Title page

Title:

Changes in postural control strategy during quiet standing in individuals with knee osteoarthritis.

Authors:

Kento Sabashi[1,2], Satoshi Kasahara[1], Harukazu Tohyama[1], Takeshi Chiba[1,2], Yuta Koshino[1], Tomoya Ishida[1], Mina Samukawa[1], Masanori Yamanaka[3]

[1] Faculty of Health Sciences, Hokkaido University, Kita 12, Nishi 5, Kita-ku, Sapporo, Hokkaido, 060-0812, Japan.

[2] Department of Rehabilitation, Hokkaido University Hospital, Kita 14, Nishi 5, Kita-ku, Sapporo, Hokkaido, 060-8648, Japan.

[3] Faculty of Health Science, Hokkaido Chitose College of Rehabilitation, Satomi 2-10, Chitose, Hokkaido, 066-0055, Japan.

Corresponding author:

Satoshi Kasahara, PT, PhD

Department of Rehabilitation Science, Faculty of Health Sciences, Hokkaido University

Kita 12, Nishi 5, Kita-ku, Sapporo, Hokkaido, 060-0812, Japan

Tel/Fax: +81-11-706-3390

E-mail: kasahara@hs.hokudai.ac.jp

Abstract.

BACKGROUND: Knee osteoarthritis (OA) impairs postural control, which may be affected by how to use lower limb joints.

OBJECTIVE: To investigate how individuals with knee OA use lower limb joints for static postural control.

METHODS: Ten patients with knee OA and thirteen healthy controls performed quiet standing for 30 s. The standard deviation of the center of mass (COM) and lower limb joint motions in the anterior-posterior (AP) and medial-lateral (ML) planes were calculated from three-dimensional marker trajectories. Pearson's correlation analysis and independent *t*-tests were conducted to investigate the relationship between COM and lower limb joint motions and to compare group difference, respectively.

RESULTS: The AP hip angular velocity alone in the knee OA group and the AP hip and knee angular velocity in the control group were significantly correlated with the AP COM velocity. The ML hip angular velocity was significantly correlated with the ML COM velocity in both groups. The knee OA group exhibited a significantly larger standard deviation of AP COM velocity than the control group.

CONCLUSIONS: Individuals with knee OA depended solely on the contribution of the hip to the AP COM velocity, which could not be successfully controlled by the knee.

Keywords:

Knee osteoarthritis, Quiet standing, Postural control, Center of mass, Kinematics

1. Introduction

Osteoarthritis (OA) of the knee is substantially prevalent in the elderly [1,2]. Patients with knee OA have knee pain [3], quadriceps weakness [4], limited knee range of motion [5], impaired proprioception of the knee [6], and structural changes (e.g., knee malalignment and cartilage degradation) [7]. Consequently, knee OA is considered one of the main causes that limits activities of daily living [8], deteriorates the quality of life [9], and increases the likelihood of falling [10]. The prevention of falls is a research priority in patients with knee OA because falls may induce incident fractures [10–12].

Poor postural control in patients with knee OA is considered to increase the likelihood of falling [13]. Patients with knee OA exhibit impairments in postural control, including increased postural sway during quiet standing compared to healthy adults [14]. Previous studies demonstrated that the amount of postural sway in this patient population is associated with knee pain [15,16], severity of knee OA [17], and knee muscle strength [18]. For the clinical assessment and rehabilitation of individuals with knee OA, it is important to understand how these factors affect joint motion during postural control.

The goal of static postural control is to stabilize the center of mass (COM) within the base of support. All joint motions, particularly those in the lower limbs, play a vital role in postural control. In general, the maintenance of equilibrium in the anterior-posterior (AP) and medial-lateral (ML) planes during quiet standing is mainly controlled by ankle and hip joint motions [19,20], while a recent study showed that knee joint motion additionally contributes to static postural control [21]. Individuals with knee OA have impaired hip and ankle joint functions as well as knee joint functions [4–6,22–25]. However, it remains unknown whether the use of lower limb joints for postural control in this patient population differs from that in healthy adults. Therefore, the purpose of this study was to investigate the changes in postural control strategies during quiet standing in individuals with knee OA. We hypothesized that

the lower limb joint motions used to control the COM motion were different between individuals with knee OA and age-matched healthy controls.

2. Methods

This study was a case-control type investigation. We received ethical approval from the Institutional Review Board of our institute, and written informed consent was obtained from all participants.

2.1. Participants

Twenty-three participants, ten individuals diagnosed with unilateral or bilateral knee OA (one male and nine females) and thirteen healthy controls (three male and ten females), participated in this study (Table 1). Knee pain in patients with knee OA was assessed using a visual analogue scale (VAS), which ranged from 0 (no pain) to 100 mm (severe pain), and the average VAS score was 23.8 mm. All participants with and without knee OA were recruited from several hospitals and local communities. *A priori* power analysis was conducted using the t-test model of G*Power 3.1.9.2 (Kiel University, Kiel, Germany). Based on our pilot study of knee angular velocity data with seven participants (three patients with knee OA and four healthy controls), the minimum sample size for this study was 10 participants per group (effect size = 1.39, alpha = 0.05, and power = 0.80).

Participants in the knee OA group were included if they were aged 50–79 years and had radiographically diagnosed knee OA with a Kellgren–Lawrence grade 2–4 in at least one knee [26]. Potential participants in both groups were excluded if they had any previous lower limb or back surgery, severe disability in walking and standing without an assistive device, any musculoskeletal disorder other than knee OA that would influence balance, or any neurological disorder.

101

102 *2.2. Experimental protocol*

103 All participants were instructed to stand as still as possible and to keep looking
104 straight ahead with their feet hip-width apart and their arms folded across the chest. During
105 testing, participants were not provided with any information on body sway. The examination
106 was performed twice for 30 s.

107 All data were collected using a motion-capture system (Cortex; Motion Analysis
108 Corporation, Santa Rosa, CA, USA), which consisted of seven cameras (Hawk cameras;
109 Motion Analysis Corporation) with a sampling rate of 200 Hz. According to the modified
110 Helen Hayes marker set [27], 25 reflective markers were positioned at the following
111 anatomical landmarks: dorsum of the foot, heel, lateral and medial malleoli, lateral shank,
112 lateral and medial femoral epicondyle, lateral thigh, anterior superior iliac spine, sacral,
113 acromion, lateral humeral epicondyle, and wrist. All markers, except the sacral, were attached
114 bilaterally.

115

116 *2.3. Data analysis*

117 All signals were processed using a custom MATLAB program (MathWorks Inc.,
118 Natick, MA, USA). Three-dimensional marker trajectories were low-pass filtered at a cutoff
119 frequency of 5 Hz using a fourth-order Butterworth filter [28].

120 The time series of AP and ML COM positions were calculated from the marker data
121 based on previously reported anthropometric data [29]. The time series of AP and ML COM
122 velocities were calculated as the first-time derivatives of COM positions. The following
123 COM-based parameters in each direction were calculated to assess the amount of body sway
124 during quiet standing: (1) standard deviation (SD) of COM position and (2) SD of COM
125 velocity [20,30].

126 The time series of AP and ML hip, knee, and ankle angles were calculated using
127 Visual 3D software (C-Motion Inc., Germantown, MD, USA). Subsequently, the time series
128 of AP and ML angular velocities were calculated as the first-time derivatives of each lower
129 limb joint angle. Knee joint motion analysis in the ML plane, or knee adduction-abduction,
130 was excluded based on the method presented in [31]. Additionally, the following kinematic
131 parameters in each direction were calculated to assess the amount of lower limb joint motions
132 during quiet standing: (1) SD of lower limb joint angle and (2) SD of lower limb joint angular
133 velocity [20]. The affected limb in patients with unilateral knee OA, the most affected limb in
134 patients with bilateral knee OA, and a randomly selected limb in healthy controls were
135 adopted in this study.
136 To compare the differences in standing posture, the mean values of COM positions
137 and lower limb joint angles during static standing were calculated. The mean values of COM
138 positions were defined as the distance from the midpoint of the bilateral heel markers in the
139 AP and ML planes. The mean values of COM positions in the AP plane were expressed as
140 positive if the COM position was anterior relative to the midpoint of the bilateral heel
141 markers, while those in the ML plane were expressed as positive if the COM position was in
142 the direction of the study limb relative to the midpoint of the bilateral heel markers. In
143 addition, lower limb joint angles in the AP plane were represented as positive for flexion
144 (dorsiflexion), while those in the ML plane were represented as positive for adduction
145 (inversion).
146
147 *2.4. Statistical analysis*
148 An independent *t*-test was used to compare the demographic data, the mean values of
149 COM positions, the mean values of lower limb joint angles, the SDs of COM position and
150 velocity, and the SDs of lower limb joint angles and angular velocities between the groups.

Then, the mean difference (the knee OA group minus the control group) and 95% confidence interval (CI) for each variable was calculated. Pearson's product correlation analysis was performed to investigate the relationship between lower limb joint motion and COM motion in the AP and ML planes in each group. In addition, Pearson's product correlation analysis was carried out to investigate the associations of the amount of body sway and lower limb joint motions with knee pain in patients with knee OA. The statistical significance level was set at $P < 0.05$. All statistical analyses were performed using IBM SPSS Statistics version 26.0 (IBM Corporation, Armonk, NY, USA).

3. Results

The mean values of COM positions for the groups in the AP (mean difference = 4.91 mm, 95% CI = -6.85 to 16.67 mm, $P = 0.395$) and ML planes (mean difference = -0.95 mm, 95% CI = -7.02 to 5.12 mm, $P = 0.748$) did not change significantly. In addition, no significant difference was found between the mean values of lower limb joint angles of groups in the AP (hip: mean difference = -0.58°, 95% CI = -6.76 to 5.60°, $P = 0.847$; knee: mean difference = 2.71°, 95% CI = -2.97 to 8.38°, $P = 0.332$; ankle: mean difference = 0.68°, 95% CI = -2.92 to 4.28°, $P = 0.698$) and ML planes (hip: mean difference = -0.55°, 95% CI = -2.66 to 1.57°, $P = 0.597$; ankle: mean difference = -2.43°, 95% CI = -8.21 to 3.35°, $P = 0.391$).

For the SD of AP and ML COM positions, there was no significant difference between the groups (AP: mean difference = 0.06 mm, 95% CI = -0.91 to 1.02 mm, $P = 0.903$; ML: mean difference = 0.51 mm, 95% CI = -0.14 to 1.16 mm, $P = 0.115$) (Fig. 1a). The SD of AP COM velocity was significantly larger in the knee OA group than that of the control group (mean difference = 0.79 mm/s, 95% CI = 0.24 to 1.35 mm/s, $P = 0.007$). Furthermore, there was no significant difference in the ML COM velocity of groups (mean difference = 0.18

176 mm/s, 95% CI = -0.27 to 0.63 mm/s, $P = 0.408$) (Fig. 1b).

177 For the SD of lower limb joint angles in the AP plane, there was no significant
178 difference between the groups (hip: mean difference = 0.06°, 95% CI = -0.09 to 0.20°, $P =$
179 0.433; knee: mean difference = 0.02°, 95% CI = -0.08 to 0.11°, $P = 0.705$; ankle: mean
180 difference = 0.03°, 95% CI = -0.03 to 0.09°, $P = 0.296$) (Fig. 2a). Regarding the ML plane,
181 the SD of hip angle was significantly larger in the knee OA group than that of the control
182 group (mean difference = 0.07°, 95% CI = 0.02 to 0.12°, $P = 0.007$). Moreover, the SD of
183 ankle angle was not significantly different between the groups (mean difference = 0.01°, 95%
184 CI = -0.08 to 0.09°, $P = 0.853$) (Fig. 2b). As for the lower limb joint angular velocities, the
185 SD of AP hip and knee angular velocities were significantly larger in the knee OA group than
186 those of the control group (hip: mean difference = 0.22 °/s, 95% CI = 0.08 to 0.35 °/s, $P =$
187 0.003; knee: mean difference = 0.17 °/s, 95% CI = 0.04 to 0.30 °/s, $P = 0.010$). In addition,
188 the SD of AP ankle angular velocity was not significantly different between groups (mean
189 difference = 0.04 °/s, 95% CI = -0.06 to 0.15 °/s, $P = 0.372$) (Fig. 3a). The SD of ML hip
190 angular velocity was significantly larger in the knee OA group than that of the control group
191 (mean difference = 0.16 °/s, 95% CI = 0.02 to 0.31 °/s, $P = 0.030$). Furthermore, the SD of
192 ML ankle angular velocity was not significantly different between groups (mean difference =
193 -0.14 °/s, 95% CI = -0.60 to 0.31 °/s, $P = 0.523$) (Fig. 3b).

194 Pearson's correlation analysis showed that the SD of AP ankle angle was positively
195 correlated with the SD of AP COM position in both groups (OA: $r = 0.662$, $P = 0.037$;
196 control: $r = 0.777$, $P = 0.002$) (Table 2). In the knee OA group, the SD of ML hip angle was
197 positively correlated with the SD of ML COM position ($r = 0.925$, $P < 0.001$), whereas in the
198 control group, the SD of ML ankle angle was positively correlated with the SD of ML COM
199 position ($r = 0.726$, $P = 0.005$). The SD of AP hip angular velocity was positively correlated
200 with the SD of AP COM velocity in the knee OA group ($r = 0.644$, $P = 0.044$). In addition,

the SDs of AP hip and knee angular velocities were positively correlated with the SD of AP COM velocity in the control group (hip: $r = 0.562$, $P = 0.046$; knee: $r = 0.673$, $P = 0.012$) (Table 3). The SD of ML hip angular velocity was positively correlated with the SD of ML COM velocity in both groups (OA: $r = 0.820$, $P = 0.004$; control: $r = 0.846$, $P < 0.001$).

In addition, there was no significant correlation between the SDs of COM and lower limb joint motions and the VAS score for knee pain ($P = 0.092 – 0.966$) (Table 4).

4. Discussion

The current study revealed that the characteristics of the postural control strategy during quiet standing in individuals with knee OA differed from those of age-matched healthy adults. The knee OA group exhibited significantly larger SDs of AP COM velocity, ML hip angle, AP hip angular velocity, AP knee angular velocity, and ML hip angular velocity than the control group. The AP ankle angle was significantly correlated with the AP COM position in both groups, while the ML hip angle and ML ankle angle were significantly correlated with the ML COM position in the knee OA and control groups, respectively. In the knee OA group, the AP hip angular velocity was significantly correlated with the AP COM velocity, while in the control group, the AP hip and knee angular velocities were significantly correlated. The ML hip angular velocity was significantly correlated with the ML COM velocity in both groups.

Previous studies suggested that the amplitude of postural sway in the AP and ML planes is generally controlled by ankle and hip joint motions [19,20]. In this study, the amplitude of AP COM position was controlled by the ankle strategy in both groups, and there was no significant difference in the amplitude of AP hip, knee, and ankle angles between the groups. In other words, the postural control strategy for controlling the AP COM position may be identical in both groups. The knee OA group used the hip strategy to control the amplitude

of ML COM position, whereas the control group used the ankle strategy. Furthermore, the amplitude of ML hip angle of the knee OA group was larger than that of the control group; however, there was no significant difference in the amplitude of ML ankle angles of the groups. Compared with healthy adults, patients with knee OA have an impaired sense of motion of the ankle joint in the ML plane; however not in the hip joint [25]. Those with knee OA may be unable to adequately control the ML COM position with the ankle strategy. Therefore, they may prefer to use the hip strategy to control the ML COM position. Furthermore, the hip strategy has less COM motion than the ankle strategy even with the same angle change because the center of rotation of hip joint is closer to the COM than that of the ankle joint [32]. Therefore, the knee OA group may have required greater ML hip joint motion than the control group.

It is believed that the hip strategy is preferable to the ankle strategy for fast COM motion in postural control [33]. The hip strategy, which results in predominant hip joint motion, includes thigh muscle activity that causes knee joint motion, although the muscles around the ankle joint are extremely unresponsive [34]. As for the control of AP COM velocity, the knee OA group used a postural control strategy using the hip joint alone, while the control group used a mixed postural control strategy using the hip and knee joints. In other words, the knee OA group depended only on the contribution of hip joint to the AP COM velocity, while the control group depended on the contribution of hip and knee joints. Therefore, the knee OA group exhibited increased body sway velocity and hip angular velocity in the AP plane. In addition, the knee joint possibly cannot be used well to control body sway velocity in the AP plane because the AP knee angular velocity in the knee OA group was not correlated with the body sway velocity in the AP plane and was larger than that of the control group. This may be affected by knee joint dysfunctions, such as quadriceps weakness [4], limited knee range of motion [5], and impaired proprioception of the knee [6]

in patients with knee OA. Our study showed that it might be necessary to maintain hip joint function and improve knee joint function for balance training in patients with knee OA. As for the control of ML COM velocity, both groups used a postural control strategy using the hip joint. Furthermore, the SD of hip angular velocity in the knee OA group was larger than that of the control group, while there was no significant difference in the SD of ML COM velocity for both groups. Postural sway velocity, such as the COM velocity, includes the direction and position changes at the next moment. Therefore, this information is considered significant for maintaining equilibrium [30,35]. The knee OA group may maintain postural stability because of an increased sensory information resulting from an increased ML hip angular velocity to compensate for an impaired sense of motion of the ankle joint in the ML plane.

Previous studies presented that knee pain was associated with postural sway in patients with mild-to-severe knee OA whose VAS score for knee pain was greater than 30 mm [15,18]. However, in this study, knee pain did not correlate with the SDs of COM and lower limb joint motions in patients with knee OA. This might be caused by mild knee pain and mild-to-moderate knee OA in this study.

This study had several limitations. First, the participants in this study had mild-to-moderate knee OA, and the average knee pain was less severe. The average knee pain in this study may have been mild because of mild-to-moderate knee OA [9]. Therefore, the participants in this study might not represent the overall characteristics of patients with knee OA. Second, the task examined in this study was quiet standing, and results from dynamic tasks, such as external perturbations, may differ from those in this study. Therefore, dynamic tasks need to be investigated in the future. Third, we used three-dimensional motion analysis to calculate the COM and lower limb joint motions. Measurement errors due to soft tissue artifacts in three-dimensional motion analysis may have affected the results of quiet standing performed in this study [36]. Finally, the number of participants in this study was limited.

Further studies with a large number of participants are needed to take into account the severity of knee OA and knee pain.

5. Conclusions

We investigated the alterations in the postural control strategies during quiet standing in individuals with knee OA. Both the knee OA and control groups utilized the ankle joint to control the AP COM position. For the control of ML COM position, the knee OA group used the hip joint, whereas the control group used the ankle joint. The knee OA group successfully controlled the AP and ML COM positions because there was no significant group difference in the amplitude of COM position. To control the AP COM velocity, the knee OA group relied on the contribution of hip joint solely, whereas the control group relied on the contribution of hip and knee joints. Both the knee OA and control groups used the hip joint to control the ML COM velocity. The knee OA group exhibited postural control impairment in the amplitude of COM velocity in the AP plane, and not in the ML plane though. Our findings suggested that the knee OA group could not successfully control the AP COM velocity using the knee joint.

Acknowledgements

The authors did not receive any grants for this study.

References

[1] Yoshimura N, Muraki S, Oka H, Mabuchi A, En-Yo Y, Yoshida M, et al. Prevalence of knee osteoarthritis, lumbar spondylosis, and osteoporosis in Japanese men and women: the research on osteoarthritis/osteoporosis against disability study. J Bone Miner Metab. 2009; 27(5): 620–8.

[2] Muraki S, Oka H, Akune T, Mabuchi A, En-yo Y, Yoshida M, et al. Prevalence of radiographic knee osteoarthritis and its association with knee pain in the elderly of Japanese population-based cohorts: The ROAD study. Osteoarthr Cartil. 2009; 17(9): 1137–43.

[3] van Dijk GM, Dekker J, Veenhof C, van den Ende CHM, Carpa Study Group. Course of functional status and pain in osteoarthritis of the hip or knee: A systematic review of the literature. Arthritis Rheum. 2006; 55(5): 779–85.

[4] Slemenda C, Brandt KD, Heilman DK, Mazzuca S, Braunstein EM, Katz BP, et al. Quadriceps weakness and osteoarthritis of the knee. Ann Intern Med. 1997; 127(2): 97–104.

[5] Hilfiker R, Jüni P, Nüesch E, Dieppe PA, Reichenbach S. Association of radiographic osteoarthritis, pain on passive movement and knee range of motion: A cross-sectional study. Man Ther. 2015; 20(2): 361–5.

[6] Knoop J, Steultjens MPM, van der Leeden M, van der Esch M, Thorstensson CA, Roorda LD, et al. Proprioception in knee osteoarthritis: a narrative review. Osteoarthr Cartil. 2011; 19(4): 381–8.

[7] Eckstein F, Le Graverand MPH, Charles HC, Hunter DJ, Kraus VB, Sunyer T, et al. Clinical, radiographic, molecular and MRI-based predictors of cartilage loss in knee osteoarthritis. Ann Rheum Dis. 2011; 70(7): 1223–30.

[8] Guccione AA, Felson DT, Anderson JJ, Anthony JM, Zhang Y, Wilson PW, et al. The effects of specific medical conditions on the functional limitations of elders in the Framingham Study. Am J Public Health. 1994; 84(3): 351–8.

[9] Muraki S, Akune T, Oka H, En-yo Y, Yoshida M, Saika A, et al. Association of radiographic and symptomatic knee osteoarthritis with health-related quality of life in a population-based cohort study in Japan: The ROAD study. Osteoarthr Cartil. 2010; 18(9):

1227–34.

[10] Smith TO, Higson E, Pearson M, Mansfield M. Is there an increased risk of falls and fractures in people with early diagnosed hip and knee osteoarthritis? Data from the Osteoarthritis Initiative. Int J Rheum Dis. 2018; 21(6): 1193–201.

[11] Bergink AP, Van Der Klift M, Hofman A, Verhaar J a N, Van Leeuwen JPTM, Uitterlinden AG, et al. Osteoarthritis of the knee is associated with vertebral and nonvertebral fractures in the elderly: The Rotterdam Study. Arthritis Rheum. 2003; 49(5): 648–57.

[12] Arden NK, Crozier S, Smith H, Anderson F, Edwards C, Raphael H, et al. Knee pain, knee osteoarthritis, and the risk of fracture. Arthritis Rheum. 2006; 55(4): 610–5.

[13] Manlapaz DG, Sole G, Jayakaran P, Chapple CM. Risk factors for falls in adults with knee osteoarthritis: A systematic review. PM&R. 2019; 11(7): 745–57.

[14] Lawson T, Morrison A, Blaxland S, Wenman M, Schmidt CG, Hunt MA. Laboratory-based measurement of standing balance in individuals with knee osteoarthritis: A systematic review. Clin Biomech. 2015; 30(4): 330–42.

[15] Hinman RS, Bennell KL, Metcalf BR, Crossley KM. Balance impairments in individuals with symptomatic knee osteoarthritis: A comparison with matched controls using clinical tests. Rheumatology. 2002; 41(12): 1388–94.

[16] Hassan BS, Mockett S, Doherty M. Static postural sway, proprioception, and maximal voluntary quadriceps contraction in patients with knee osteoarthritis and normal control subjects. Ann Rheum Dis. 2001; 60(6): 612–8.

[17] Birmingham TB, Kramer JF, Kirkley A, Inglis JT, Spaulding SJ, Vandervoort AA. Association among neuromuscular and anatomic measures for patients with knee osteoarthritis. Arch Phys Med Rehabil. 2001; 82(8): 1115–8.

[18] Hunt MA, McManus FJ, Hinman RS, Bennell KL. Predictors of single-leg standing

balance in individuals with medial knee osteoarthritis. Arthritis Care Res. 2010; 62(4): 496–500.

[19] Winter DA, Prince F, Frank JS, Powell C, Zabjek KF. Unified theory regarding A/P and M/L balance in quiet stance. J Neurophysiol. 1996; 75(6): 2334–43.

[20] Madigan ML, Davidson BS, Nussbaum MA. Postural sway and joint kinematics during quiet standing are affected by lumbar extensor fatigue. Hum Mov Sci. 2006; 25(6): 788–99.

[21] Yamamoto A, Sasagawa S, Oba N, Nakazawa K. Behavioral effect of knee joint motion on body's center of mass during human quiet standing. Gait Posture. 2015; 41(1): 291–4.

[22] Hinman RS, Hunt MA, Creaby MW, Wrigley T V., McManus FJ, Bennell KL. Hip muscle weakness in individuals with medial knee osteoarthritis. Arthritis Care Res. 2010; 62(8): 1190–3.

[23] Vårbakken K, Lorås H, Nilsson KG, Engdal M, Stensdotter AK. Relative difference in muscle strength between patients with knee osteoarthritis and healthy controls when tested bilaterally and joint-inclusive: An exploratory cross-sectional study. BMC Musculoskelet Disord. 2019; 20(1): 593.

[24] Levinger P, Menz HB, Fotoohabadi MR, Feller JA, Bartlett JR, Bergman NR. Foot posture in people with medial compartment knee osteoarthritis. J Foot Ankle Res. 2010; 3: 29.

[25] Mani E, Tüzün EH, Angın E, Eker L. Lower extremity proprioceptive sensation in patients with early stage knee osteoarthritis: A comparative study. Knee. 2020; 27(2): 356–62.

[26] Kellgren JH, Lawrence JS. Radiological assessment of osteo-arthrosis. Ann Rheum Dis. 1957; 16(4): 494–502.

[27] Kadaba MP, Ramakrishnan HK, Wootten ME. Measurement of lower extremity

kinematics during level walking. J Orthop Res. 1990; 8(3): 383–92.

[28] Boyas S, Medd ER, Beaulieu S, Boileau A, Lajoie Y, Bilodeau M. Older and young adults adopt different postural strategies during quiet bipedal stance after ankle plantarflexor fatigue. Neurosci Lett. 2019; 701: 208–12.

[29] Gard SA, Miff SC, Kuo AD. Comparison of kinematic and kinetic methods for computing the vertical motion of the body center of mass during walking. Hum Mov Sci. 2004; 22(6): 597–610.

[30] Pollock CL, Hunt MA, Vieira TM, Gallina A, Ivanova TD, Garland SJ. Challenging standing balance reduces the asymmetry of motor control of postural sway poststroke. Motor Control. 2019; 23(3): 327–43.

[31] Boyas S, Hajj M, Bilodeau M. Influence of ankle plantarflexor fatigue on postural sway, lower limb articular angles, and postural strategies during unipedal quiet standing. Gait Posture. 2013; 37(4): 547–51.

[32] Ogaya S, Okita Y, Fuchioka S. Muscle contributions to center of mass excursion in ankle and hip strategies during forward body tilting. J Biomech. 2016; 49(14): 3381–6.

[33] Horak FB, Henry SM, Shumway-Cook A. Postural perturbations: New insights for treatment of balance disorders. Phys Ther. 1997; 77(5): 517–33.

[34] Horak FB, Nashner LM. Central programming of postural movements: Adaptation to altered support-surface configurations. J Neurophysiol. 1986; 55(6): 1369–81.

[35] Masani K, Popovic MR, Nakazawa K, Kouzaki M, Nozaki D. Importance of body sway velocity information in controlling ankle extensor activities during quiet stance. J Neurophysiol. 2003; 90(6): 3774–82.

[36] Leardini A, Chiari L, Della Croce U, Cappozzo A. Human movement analysis using stereophotogrammetry. Part 3. Soft tissue artifact assessment and compensation. Gait Posture. 2005; 21(2): 212–25.

Tables

Table 1

Demographic data of the knee OA and control groups

	Knee OA	Control	*P*
Age (years)	67.8 (8.9)	63.5 (8.6)	0.258
Height (cm)	153.8 (7.5)	156.1 (8.2)	0.488
Weight (kg)	56.8 (15.2)	57.7 (10.3)	0.866
BMI (kg/m^2)	23.7 (4.1)	23.6 (3.1)	0.924
Kellgren–Lawrence grade (n)			
Grade 2	4		
Grade 3	6		
VAS score for knee pain (mm)[a]	23.8 (17.8)		

Data are reported as mean values (standard deviation).

OA: osteoarthritis, BMI: body mass index, VAS: visual analogue scale.

[a] VAS score for knee pain ranges from 0 (no pain) to 100 mm (severe pain).

Table 2

Pearson's correlation coefficients between each lower limb joint angle and COM position

	Knee OA		Control	
	r	*p*	*r*	*p*
SD of AP COM position				
SD of AP hip angle	0.129	0.722	0.207	0.497
SD of AP knee angle	0.311	0.382	0.418	0.155
SD of AP ankle angle	**0.662**	**0.037***	**0.777**	**0.002***
SD of ML COM position				
SD of ML hip angle	**0.925**	**< 0.001***	0.366	0.219
SD of ML ankle angle	0.239	0.507	**0.726**	**0.005***

COM: center of mass, OA: osteoarthritis, SD: standard deviation, AP: anterior-posterior, ML: medial-lateral.

* $P < 0.05$.

Table 3

Pearson's correlation coefficients between each lower limb joint angular velocity and COM velocity

	Knee OA		Control	
	r	*P*	*r*	*P*
SD of AP COM velocity				
SD of AP hip angular velocity	**0.644**	**0.044***	**0.562**	**0.046***
SD of AP knee angular velocity	0.539	0.108	**0.673**	**0.012***
SD of AP ankle angular velocity	0.424	0.222	0.427	0.146
SD of ML COM velocity				
SD of ML hip angular velocity	**0.820**	**0.004***	**0.846**	**< 0.001***
SD of ML ankle angular velocity	-0.023	0.949	0.212	0.488

COM: center of mass, OA: osteoarthritis, SD: standard deviation, AP: anterior-posterior, ML: medial-lateral.

* $P < 0.05$.

Table 4

Pearson's correlation coefficients between the COM and lower limb joint motions and knee pain in patients with knee OA

	Knee OA	
	r	*P*
VAS score for knee pain		
SD of AP COM position	-0.352	0.319
SD of ML COM position	-0.196	0.588
SD of AP COM velocity	-0.182	0.614
SD of ML COM velocity	-0.105	0.772
SD of AP hip angle	0.467	0.173
SD of AP knee angle	0.449	0.193
SD of AP ankle angle	-0.322	0.364
SD of ML hip angle	-0.015	0.966
SD of ML ankle angle	0.561	0.092
SD of AP hip angular velocity	-0.196	0.587
SD of AP knee angular velocity	-0.044	0.904
SD of AP ankle angular velocity	0.145	0.689
SD of ML hip angular velocity	-0.134	0.711
SD of ML ankle angular velocity	0.495	0.146

COM: center of mass, OA: osteoarthritis, VAS: visual analogue scale, SD: standard deviation, AP: anterior-posterior, ML: medial-lateral.

Figure captions

Fig. 1. Group-averaged SD of COM position (a) and velocity (b) in the AP and ML planes. The white bar indicates the knee OA group and the black bar indicates the control group. * $P < 0.05$.

Fig. 2. Group-averaged SD of hip, knee, and ankle angles in the AP (a) and ML (b) planes. The White bar indicates the knee OA group and the black bar indicates the control group. * $P < 0.05$.

Fig. 3. Group-averaged SD of hip, knee, and ankle angular velocities in the AP (a) and ML (b) planes. The white bar indicates the knee OA group and the black bar indicates the control group. * $P < 0.05$.

Figures

Figure 1

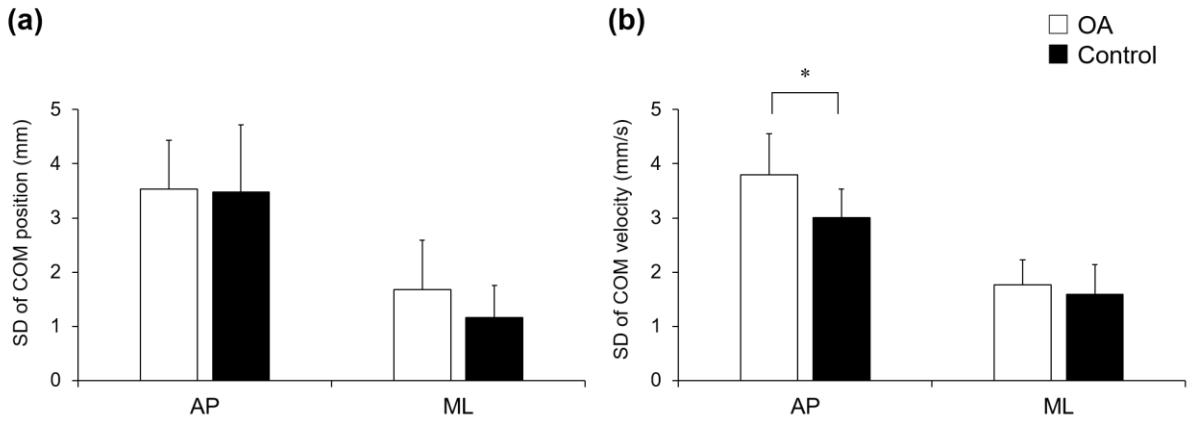
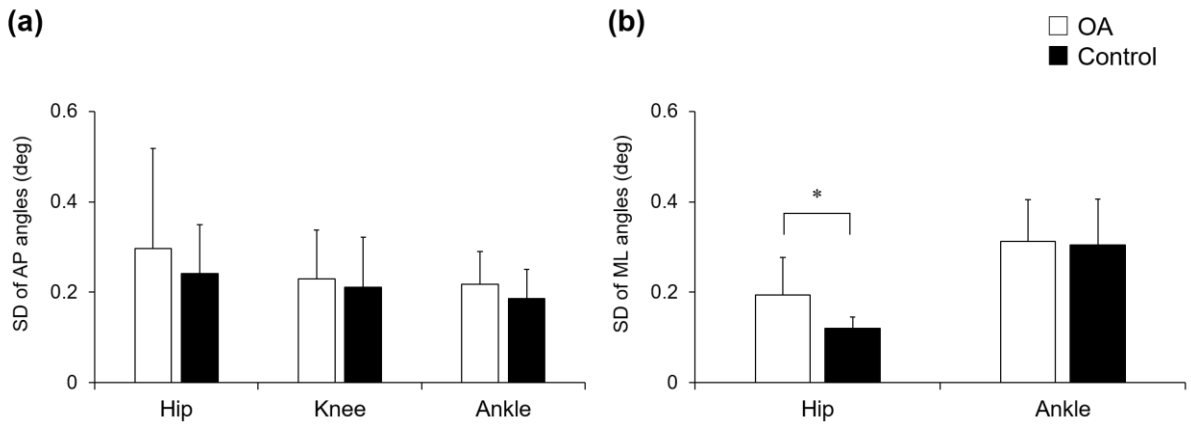
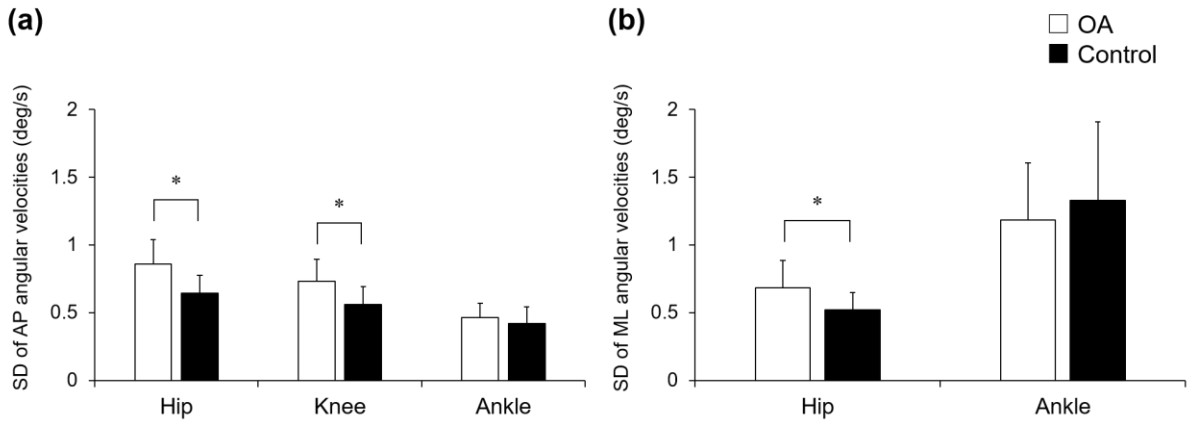


Figure 2


(a)
(b)
OA
Control
SD of AP angles (deg)
SD of ML angles (deg)
0.6
0.4
0.2
0
*
Hip
Knee
Ankle
Hip
Ankle

522 **Figure 3**



523