J. Phys. Ther. Sci.
24: 681–685, 2012

The Role of the Supraspinal Center during Soleus Stretching Reflexes with Simultaneous Vibration

Masahiro Sakita, Msc, PT[1)], Shinichiro Murakami, PhD, PT[2)], Yoshiki Ishii, PhD, PT[2)], Takafumi Saito, Msc, PT[3)], Shuzo Kumagai, PhD[4)]

[1)] *Department of Physical Therapy, Faculty of Health Sciences, Kyoto Tachibana University: 34 Oyakeyamada, Yamashina Ward, Kyoto City, Kyoto 607-8175, Japan. TEL: +81 75-574-4315, FAX: +81 75-574-4122, E-mail: sakita@tachibana-u.ac.jp*
[2)] *Department of Physical Therapy, Faculty of Health Care Sciences, Himeji Dokkyo University*
[3)] *Department of Physical Therapy, Aso Rehabilitation College*
[4)] *Institute of Health Science, Kyushu University*

Abstract. [Purpose] We examined whether monoaminergic brain stem centers contribute to reflexive soleus (Sol) activity when vibration is applied to ankle joints on a moving platform. [Methods] Ten male subjects (23–35 years) stood with their eyes closed on a movable platform. Vibrators (92 Hz) were applied to the malleolus and Achilles' tendon. Sol electromyographic (EMG) responses of short- (SLR) and medium-latency reflexes (MLR) during platform movement were collected under the control, Sol vibration (SV), and malleolus vibration (MV) conditions. The SLR, MLR areas and their latencies were measured. [Results] The Sol SLR and MLR onsets were significantly delayed under the SV and MV conditions compared to the control condition. The intercept of the regression line under the MV conditions was significantly greater than under the SV condition. [Conclusion] Delays of SLR and MLR onset under the SV and MV conditions might correspond to the length of time required for temporal summation of α-motoneurons due to inhibition of afferent fibers. A rise in the intercept of the regression line under the MV condition means an increase of MLR area. That is, the monoaminergic brain stem centers compensated for stimulation of the group II interneuron via ankle joint afferents acting against the inhibition of the stimulation of Sol α-motoneurons.
Key words: Ankle joint, Group II fibers, Medium latency reflex

(This article was submitted Feb. 1, 2012, and was accepted Mar. 5, 2012)

INTRODUCTION

Signals mediated by Ia and group II afferent fibers from peripheral leg muscles contribute to two component reflexive homonymous muscle responses during static and dynamic standing. It has been shown that a sudden toe-up rotation on a platform being used for standing produces two peak electromyogram (EMG) responses mediated by Ia and group II afferents during stretching of the soleus (Sol) and flexor digitorum brevis (FDB) muscle [1–3)]. The first component is a short-latency reflex (SLR) that takes place at about the latency of the monosynaptic reflex arc. It originates in the spindle primaries and is mediated by group Ia large afferent fibers. The second component is a medium-latency reflex (MLR) that is transmitted to the spinal cord from spindle secondary terminations by group II afferent fibers[3)]. Several studies have reported abnormal SLR and MLR activities of Sol and FDB in the legs of subjects who have peripheral neuropathy cause standing postural instability [4–6)]. These studies concluded that delayed and decreased SLR and MLR activities are the result of dysfunction of Ia and group II afferent fibers in mediating signals from muscle spindles and cause large postural sway. It is known that afferent signals from sole cutaneous receptors, other than Ia and group II afferent signals from muscle spindles in peripheral leg muscles, contribute to peripheral muscle activity in upright stance [7, 8)]. According to the major results of recent studies of reflexive Sol and FDB activities during static standing, Sol and FDB activities are affected by not only afferent signals from homonymous muscle spindles, but also by sole cutaneous afferent signals. However, little is known about the influence of afferent signals from ankle joints on reflexive peripheral muscle activity in the upright posture. One study examined whether afferent signals decreased by local anesthesia of the ankle joint affected the stability of upright posture. Errors of passive position sense were significantly increased in comparison to active position sense errors, and the difference in stability of one-leg standing between the pre- and post-anaesthetic block was not significant[9)]. Thus, the afferent signals from ankle joint may thus play a dominant role on passive position sense, to which afferent signals from the muscle spindle do not contribute, even though muscle spindle afferents are predominantly involved in the stability of static standing. No study has yet examined the changes in SLR and MLR activities of peripheral muscles in the leg while standing on one-leg with the ankle joint under

anesthesia. If the afferent signals from the ankle joint affect the detection of passive movement, SLR and MLR activities of peripheral muscles would be influenced when the feet are perturbed during static standing. Accordingly, we have to consider whether the SLR and MLR activities of peripheral muscles are affected by decreased afferent signals from the ankle joint under unstable conditions.

We also have to consider that the supraspinal center affects stimulation of α-motoneurons of peripheral muscles when a person stands. The amplitude of MLR in leg muscles is sensitive to whether or not the subject can predict the postural task and whether the posture of the standing subject is stabilized or not[10, 11]. When a standing subject's balance is perturbed and the subject unable to predict the postural task, MLR activity of the leg muscles significantly increases compared to stable standing with predictable conditions for the postural task[12]. It has been concluded that monoaminergic brain stem centers selectively modulate the stimulation of the interneuronal pathways through group II afferents from homonymous spindle secondaries. However, it is not clear whether MLR activity of the peripheral muscles are affected by modulation from monoaminergic brain stem centers, if presynaptic or disynaptic inhibitions occur in afferent fibers connecting with the ankle joints during ankle vibration.

To cause hypoesthesia, mechanical vibrations were applied to Sol and malleolus of the subjects[13–15]. Vibration applied to peripheral leg muscles causes presynaptic inhibition of Ia and group II afferent fibers, and then stimulates the α-motoneurons of homonymous muscles via a decrease in interneuron numbers[16, 17]. In contrast, vibration applied to the malleolus raises the activation thresholds of the ankle mechanoreceptors making it harder to stimulate the receptors because of the receptors' raised threshold[18]. Information on the level of the vibration frequency used in several studies was helpful for our research. The vibration frequency which effectively raises the thresholds of muscle spindles and mechanoreceptors is approximately 100 Hz[14, 19, 20].

Therefore, we examined whether vibration near 100 Hz of the Sol and ankle joint affects Sol SLR and MLR activities during platform movements. We also examined whether monoaminergic brain stem centers modulate Sol MLR activity when vibration is applied to the Sol and ankle joint.

SUBJECTS AND METHODS

Ten healthy male subjects (aged 23–35 years, with a mean age of 27.9 years of age) participated in the experiments. The subjects gave their informed consent and the study conformed to the Declaration of Helsinki. The current research began after approval was obtained from the ethical committee of the Health Science Center of Kyushu University.

The subjects were asked to stand with their eyes closed, arms by their side with both feet on a movable platform (Equi-test version 8.1, NeuroCom Inc. USA). The center of mass (COM) of the subject was observed on the anterior-posterior and medial-lateral axes on a computer screen. The dot point, which indicates the COM of the subject, was observed before the platform perturbation. The COM was placed at the intersection point of the coordinate axes by the tester. The feet were grounded and the subject's height on the platform was taken into account. The movement of the platform was a backward displacement, which induces stretch and consequent reflex responses in the Sol muscle. Table 1 shows the amplitude of the movement of the platform. One series of measurements included 18–21 trials and the time interval between each trial within a series varied randomly from 1.5 to 2.5 seconds.

Table 1. Platform displacement amplitude

Intensity	Duration time (ms)	Perturbation (cm)
large	400	4.6–6.0

The distance of platform movement was related to the height of the subject

The vibrators contained a DC motor with an eccentric the embedded in rectangular plastic case, 3 cm wide, 6 cm long and approximately 2 cm high (MCL-1701, Alinco Inc. Japan). Vibrators (92 Hz) were bilaterally fixed to the Achilles' tendons (Sol vibration condition, SV) and to the lateral and medial malleolus (malleolus vibration condition, MV) by elastic bands. The vibrators were turned off in the control condition (without vibration). In the SV and MV conditions, vibration was applied for one minute before the beginning of platform movement and was continued for 18 to 21 trials (about six minutes respectively). The vibrator produced a peak-to-peak force of about 4 N (4.13 ± 0.12 N), as measured by the strain gauge (EM-555, Noraxon Inc. USA) that was placed between the skin and the vibrator[14].

Three trials on the platform comprised a single unit. The subject executed a single unit 6 or 7 times (18–21 trials) without rest. In each unit, the interval between each trial was randomly set to vary from 1.5 to 2.5 seconds. Each condition was examined randomly. Noda et al.[13] reported that 5 minutes of conditioning vibration applied to the malleolus of normal subjects increased the vibratory perception threshold at 10, 60 and 120 seconds, but not at 5 minutes. Therefore, a rest period of 5 minutes was provided between the measurement series during which the subjects adopted a comfortable sitting position. Sol muscle EMG responses to backward platform movement under each of three conditions were collected for each subject.

EMGs were recorded using surface electrodes. The distance between the surface electrodes was approximately 2 cm. The electrodes were positioned on the skin 3 cm below the bottom of the medial head of the gastrocnemii to record the EMG activity of Sol on the right leg. The EMG signal was amplified (10,000×) and band-pass filtered from 10 to 500 Hz. The analog signal was digitally converted at a sampling rate of 1 kHz, and the onset signal of platform movement was synchronously recorded on a personal computer. All of the identified EMG wave shapes were rectified. The acquisition period was 350 ms, with the platform movement starting at 100 ms from the onset.

All Sol responses of EMG were smoothed (time constant 10 ms), and the onsets of SLR and MLR in Sol were defined as when the EMG signal rose above 2 SDs of the mean value of the background EMG activity before the beginning of

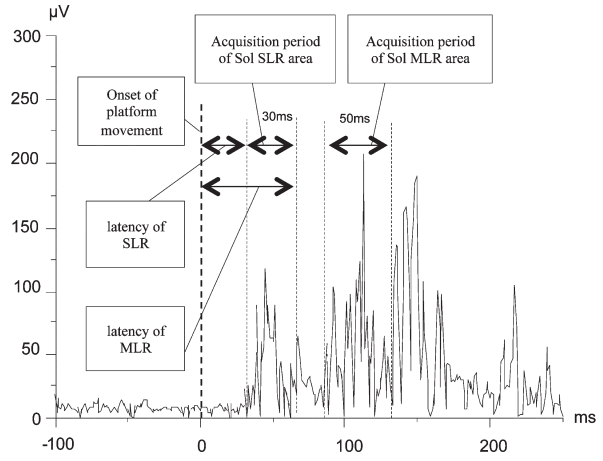

Fig. 1. Samples of measurement items of rectified EMGs of Sol This panel shows a representative sample of the rectified Sol EMG during the backward platform movement. The measurement items were the latencies of Sol SLR and MLR onsets, the SLR area (%MIVC) and MLR area (%MIVC).

platform movement. The responses in the stretched Sol were classified as SLR or MLR when their onset latencies were shorter or longer than 60 ms [3, 14], respectively. The SLR and MLR areas were calculated using the average of the rectified and filtered (time constant 1 ms) EMG traces. In our pilot study, the acquisition periods of rectified and integrated SLR and MLR EMG areas in Sol were respectively determined at 30 ms and 50 ms by smoothed EMG (unpublished). The areas were measured in a time window of 30 ms for Sol SLR and 50 ms for Sol MLR from the onset of the responses[14] (Fig. 1). Time windows of the same acquisition periods were then used to measure the areas of the responses in each single trial under each of the three conditions. Sol SLR and MLR EMG responses of each subject were normalized with respect to the EMG activity of an equivalent duration recorded during maximal isometric voluntary contraction (MIVC) of the Sol muscles to compare the findings across all subjects. The epoch of acquisition of the EMG MIVC signal began 2 s after MIVC onset and lasted for 500 ms.

A one-way (3 group × 2 responses) analysis of variance (ANOVA) was performed for the three conditions to compare the latencies of Sol SLR and MLR onsets. Analysis of covariance (ANCOVA) was used to assess the effects of Sol MLR EMG area with respect to SLR EMG area and each of the three conditions. The three conditions were covariance (independent variables) and Sol MLR EMG area was a dependent variable. The Bonferroni/Dunn post hoc test was employed when the results of ANOVA were significant. A linear regression analysis was used to evaluate the trials under each of the three conditions to characterize the relationship of the Sol SLR and MLR areas. The regression coefficient and intercepts of the three conditions were evaluated with the Bonferroni/Dunn test (the SLR area was a covariate). P values of less than 0.05 were considered statistically significant.

RESULTS

Table 2 shows the overall average of onset latencies of Sol SLR and MLR in response to the platform backward movement under the three conditions. ANOVA revealed the vibration had a significant effect on the latencies of Sol SLR ($F=93.879$; d.f.$=2, 561$; $p<0.0001$) and MLR ($F=36.957$; d.f.$=2, 561$; $p<0.0001$). Vibration caused a significant delay in Sol SLR of approximately 2.5 ms under the SV condition and 2 ms under the MV condition compared to the control value (post hoc test, $p<0.01$ and $p<0.01$, respectively). Vibration induced a significant delay in Sol MLR of 6 ms under the SV condition and of approximately 3 ms under the MV condition in comparison to the control value (post hoc test, $p<0.01$ and $p<0.01$, respectively).

From the analyses of the effects of Sol MLR EMG area with respect to SLR EMG area among the three conditions (ANCOVA), Sol MLR EMG area was affected by Sol SLR EMG area ($F=300.323$; d.f.$=1, 561$; $p<0.0001$) and conditions ($F=3.154$; d.f.$=2, 561$; $p<0.05$). There was not an interaction between covariate of the conditions and independent variable of Sol SLR areas (Table 3).

A slightly positive relationship was found between the Sol MLR and SLR areas under the control condition ($y=1.896+0.675x$; $p<0.0001$). A more positive relationship was found between the two areas under SV ($y=1.605+0.809x$; $p<0.0001$). The regression line under the MV condition was moderately positive between the two areas, but the coefficient of regression was slightly lower than that under the control condition ($y=2.254+0.641x$; $p<0.0001$). The coefficients of regressions were not different between the SV and MV conditions. The intercept under the MV condition rose significantly more than under the SV condition (Bonferroni/Dunn test; $p<0.01$). In Table 3, the intercept of the regression line was lower under the SV condition than under the MV condition.

DISCUSSION

Both Sol SLR and MLR latencies under the SV condition increased. There is accumulating evidence that Ia and group II afferent fibers from Sol muscle spindles show presynaptic inhibition when a vibration frequency of nearly 100 Hz is applied[13, 14, 21–23]. This evidence will help us to have a thorough understanding of the delay mechanism of Sol SLR and MLR onsets. Delays of Sol SLR and MLR onset might correspond to the delay of temporal summation of α-motoneurons due to presynaptic inhibition of Ia and group II afferent fibers.

The onset latency of Sol SLR and MLR was also extended under the MV condition. It is documented that afferent fibers from the ankle joints have disynaptic connections to α-motoneurons of the quadriceps femoris muscles[24]. From these delays of Sol SLR and MLR latencies under the MV, the afferents from the ankle joints are also assumed to constitute the pathways to α-motoneurons of Sol. The afferent fibers from several mechanoreceptors of the ankle joints are group I and II afferent fibers[25]. Thus, excessive afferent signals from the ankle joint under vibration seem to

Table 2. One-way analysis of variance of differences among the control, Sol vibration and malleolus vibration conditions of the onsets of Sol SLR and MLR

	Condition		
	Control	Sol vibration (SV)	Malleolus vibration (MV)
Onset of Sol SLR (ms)	38.1 ± 0.2	44.6 ± 0.4§§	41.2 ± 0.4** ††
Onset of Sol MLR (ms)	68.5 ± 0.6	74.6 ± 0.4§§	71.6 ± 0.5** ††

§§ indicates significant difference ($p<0.01$) between control and SV conditions in post hoc test (The Bonferroni/Dunn test). ** indicates significant difference ($p<0.01$) between control and MV conditions in post hoc test (The Bonferroni/Dunn test). †† indicates significant difference ($p<0.01$) between SV and MV conditions in post hoc test (The Bonferroni/Dunn test). SLR: short latency reflex, MLR: medium latency reflex. Each value is mean ± S.E.

Table 3. Coefficient and intercept values of the linear regression models of $\log_e$ Sol MLR area (dependent variable), $\log_e$ Sol SLR area (independent variable), and condition (covariance) with analysis of covariance

	Condition (Covariance)		
	Control	Sol vibration (SV)	Malleolus vibration (MV)
Regression Coefficient	0.68**	0.81**	0.64**
Intercept	1.90**	1.61**	2.25** ††

Log_e MLR area, $\log_e$ SLR area and condition are the dependent variable, independent variable and covariance, respectively. **: significance of regression coefficient and intercept ($p<0.01$). ††: significant difference ($p<0.01$) of intercepts between SV and MV conditions in post hoc test (The Bonferroni/Dunn test)

inhibit interneurons (disynaptic inhibition) via group I and II afferent fibers from the ankle joint. These clear delays in Sol SLR and MLR onsets under MV support the notion that disynaptic inhibition of interneurons connected to group I and II afferent fibers from the ankle joint extend the time required temporal summation of Sol α-motoneurons.

In the regression analyses of the Sol MLR and SLR areas under the three conditions, the coefficients regression of the SV and MV conditions were not different. In contrast, the intercept under the MV conditions was greater than under the SV condition. Therefore, this result indicates that Sol MLR activity increases when vibration is applied to the ankle joint but decreases when vibration is applied to the homonymous muscles. It is documented that the Sol SLR and MLR areas with toe-up rotation, during Achilles tendon vibration of 90 Hz, of standing subjects exhibited significant decreases in comparison to the absence of vibration[14]. This finding is explained by the presynaptic inhibition of Ia and group II afferents from muscle spindles which are being vibrated[16, 21–23]. Sol MLR area under the SV condition is consistent with the results of previous studies in which vibration was applied to Sol. Therefore, we think that vibration applied to Sol α-motoneurons decreased because of presynaptic inhibition of Ia and group II afferent fibers induced by Sol vibration.

However, the intercept of the regression line was higher under the MV condition than under the SV condition. This result does not support the theory of disynaptic inhibition alone, because the Sol MLR area during the MV condition increased. When subjects support themselves by holding onto a stable frame, then the same foot rotation elicits responses of a smaller magnitude, of less than 20% on the average of the control value [23, 26]. This finding led to the conclusion that monoaminergic brain stem centers selectively modulate the stimulation of the interneuronal pathways responsible for the transmission of group II input[19]. In our pilot study, the latencies and amplitudes of Sol SLR and MLR were respectively delayed and decreased by evoked-potentials (MEB-9404, Nihon-Kohden Inc., Japan), when vibration was applied to the malleolus of stable standing subjects, compared to the control condition (unpublished). That is, monoaminergic brain stem centers may compensatorily excite the interneuronal pathway via group I and II afferents from the ankle joint while disynaptic inhibition took place during ankle vibration under unstable standing conditions. This compensatory effect of monoaminergic brainstem centers was corroborated by the results of another of our studies, which demonstrated that the SLR and MLR areas of plantae muscles decreased under the same MV condition[27]. The monoaminergic brain stem centers may predominantly modulate reflexive Sol activity when standing conditions are unstable, and afferent signals from the ankle joint would be inhibited because plantae muscle activity is strongly inhibited by excessive afferent signals from the ankle joint during vibration. Moreover, Sol activity is directly related to the intensity of the ground reaction force and the displacement of the center of pressure[28, 29].

We should point out that compensatory modulation from the supraspinal centers had little effect on the Sol MLR area under the SV condition similar to the MV condition. This

discrepancy might be caused by the character of the perturbation. The perturbation used in our present study was the backward perturbation of a platform. It is known that the decrement of afferent signals from the ankle increases error in passive position sense of the ankle joint, but does not affect stability during static standing because of compensatory inputs from muscle spindles as a result of the anesthetsia of the ankle joint[9]. The afferent signals from Sol muscle spindles would contribute to stability during static standing, however the afferent signals from the ankle joint rather than from Sol muscle spindles might contribute to reflexive Sol activity during passive movement of the feet. Therefore, supraspinal centers might not participate in modulating the stimulation of interneuronal pathways involved in Ia and group II afferent fibers from Sol muscle spindles under SV, even if the afferent fibers from Sol muscle spindles resulted in presynaptic inhibition during Achilles' tendon vibration.

Another point we must notice is the composition type and motor units of the Sol muscle fibers. Previous investigations have demonstrated that the Sol fibers consist of 81% type I, 16% type IIa and 0% type IIb fibers[30]. Moreover, it has been proposed that α-motoneurons that innervate fiber types I and II mainly have connections with group II and group I afferent fibers in the peripheral nerves[31]. Since the fiber type of Sol is mostly type I fiber, group II afferent fibers from the ankle joint and other group II afferent fibers from Sol muscle spindles that mediate Sol MLR activity might form concentrative synaptic connections with Sol α-motoneurons innervating type I fibers. Therefore, monoaminergic brain stem centers might provide compensatory stimulus to the α-motoneurons of Sol, even though the Sol MLR area would be expected to decrease with disynaptic inhibition of group II afferents under MV.

REFERENCES

1) Nardone A, Grasso M, Giordano A, et al.: Different effect of height on latency of leg and foot short- and medium- latency EMG responses to perturbation of stance in humans. Neurosci Lett, 1996, 206: 89–92. [Medline] [CrossRef]
2) Schieppati M, Nardone A, Siliotto R, et al.: Early and late stretch responses of human foot muscles induced by perturbation of stance. Exp Brain Res, 1995, 105: 411–422. [Medline]
3) Schieppati M, Nardone A: Group II spindle afferent fibers in humans: their possible role in the reflex control of stance. Prog Brain Res, 1999, 123: 461–472. [Medline] [CrossRef]
4) Nardone A, Galante M, Pareyson D, et al.: Balance control in sensory neuron disease. Clin Neurophysiol, 2007, 118: 538–550. [Medline] [CrossRef]
5) Nardone A, Schieppati M: Group II spindle fibres and afferent control of stance. Clues from diabetic neuropathy. Clin Neurophysiol, 2004, 115: 779–789. [Medline] [CrossRef]
6) Nardone A, Tarantola J, Miscio G, et al.: Loss of large-diameter spindle afferent fibres is not detrimental to the control of body sway during upright stance: evidence from neuropathy. Exp Brain Res, 2000, 135: 155–162.
7) Kavounoudias A, Roll R, Roll JP: Foot sole and ankle muscle inputs contribute jointly to human erect posture regulation. J Physiol, 2001, 532: 869–878. [Medline] [CrossRef]
8) Sakita M, Takasugi S, Kawano I, et al.: Effects of displacements of center of gravity and center of pressure on standing postural responses to platform translations after cooling of soles and crural muscles. Rigakuryoho Kagaku, 2007, 22: 449–455 (in Japanese). [CrossRef]
9) Konradsen L, Ravn JB, Sørensen AI: Proprioception at the ankle. The effect of anaesthetic blockade of ligament receptors. J Bone Joint Surg Br, 1993, 75: 433–436. [Medline]
10) Schieppati M, Nardone A: Time-course of 'set'-related changes in muscle responses to stance perturbation in humans. J Physiol, 1995, 487: 787–796. [Medline]
11) Nardone A, Corrà T, Schieppati M: Different activations of the soleus and gastrocnemii muscles in response to various types of stance perturbations in man. Exp Brain Res, 1990, 80: 323–332. [Medline] [CrossRef]
12) Nardone A, Giordano A, Corrà T, et al.: Responses of leg muscles in humans displaced while standing. Effects of types of perturbation and of postural set. Brain, 1990, 113: 65–84. [Medline] [CrossRef]
13) Noda K, Umeda F, Asou N, et al.: Correlation of peripheral nerve fatigue following vibratory stimulation with hyperglycemia in diabetic patients. Diabetes Res Clin Pract, 1994, 25: 27–33. [Medline] [CrossRef]
14) Bove M, Nardone A, Schieppati M: Effects of leg muscle tendon vibration on group Ia and group II reflex responses to stance perturbation in humans. J Physiol, 2003, 550: 617–630. [Medline] [CrossRef]
15) Burke D, Hagbarth KE, Löfstedt L, et al.: The responses of human muscle spindle endings to vibration of non-contracting muscles. J Physiol, 1976, 261: 673–693. [Medline]
16) Hagbarth KE, Wallin G, Lofstedt L: Muscle spindle responses to stretch in normal and spastic subjects. Scand J Rehabil Med, 1973, 5: 156–159. [Medline]
17) Jankowska E: Interneuronal relay in spinal pathways from proprioceptors. Prog Neurobiol, 1992, 38: 335–378. [Medline] [CrossRef]
18) Mountcastle VB, LaMotte RH, Carli G: Detection thresholds for stimuli in humans and monkeys: comparison with threshold events in mechanoreceptive afferent nerve fibers innervating the monkey hand. J Neurophysiol, 1972, 35: 122–136. [Medline]
19) Inami K, Chiba K, Toyama Y: Determination of reference intervals for vibratory perception thresholds of the lower extremities in normal subjects. J Orthop Sci, 2005, 10: 291–297. [Medline] [CrossRef]
20) Westlake KP, Culham EG: Sensory-specific balance training in older adults: effect on proprioceptive reintegration and cognitive demands. Phys Ther, 2007, 87: 1274–1283. [Medline] [CrossRef]
21) Edgley SA, Jankowska E: An interneuronal relay for group I and II muscle afferents in the midlumbar segments of the cat spinal cord. J Physiol, 1987, 389: 647–674. [Medline]
22) Jankowska E, Slawinska U, Hammar I: Differential presynaptic inhibition of actions of group II afferents in di- and polysynaptic pathways to feline motoneurones. J Physiol, 2002, 542: 287–299. [Medline] [CrossRef]
23) Jankowska E, Slawinska U, Hammar I: On organization of a neural network in pathways from group II muscle afferents in feline lumbar spinal segments. J Physiol, 2002, 542: 301–314. [Medline] [CrossRef]
24) Marchand-Pauvert V, Nicolas G, Burke D, et al.: Suppression of the Hreflex in humans by disynaptic autogenetic inhibitory pathways activated by the test volley. J Physiol, 2002, 542: 963–976. [Medline] [CrossRef]
25) Freeman MA, Wyke B: The innervation of the ankle joint. An anatomical and histological study in the cat. Acta Anat (Basel), 1967, 68: 321–333. [Medline] [CrossRef]
26) Roll JP, Vedel JP, Ribot E: Alterration of proprioceptive messages induced by tendon vibration in man: a microneurographic study. Exp Brain Res, 1989, 76: 213–222. [Medline] [CrossRef]
27) Sakita M, Ishii Y, Takasugi S, et al.: Effects of short and medium latency reflexes of the plantae muscle with ankle vibration during sudden foot movement. J Phys Ther Sci, 2012, 24: 83–87. [CrossRef]
28) Sasagawa S, Ushiyama J, Masani K, et al.: Balance control under different passive contributions of the ankle extensors: quiet standing on inclined surfaces. Exp Brain Res, 2009, 196: 537–544. [Medline] [CrossRef]
29) Mezzarane RA, Kohn AF: Control of upright stance over inclined surfaces. Exp Brain Res, 2007, 180: 377–388. [Medline] [CrossRef]
30) Henriksen EJ, Bourey RE, Rodnick KJ, et al.: Glucose transporter protein content and glucose transport capacity in rat skeletal muscles. Am J Physiol, 1990, 259: E593–E598. [Medline]
31) Kanning KC, Kaplan A, Henderson CE: Motor neuron diversity in development and disease. Annu Rev Neurosci, 2010, 33: 409–440. [Medline] [CrossRef]