimodal analysis of STS may provide clinically relevant information beyond conventional performance-based tests by capturing how the task is performed

rather than only whether it is completed. By integrating biomechanical and neuromuscular measures, this approach may help characterize compensatory strategies, support patient stratification, guide individualized rehabilitation planning, and monitor functional changes over time in individuals with knee osteoarthritis.

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