J. Human Ergol., **37**: 91-102, 2008

ATTENTION OF POSTURAL CONTROL ON FOOT SOMATOSENSOR DISTURBANCE CAUSED BY THE COMPRESSION OF BLOOD VESSELS

SHINICHI DEMURA[1], SHUNSUKE YAMAJI[2*], TAMOTSU KITABAYASHI[3], TAKAYOSHI YAMADA[4] AND MASANOBU UCHIYAMA[5]

[1] *Department of Physical Education, Kanazawa University, Kakuma, Kanazawa 920-1192, Japan*
[2] *University of Fukui, Faculty of Medical Sciences, Sport Medicine, 23-3, Shimoaizuki, Matsuoka, Eiheiji, Yoshida, Fukui 910-1193, Japan / *Email: yamaji@u-fukui.ac.jp*
[3] *Tokyo University of Science, Faculty of Science Division 1, 1-3, Kagurazaka, Shinjuku, Tokyo 162-8601, Japan*
[4] *Fukui National College of Technology, Geshi, Sabae 916-8507, Japan*
[5] *Kanazawa College of Art, 5-11-1, Kodatsuno, Kanazawa 920-8656, Japan*

This study aimed to compare the center of foot pressure (CFP) during different ischemia periods by cuff compression. Ten healthy young adult males held their CFP for 1 min before and after cuff compression (250 mmHg) of lower limb blood vessels during two different compression periods (10 min and 26 min). Two-point discrimination thresholds and electric stimulus perception thresholds of each subject's right foot sole were measured during cuff compression to examine changes in the perceived level of cutaneous and proprioceptive sensation by blood flow inhibition. In addition, oxygenation kinetics in the anterior tibial muscle tissue was examined by near-infrared spectroscopy during cuff compression. The CFP was evaluated by using 36 parameters. CFP deflection tended to increase after cuff compression and to be larger over a long ischemic time (26 min) as compared with a short ischemic time (10 min). The proprioceptive perception threshold using electric stimulation showed a significant interaction and was higher after 26 min ischemia than at the initial timepoint and after 10 min ischemia. The Δ[Oxy Hb/Mb], Δ[Deoxy Hb/Mb] and Tissue oxygenation index (TOI) changed significantly after both ischemic conditions than at the initial condition. There was no significant difference in the Tissue oxygenation index and Δ[Deoxy Hb/Mb] between both ischemic conditions. Many parameters regarding the deflection velocity and anteroposterior and lateral deflection had significantly larger values after 26 min ischemia than at the initial time point and after 10 min ischemia. Postural deflection may not be affected by the oxygen deficiency of muscle tissues caused by the compression of blood vessels for 10 min, but strongly affected by the cutaneous and proprioceptive sensation disturbances occurring from compression lasting over 10 min.

Key words: blood flow; center of pressure; cutaneous sensory disturbance; near infrared spectroscopy; somatosensor

INTRODUCTION

Standing posture is controlled by integrated afferent information from the vestibule/semicircular canal system, the optic system and the somatesthetic system in the cerebellum and brain stem (Markham, 1987). The deflection degree is generally recorded in two dimensional time series coordinates of the center of foot pressure (CFP) displacement.

In order to clarify the mechanism of standing posture control, earlier studies stimulated the function or organ targeted for disturbance and examined the trends of deflation. For example, Wada and Sasaki (1990) studied the contribution of visual information for attitude retention, examining the

Received 12 September 2007; accepted 30 August 2008.

results of a condition in which only darkroom conditions and the foveal vision field are given as factors in obstructing the optic system.

In addition, many researchers have examined changes in CFP deflection by alcohol intake, as a disturbance stimulus of the vestibulum/semicircular canals system and the nervous system (Kubo et al., 1989; Uimonen et al., 1994; Mangold et al., 1996; Nieschalk et al., 1999; Demura et al., 2001; Yamaji et al., 2001; Kitabayashi et al., 2002, 2003). Because alcohol intake is an easy way to disturb the vestibulum/semicircular canals and the nervous system, it is used in many studies (Franks, et al., 1976; Kubo et al., 1989; Uimonen et al., 1994; Roebuck, et al., 1998; Nieschalk, et al., 1999).

The somatesthetic system contributes to attitude retention by transmitting the sensory information as afferent information after perceiving body sway from the sole. Thus, the somatesthetic system plays an important role in postural control together with the vestibulum/semicircular canals system and the optic system (Ekdahl et al., 1989; Grigorova et al., 2001). In previous studies, the contribution of the somatesthetic system, such as cutaneous sensation and proprioceptors (muscle and nurotendinous spindles), was examined by using various intervention stimuli by vibration (Ledin et al., 2003; Stål et al., 2003; Meyer et al., 2004), muscle fatigue (Vuillerme et al., 2005), iontophoretic anesthesia (Magnusson et al., 1990), cooling (Sakita et al., 2006), compression (Pollak et al., 1976; Mori, 1987, Demura and Uchiyama, 2005a, 2005b) and foam rubber-platform and sponge (Grigorove et al., 2001).

These methods aimed to disturb proprioceptive or cutaneous sensation. Rothwell (1994) reported that the frequency of body sway enhanced by about 1 Hz when the proprioceptive sensation of the lower leg and foot were paralyzed by low-frequency electric stimuli in a healthy person. He also reported that when paralyzing only the proprioceptive sensation of the foot, the frequency of body sway appeared in the lower band (below 1Hz) only during standing with the eyes closed. Therefore, the effect of disturbance on body sway is considered to be larger for proprioceptive sensation than for cutaneous sensation. The disturbance in cutaneous sensation causes loss of the spinal cord-mediated postural reflexes. Moreover, when disturbing the action of muscle spindles for proprioceptive sensation, maintaining a standing position becomes difficult (Rothwell, 1994).

In Japanese daily living, Japanese styles of sitting, like *seiza* (sitting straight) and *agura* (sitting cross-legged), are considered to cause ischemia in the thighs and to decrease proprioceptive and cutaneous sensation. Femoral arteries delivering oxygen to the cells of the lower limb tissues reach the anterior and posterior tibial recurrent arteries through the inside of the knee joints (popliteal artery). It is reported that due to large knee flexion, *seiza* has harmful effects not only on the skeletal system but also on the hemodynamics of the lower limbs. Ischemia in the lower extremities causes an oxygen deficiency in muscles or nerve cells, and abnormal cutaneous sensation and disturbance of proprioceptive sensation occur (Iwanaga et al., 1993; Demura and Uchiyama, 2005a, 2005b).

In addition, physiological responses, including the respiratory and cardiovascular systems, can lead to large sway during standing posture. Thus, just after releasing hemostasis (e.g. standing from a Japanese-style sitting position), temporal anaemia caused by a sudden fall of blood pressure may disturb posture control.

Demura and Uchiyama (2005a, 2005b) reported that 30 min of *seiza* caused an increase in the changes of the CFP. However, it is possible that the influence of *seiza* on thigh ischemia differs between individuals because of differences in each subject's body mass, anatomic locations of blood vessels and muscle mass. Moreover, it has not been clarified whether the influence of ischemia on body sway occurs at the time of ischemia onset or at the time of cutaneous or proprioceptive sensation loss.

This study aimed to examine the effect on the CFP of cutaneous or propriocetive sensation loss induced by different ischemia periods and re-perfusion.

MATERIALS AND METHODS

Subjects

Ten healthy male adults (age: 21.2 ± 3.1 years, height: 173.0 ± 6.2 cm, body weight: 66.6 ± 6.2 kg) participated in this study. The subjects' physical characteristics were almost the same as the age-matched national standard values (Laboratory of Physical Education in Tokyo Metropolitan University, 2000). All the subjects were informed in advance of the contents of the experiment and any attendant risks. They gave their written agreement to voluntarily participate in the experiments. This study was approved by the Kanazawa University human subjects ethics committee.

Experimental design

All subjects used a cuff that was wrapped around the proximal side of the thigh. Their thigh was then compressed at 250 mmHg. Based on the result of a preliminary experiment, two different compression periods (10 min and 26 min) were used in the present study. In the 10 min period, tissue oxygenation, monitored by near infrared spectroscopy (NIRS) placed on the pressurized lower leg, changed and reached a steady state. A similar phenomenon was observed in a previous study (Kimura et al., 1999).

In the 26 min period, cutaneous sensation level in the foot sole decreased markedly. We examined the effect of blood flow inhibition on the CFP deflection and the perceived level of cutaneous and proprioceptive sensation before and after cuff inflation in both compression periods. The measurement was done once a day in either condition with at least a one-day interval until the subsequent experiment. The plan was conducted under a crossover design of both cuff compression conditions.

Experimental protocol

Figure 1 shows an outline of the experiment procedure used in this study. First, the CFP deflection and the perceived level of foot cutaneous and proprioceptive sensation were measured after the subject was in a sitting position for 10 min (as initial condition data). In the measurement of CFP, the subject stood barefooted on a stabilometer footprint (G5500, Anima, Japan) and kept Romberg's posture in which the inner sides of both feet touched, while looking forward at a fixed point (2m). The CFP measurement was done twice under the initial condition and once under the cuff condition. The rest (sitting position on high back chair) between trials was for one minute. All subjects practiced CFP measurement once prior to the experiment. Afterwards, their blood flow to the lower limb was occluded by compression at 250 mmHg in the femoral region during sitting (Spygmo Manometer, VAN, JAPAN). After the designated time, the subject's CFP and the perceived level of cutaneous sensation in standing position were re-measured immediately after removing the cuff. The state of tissue oxygenation in the pressurized leg (tibialis anterior muscle) was monitored throughout the experiment using NIRS. In addition, all subjects were instructed to avoid sleep, heavy exercise, eating and drinking for two hours before the experiment.

Tissue oxygenation

An NIRS instrument (NIRO-300, Hamamatsu Photonics, Japan) was used in this study. The NIRS instrument utilized four wavelengths (775, 813, 850 and 913 nm) with an algorithm based on the Lambert-Beer theory to measure the oxygenation change in the tibialis anterior muscle. The instrument provided separate measurements of changes in oxygenated Mb and Hb concentrations (Δ[Oxy Hb/Mb]), changes in deoxygenated Mb and Hb concentrations (Δ[Deoxy Hb/Mb]) and tissue oxygen saturation (tissue oxygenation index: TOI). The values were quantified with respect to initial control values arbitrarily set equal to zero and were expressed as changes from the initial time (Δ[Oxy Hb/Mb] and Δ[Deoxy Hb/Mb]: nmol/L, TOI: %). The probe unit consisted of a photodiode as the photo detector and a light-emitting diode. The distance between the photodiode and the LED was 5 cm. The probe was attached to the skin with an adhesive tape. Pen marks were made over the

Experimental condition 1						
Time	10 min	1 min	2 min	10 min	1 min	
Protocol	Rest	Standing	Rest	Ischemia by cuff	Standing	
CFP mesurement		CFP			CFP	
Oxygenation kinetics by NIRS			Continuous measurement with 1 Hz sampling frequency			
Electric stimulus perception			▲	Every 1 min with cuff		▲
Two-point discrimination			△	Every 2 min with cuff		△

Experimental condition 2						
Time	10 min	1 min	2 min	26 min	1 min	
Protocol	Rest	Standing	Rest	Ischemia by cuff	Standing	
CFP mesurement		CFP			CFP	
Oxygenation kinetics by NIRS			Continuous measurement with 1 Hz sampling frequency			
Electric stimulus perception			▲	Every 1 min with cuff		▲
Two-point discrimination			△	Every 2 min with cuff		△

Fig. 1. Scheme of the experiment in this study. ▲ and △ are measurements after CFP.

skin to indicate the edges of the probe to check for any sliding and for accurate probe repositioning in the subsequent experiment. The sampling frequency of the NIRS was 1 Hz.

Effect of subcutaneous fat thickness

The skin and subcutaneous fat thickness was measured by using a B-mode ultrasound imagine machine (EUB-200, Hitachi Medical, Japan) on the surface of the tibialis anterior muscle, and ranged from 2 ~ 4 mm among the subjects. *In vivo* NIRS measurement must account for the fat layer since the light path length (depth of the detection layer) depends on the distance between the light source and the detector. According to Monte Carlo simulation studies of skin, adipose, and muscle layer scattering and absorption characteristics for NIR light, as well as *in vivo* measurements, a source-detector spacing of 2 cm is enough to detect the NIR light passing through the muscle layer, even when the thickness of the adipose tissue is 15 mm. Thus the 5 cm source-detector distance of the instrument (NIRO-300) utilized in the present study seems adequate to follow oxygenation changes in a shallow area of the superficial muscle.

Electric stimulus perception threshold

The electric stimulus perception threshold in the right foot sole was measured to examine the change in the perceived level of proprioceptive sensation by blood flow inhibition. A medical use low frequency therapy device (HV-F125, OMRON, Japan) was used for the measurement. The two electrodes were attached to the right foot sole separated by 5 cm. The voltage of the device was increased slowly, and the voltage where a subject felt the electric stimulus was recorded.

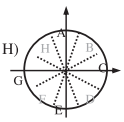
Two-point discrimination threshold

A two-point discrimination threshold in the right foot sole of a subject was measured to examine the perceived level of cutaneous sensation and to measure the cutaneous sensation threshold. The two-point discrimination threshold (cm) at rest was assumed as the reference, and two points were recorded every 2 min during ischemia.

Center of foot pressure (CFP)

A stabilometer was used (G5500, Anima, Japan) for CFP measurement. This instrument can calculate the CFP of vertical loads from the values of three vertical load sensors, which are put on the peak of an isosceles triangle on a level surface. Data was sampled at 20 Hz and transferred to a PC following A/D conversion (Demura et al., 2001; Kitabayashi et al., 2002, 2003). The evaluation parameters for CFP deflection were 36 variables with trial-to-trial reliability and logical validity (Demura et al., 2001; Yamaji et al., 2001; Kitabayashi et al., 2002, 2003) (Table 1).

Table 1. Characteristics of 36 COP parameters. (Kitabayashi. T. et al., 2003).

Domains	№	Parameters	Unit	Characteristics
Factor 1: Deflection velocity	1	Mean path length	cm/s	Mean length of center of foot pressure (CFP) path
	2	Standard deviation of X-axis sway	cm/s	Equation: $S_x = \sqrt{\sum (X_i - \bar{X})^2 / N}$
	3	Standard deviation of Y-axis sway	cm/s	Equation: $S_y = \sqrt{\sum (Y_i - \bar{Y})^2 / N}$
	4	Mean velocity of X-axis sway	cm/s	Mean velocity of X-, Y-axis for body-sway
	5	Mean velocity of Y-axis sway	cm/s	
	6	Root mean square of velocity	cm/s	Root mean square of sway velocity
	7	Mean vector length of A direction velocity	cm/s	Mean distance of body-sway velocity in 4 directions (A to H) 
	8	Mean vector length of C direction velocity	cm/s	
	9	Mean vector length of E direction velocity	cm/s	
	10	Mean vector length of G direction velocity	cm/s	
Factor 2: Anteroposterior deflection	11	Root mean square	cm	Equation: $\sqrt{\frac{1}{2}\left\{\left(\sum X_i - \bar{X}\right)^2 + \left(\sum Y_i - \bar{Y}\right)^2\right\} / N}$ Dispersion from CFP
	12	Root mean square of Y-axis	cm	Equation: $\sqrt{\left(\sum Y_i - \bar{Y}\right)^2 / N}$ Dispersion from CFP for Y-axis
	13	Standard deviation of Y-axis velocity	cm	Standard deviation of Y-axis velocity
	14	Area surrounding mean path length	1/cm	The circumference area divided into total path length
	15	Area surrounding maximal amplitude rectangle	cm^2	Area surrounding maximal amplitude rectangle for each axis
	16	Area surrounding root mean square	cm^2	Area of the circle making the actual effective radius value
	17	Mean vector length of A direction sway	cm	Mean distance of body-sway sway in back and forth directions (A and E)
	18	Mean vector length of E direction sway	cm	
	19	Mean CFP of Y-axis	cm	Mean displacement of CFP for Y-axis
	20	Ratio of A domain for power spectrum of Y-axis	%	Power spectrum area by the Fourier translate for the body-sway value (Y-, R-direction) divided A domain. A domain; 0-0.2 Hz
	21	Ratio of A domain for power spectrum of R-axis	%	
Factor 3: Lateral direction	22	Root mean square of X-axis	cm	Equation: $\sqrt{\left(\sum X_i - \bar{X}\right)^2 / N}$ Dispersion from CFP for X-axis
	23	Standard deviation of X-axis velocity	cm	Standard deviation of X-axis velocity
	24	Ratio of A domain for power spectrum of X-axis	%	Power spectrum area by the Fourier translate for the body-sway and velocity value (X-direction) divided by A domain. A domain; 0-0.2 Hz
	25	Ratio of A domain for power spectrum of X-axis velocity	%	
	26	Mean vector length of C direction sway	cm	Mean distance of body-sway in 4 directions (A to H)
	27	Mean vector length of G direction sway	cm	
	28	Ratio of A domain for power spectrum of Y-axis velocity	%	Power spectrum area by the Fourier translate for the body-sway velocity value (Y-direction) divided by A domain
Factor 4: High frequency band of power spectrum	29	Ratio of C domain for power spectrum of X-axis sway	%	Power spectrum area by the Fourier translate for the body-sway value (X-, Y-, R-direction) divided C domain. C domain; above 2 Hz
	30	Ratio of C domain for power spectrum of Y-axis sway	%	
	31	Ratio of C domain for power spectrum of R-axis sway	%	
	32	Ratio of C domain for power spectrum of X-axis velocity	%	Power spectrum area by the Fourier translate for the body-sway velocity (X-, Y-, R-direction) divided by C domain. C domain; above 2 Hz
	33	Ratio of C domain for power spectrum of Y-axis velocity	%	
	34	Ratio of C domain for power spectrum of R-axis velocity	%	
	35	Mean CFP of X-axis	cm	Mean displacement of CFP for X-axis
	36	Ratio of A domain for power spectrum of R-axis velocity	%	Power spectrum area by the Fourier translate for the body-sway velocity (R-direction) divided by A domain.

Statistical analysis

This study had two different ischemia periods, and each experiment was carried out on a separate day. Therefore, the reproducibility between trials had to be examined. In regard to tissue oxygenation and the cutaneous sensation threshold, the cross correlation coefficient of the two trials until the 10 min ischemia period was calculated in order to examine reproducibility. The paired Student's t-test was used to compare between values obtained at rest and at 10 min of ischemia. In order to examine the effect of two different ischemia times on tissue oxygenation and cutaneous sensation, two-way ANOVA (ischemia time and day) was conducted. After checking that there were no significant differences in the CFP parameters during the rest between two ischemia periods, one-way ANOVA was used to reveal the mean difference of the CFP between rest, 10 min and 26 min after ischemia. This data model for analysis was hypothesized the same time-series data. Tukey's HSD test was used as a multiple comparison. The level of statistical significance was set at $p<0.05$.

RESULTS

Figure 2 shows the mean change in tissue oxygenation kinetics and cutaneous and proprioceptive sensation thresholds during and after cuff compression. Although each ischemia condition was carried out on a different day, there was a very close resemblance in the trend of either NIRS parameter until 10 min. All oxygenation parameters reached an almost steady state at 10 min. Two-point discrimination thresholds evaluating the cutaneous sensation did not decrease in all subjects for 10 min, and decreased markedly after 12 min. On the other hand, electric stimulus perception threshold evaluating the proprioceptive sensation tended to increase slowly after the compression and increase markedly after 13 min.

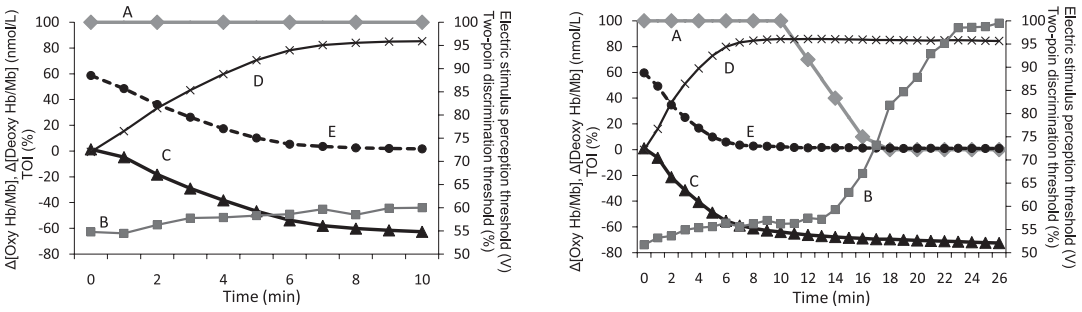


Fig. 2. Average change curve in cutaneous sensation thresholds and tissue oxygenation kinetics during ischemia. Left panel; 10 min, Right panel; 26 min.
A: Percentage of subjects unsuccessful in two-point discrimination, B: Electric stimulus perception thresholds, C: Δ[Oxy Hb/Mb], D: Δ[Deoxy Hb/Mb], E: TOI

Table 2 shows the results of the mean difference and cross correlation coefficients of NIRS parameters and the electric stimulus perception threshold for 10 min between both ischemia conditions (different days). To compare both conditions, the time-series data in 26 min ischemia condition used for analysis was sampled during the initial 10 min. Two-point discrimination threshold was excluded from the statistical analysis, because it did not decrease during 10 min in all subjects (Fig. 2). The cross correlation coefficient was more than 0.9 for either variable (Table 2). In addition, there was no significant difference between average values of each variable obtained under different ischemia conditions.

Table 2. Mean differences and cross-correlation coefficients for 10 min between both ischemia conditions regarding NIR parameters and the cutaneous sensation threshold.

Parameters	10 min		26 min		t-value	Rxy	SE	lag
	Mean	SD	Mean	SD				
Δ[Oxy Mb/Hb]	6.00	1.11	5.62	1.23	1.30	0.999	0.041	0
Δ[Deoxy Mb/Hb]	-62.56	6.65	-63.87	4.05	0.52	0.998	0.041	0
TOI	85.24	9.05	85.70	4.86	-0.19	0.994	0.041	0
Cutaneous sensation threshold	1.70	1.61	2.26	2.45	-1.06	0.929	0.302	0

Note: There is no significant difference in any parameter. Rxy: Cross-correlation coefficient between both ischemia conditions at initial 10 min.

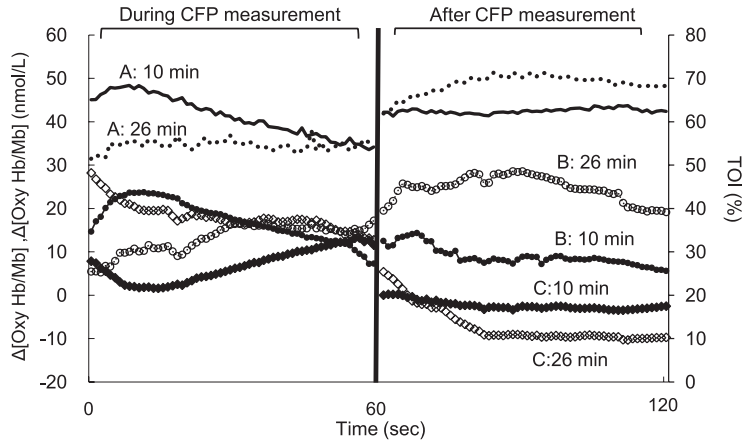


Fig. 3. Average changes of tissue oxygenation for one minute after cuff was taken off and the CFP measurements ended. A: TOI, B: Δ[Oxy Hb/Mb], C: Δ[Deoxy Hb/Mb]

Figure 3 shows the mean change of tissue oxygenation for two min after the cuff compression. The tissue oxygenation after 26 min ischemia was constant but lower than that after 10 min ischemia. Δ[Deoxy Hb/Mb] after the 10 min ischemia increased, whereas it after 26 min decreased from a higher level.

Table 3 shows the result of two-way ANOVA (ischemia condition, time) for tissue oxygenation and the cutaneous sensation threshold before and after the cuff compression. The electric stimulus perception threshold (V) showed a significant interaction, and it was significantly higher after 26 min ischemia than at the initial condition and after 10 min ischemia. For tissue oxygenation, there was a significant interaction for Δ[Oxy Hb/Mb], and was a significant factor in the tissue oxygenation index (TOI) and Δ[Deoxy Hb/Mb] before and after the cuff compression. The Δ[Oxy Hb/Mb] and TOI showed significantly lower values in both ischemic conditions than in the initial condition. The Δ[Deoxy Hb/Mb] was significantly higher in both ischemic conditions than in the initial condition. There was no significant difference in tissue oxygenation index and Δ[Deoxy Hb/Mb] between both ischemic conditions. Because of the quite similar value of the initial conditions and similar time sequence of the NIRS parameters obtained on separate days, all events were considered to be equivalent ischemia conditions through the same time series from the initial condition to both ischemia time conditions.

Figure 4 shows a typical example of CFP deflection affected by ischemia. CFP deflection after ischemia tended to be larger than that at the initial condition. In addition, a prolonged ischemia time (26 min) produced more CFP deflation than 10 min.

Table 4 shows the result of one-way ANOVA for the CFP deflection levels of the initial condition and after ischemia (10 min later and 26 min later). In the initial condition, the average value of

Table 3. Two-way ANOVA (Condition × time) for tissue oxygenation and the cutaneous sensation threshold.

Parameters	10 min				26 min				Two way ANOVA			Post-hoc (Tukey's HSD)		
	Initial		Cuff out		Initial		Cuff off		F	F	F	Condition	Time	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Condition	Time	Interaction	Cuff out	10 min	26 min
Cutaneous sensation threshold	5.48	0.93	6.00	1.06	5.17	1.08	9.95	0.15	38.71 *	245.92 *	88.65 *	10 < 26		I < C
Δ[Oxy Mb/Hb]	1.41	4.85	-62.56	6.31	1.09	1.42	-72.46	4.08	8.64 *	3546.03 *	24.95 *	10 > 26	I > C	I > C
Δ[Deoxy Mb/Hb]	-0.69	4.75	85.24	8.59	-0.25	2.34	84.20	5.29	0.02	2093.81 *	0.34		I < C	I < C
TOI	58.65	4.62	1.70	1.53	59.52	6.56	0.96	1.07	0.01	1521.30 *	0.95		I > C	I > C

Note: *: $p < 0.05$, I > C : Parameter at cuff out was significantly lower than one at initial condition.

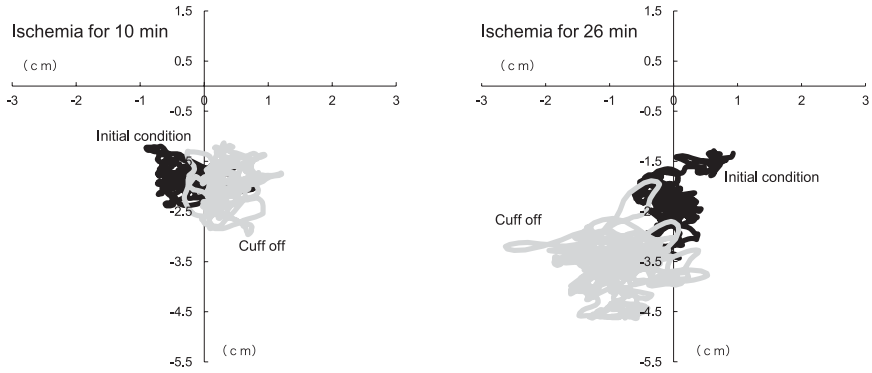


Fig. 4. Typical examples of CFP deflection at initial timepoint and cuff-off conditions.

CFP deflection for both experimental days was calculated. The parameters regarding deflection velocity (parameter No. 1-10) for 26 min ischemia were significantly higher in comparison with the initial level and 10 min ischemia. For parameters regarding anteroposterior deflection and lateral deflection (parameter No. 11-28), the CFP deflection distance at 26 min ischemia was significantly greater than that in other conditions.

Other deflection distance variables tended to be higher for 26 min ischemia than for other conditions, even though not significantly. In the parameters regarding high frequency power spectrum (parameter No. 29-36), there was no significant difference between the conditions, except for the X-axis of mean CFP deflection. The mean CFP position, which shows the mean displacement of deflection, did not reveal any significant difference between the conditions except for the Y-axis.

DISCUSSION

In order to occlude the blood flow to the lower leg of a subject, the cuff around the thigh was inflated at 250 mmHg. This pressure was the same as in a previous study (Aaslid et al.:1989, Traon et al.: 2002). Tissue oxygen saturation fell remarkably between 360 seconds from the beginning of compression, and kept a constant state afterwards. Therefore, it is supposed that arteriovenous ischemia was almost complete at that point.

When ischemia was done for a long time using cuff inflation, some physiological changes are observed (Iwanaga et al., 1993). First, ischemia of lower extremities inhibits oxygen supply to a leg muscle group, and causes loss of cutaneous and proprioceptive sensation (numbness of leg) and a fall of muscle contractile properties. The blood pressure rises temporarily due to the reduction of the circulation region by cuffing. Afterwards, the cuff around the thigh is released just before CFP measurement. Immediately after releasing the cuff, because blood flow to tissue increases remarkably by reperfusion, oxygen is supplied to each tissue. However, loss of cutaneous and proprioceptive sensations are not recovered immediately. In addition, almost all subjects complained of subjective symp-

Table 4. One way ANOVA for CFP deflection parameters of initial condition and after ischemia (10 min and 26 min).

Parameters	Initial		10 min		26 min		ANOVA	Post-hoc (HSD)
	Mean	SD	Mean	SD	Mean	SD	F-value	
1 Mean path length	0.94	0.20	1.06	0.25	2.28	0.72	27.03 †	Initial, 10 < 26
2 Standard deviation of X-axis sway	0.82	0.20	0.96	0.24	2.05	0.66	26.84 †	Initial, 10 < 26
3 Standard deviation of Y-axis sway	0.98	0.19	1.08	0.27	2.23	0.61	31.56 †	Initial, 10 < 26
4 Mean velocity of X-axis sway	0.59	0.12	0.64	0.16	1.34	0.35	30.78 †	Initial, 10 < 26
5 Mean velocity of Y-axis sway	0.49	0.12	0.58	0.14	1.23	0.40	26.45 †	Initial, 10 < 26
6 Root mean square of velocity	1.28	0.27	1.45	0.34	3.03	0.87	30.87 †	Initial, 10 < 26
7 Mean vector length of A direction velocity	0.77	0.18	0.92	0.20	1.92	0.67	22.64 †	Initial, 10 < 26
8 Mean vector length of C direction velocity	0.92	0.19	1.03	0.22	2.14	0.66	27.34 †	Initial, 10 < 26
9 Mean vector length of E direction velocity	0.78	0.21	0.91	0.21	1.95	0.61	29.50 †	Initial, 10 < 26
10 Mean vector length of G direction velocity	0.95	0.19	1.05	0.25	2.03	0.56	31.42 †	Initial, 10 < 26
11 Root mean square	0.72	0.16	0.72	0.19	0.99	0.18	34.68 †	Initial, 10 < 26
12 Root mean square of Y-axis	0.57	0.16	0.53	0.11	0.70	0.12	12.19 †	Initial, 10 < 26
13 Standard deviation of Y-axis velocity	0.57	0.16	0.53	0.11	0.70	0.12	12.19 †	Initial, 10 < 26
14 Area surrounding mean path length	22.82	5.34	22.11	6.99	20.03	4.42	1.82	
15 Area surrounding maximal amplitude rectangle	6.23	1.98	7.59	4.14	14.80	4.25	33.67 †	Initial, 10 < 26
16 Area surrounding root mean square	1.76	0.75	1.73	0.90	3.16	1.08	31.74 †	Initial, 10 < 26
17 Mean vector length of A direction sway	0.70	0.19	0.61	0.13	0.94	0.32	8.47	
18 Mean vector length of E direction sway	0.65	0.20	0.70	0.28	0.82	0.08	4.09	
19 Mean CFP of Y-axis	-1.67	0.87	-1.14	0.99	-2.68	0.66	5.72	
20 Ratio of A domain for power spectrum of Y-axis	32.23	3.06	34.48	4.72	24.51	5.14	13.67 †	Initial, 10 > 26
21 Ratio of A domain for power spectrum of R-axis	26.25	2.59	26.41	3.67	18.45	4.35	6.51	
22 Root mean square of X-axis	0.41	0.09	0.48	0.16	0.69	0.14	23.64 †	Initial, 10 < 26
23 Standard deviation of X-axis velocity	0.41	0.09	0.48	0.16	0.69	0.14	23.64 †	Initial, 10 < 26
24 Ratio of A domain for power spectrum of X-axis	29.65	5.27	30.38	3.93	22.19	3.84	14.13 †	Initial, 10 > 26
25 Ratio of A domain for power spectrum of X-axis velocity	5.37	1.29	5.47	0.99	3.54	0.77	15.37 †	Initial, 10 > 26
26 Mean vector length of C direction sway	0.51	0.15	0.59	0.18	0.88	0.23	14.82 †	Initial, 10 < 26
27 Mean vector length of G direction sway	0.52	0.12	0.59	0.23	0.86	0.21	8.03	
28 Ratio of A domain for power spectrum of Y-axis velocity	6.74	1.55	7.03	2.30	3.92	0.82	9.07	
29 Ratio of C domain for power spectrum of X-axis sway	13.89	3.06	14.87	3.74	17.61	5.49	2.22	
30 Ratio of C domain for power spectrum of Y-axis sway	19.54	2.97	15.82	4.75	16.61	4.76	2.03	
31 Ratio of C domain for power spectrum of R-axis sway	16.10	3.09	14.41	2.18	21.19	5.02	6.01	
32 Ratio of C domain for power spectrum of X-axis velocity	24.22	2.97	28.23	2.82	30.23	6.38	5.02	
33 Ratio of C domain for power spectrum of Y-axis velocity	24.38	2.08	23.67	2.28	28.86	4.61	7.49	
34 Ratio of C domain for power spectrum of R-axis velocity	41.82	3.82	42.42	3.15	46.56	3.90	6.72	
35 Mean CFP of X-axis	0.18	0.32	0.05	0.77	-2.48	1.40	24.44 †	Initial, 10 > 26
36 Ratio of A domain for power spectrum of R-axis velocity	8.59	0.61	9.00	1.41	9.14	2.13	0.01	

Note: † : $p < \alpha'$ ($\alpha' = 0.05/36$)

toms, such as palsy or numbness at the thigh, and this may affect the minute control of muscle or joint action.

Furthermore, a temporary decrease in blood pressure may occur when a sitting position posture is assumed from a standing position posture. This sudden decrease of blood pressure would activate a baroreceptor reflex in the cervix and artery bow. Therefore, in the short period of time for a position change, blood pressure may be disrupted. Thus, with the effect of such a physiological change, CFP deflection would change before and after cuff inflation/release.

According to Kopell et al. (1962), the same symptom as entrapment neuropathy occurs with temporal compression of the body. There are the compression theory (Castaldo and Ochoa, 1984) and the occlusion theory (Sunderland, 1990) as the main cause of entrapment neuropathy. Although we cannot strongly assert the causes because the blood flow and the effect of mechanical stimulus to the peripheral nervous system were not measured in this study, the temporal compression of the thigh may produce nerve pressure in addition to the above-stated occlusion effect.

In the condition of arteriovenous occlusion (250~300mmHg), the oxygenation kinetics reflects the blood flow state, and the steady state after marked decrease of Δ[Oxy Hb/Mb] indicates the ischemic state (Kimura et al., 1999). The changing value of oxygenation kinetics in this study was similar in extent to the value in previous studies (Kimura et al., 1999, Bringard and Perrey, 2004).

We confirmed in a preliminary study that for the 10 min ischemia, [Oxy Hb/Mb] was the time reaching a minimum level, which suggested that oxygen stored in muscle tissues was running out. In addition, for 26 min, the cutaneous and proprioceptive sensation were lost as well as depletion of

stored oxygen. The change in Δ[Oxy Hb/Mb] and TOI at 10 min ischemia were 88.3% and 94.3% of those at 26 min, respectively, which implied the depletion of oxygen stored in muscle tissues. However, there were significant differences in muscle tissue oxygenation kinetics after releasing hemostasis between both ischemia periods. Peripheral blood flow after ischemia turns to hyperemia by reflex action. This response after the 26 min ischemia tended to be slow as compared with that after the 10 min ischemia. In the 26 min ischemia, the increase of TOI and Δ[Oxy Hb/Mb] after releasing hemostasis required more than 1 min and Δ[Deoxy Hb/Mb] continued to decline. Therefore, the recovery of oxygenation kinetics differed between the different ischemia periods, and it may affect body sway after releasing hemostasis.

In addition, although all subjects were successful in a two-point discrimination threshold test with an ischemia time of 10 min, they later became unsuccessful at 26 min. From this phenomenon, the difference in the both ischemia periods is the presence or loss of cutaneous and proprioceptive sensation. Although these two conditions were carried out on separate days, the cross correlation coefficient of time series variables for 10 min, where the tissue oxygen dynamic phase passed after cuff compression, showed an extremely high value in either variable (Table 2). Therefore, the present results from the initial condition to ischemia of 10 min and 26 min were considered to be due to one factor (time).

In the results of this study, a prolonged ischemia time of 26 min produced more CFP deflation than that of 10 min. In addition, the proprioceptive sensation threshold after 26 min ischemia was significantly larger than that at the initial condition and after 10 min ischemia. Important sensory receptors for maintaining body posture, such as muscle or neurotendinous spindles of the sole, may not work normally when compression lasts more than 10 min.

In the foot pressure center deflection of each subject, the CFP tracing for 10 min ischemia, was almost the same (unchanged) as that for the initial condition. On the other hand, CFP for 26 min ischemia swayed more in the antero-posterior direction in most subjects as compared with the initial condition, and the range became very remarkable. In addition, one subject deviated from the deflection measuring range spasmodically. Furthermore, all subjects complained of the subjective symptom of palsy at the foot after 26 min ischemia and this probably suggests that the proprioceptive sensation of the lower extremities was lost for 26 min ischemia and led to the greater CFP deflection in the present study.

Kitabayashi et al. (2003) interpreted four factors based on the result of the factor analysis: factors for deflection velocity, for anteroposterior deflection, for lateral direction and for the high frequency band of the power spectrum. Many parameters including deflection frequency (per time), anteroposterior deflection and lateral deflection showed higher values after 26 min ischemia than at the initial condition and after 10 min ischemia. The parameters regarding deflection velocity and deflection area after 26 min ischemia increased to a level twice as large as that in other conditions. This result suggests that size of postural deflation would be accompanied by rapid loss of proprioceptive sensation.

These observations also suggest that loss of proprioceptive sensation increased the velocity and magnitude of a postural deflection. One possibility assumed for the increase in the lateral deflection may be that the cuff compression is only on one leg, and the probability was weighted towards the leg which was not made ischemic during a standing position. Therefore, it cannot be concluded that lateral deflection is characteristic to be paralysis of proprioceptive sensation only from the result of this study.

A remarkable increase in standard deviation of deflection velocity in all directions for 26 min ischemia suggests an increase in spasmodic postural deflections (Figure 4). Rothwell (1994) reported that paralysis of proprioceptive sensation of both the lower leg and the foot brought about 1Hz postural deflections, but loss of proprioceptive sensation only for the foot caused slow deflection (less than 1Hz). Since deflection velocity and area increased after loss of cutaneous sensation of the foot and the low frequency component (0.02-1Hz) of the deflection decreased, the present results partly support Rothwell's result. Therefore, these phenomena suggest that loss of a feed-back loop from the

cutaneous and proprioceptive sensation delayed posture control for deflection.

On the other hand, simple ischemia and hypoxia would less affect a static standing posture (CFP deflection). Although electromyograms and muscle force were not analyzed in this study, even if oxygen supply to muscle tissue is insufficient and there is a loss of muscle force, this will not affect a static standing posture if the proprioceptive sensation is intact.

In conclusion, although postural deflection may not be affected by the oxygen deficiency in the muscle tissues caused by the compression of blood vessels for 10 min, the cutaneous and proprioceptive sensation disturbance occurring following the condition may strongly affect postural control.

REFERENCES

Aaslid, R, Lingedaard, KF, Sorteberg, W and Normes, H (1989) Cerebral autoregurlation dynamics in humans. *Stroke*, **1**: 45-52.

Bringard, A, Perrey, S (2004) Influence of repeated isometric contractions on muscle deoxygenation and pulmonary oxygen uptake kinetics in humans. *Clin. Physiol. Funct. Imaging*, **24**: 229-236.

Castaldo, JE, Ochoa, JL (1984) Mechanical injury of peripheral nerves. Fine structure and dysfunction. *Clin. Plast. Surg.*, **11**: 9-16.

Demura, S, Yamaji, S, Noda, M, Kitabayashi, T and Nagasawa, Y (2001) Examination of parameters evaluating the center of foot pressure in static standing posture from the viewpoints of trial-to-trial reliability and interrelationships among parameters. *Equilib. Res.*, **60**: 44-55.

Demura, S, Uchiyama, M (2005a) Effect of traditional floor sitting on postural control after standing. *J. Hum. Ergol.*, **34**: 13-23.

Demura, S, Uchiyama, M (2005b) Effect of Japanese sitting style (seiza) on the center of foot pressure after standing. *J. Physiol. Anthropol. Appl. Human Sci.*, **24**: 167-73.

Ekdahl, C, Jarnlo, GB and Andersson, SI (1989) Standing balance in healthy subjects. Scandinavian *J. Rehabil. Med.*, **21**: 187-195.

Franks, HM, Hensley, VR, Hensley, WJ, Starmer, GA and Teo, RKC (1976) The relationship between alcohol dosage and performance decrement in human. *J. Stud. Alcohol*, **37**: 284-297.

Grigorova, V, Ivanov, I and Stambolieva, K (2001) Effect of sensory inputs alteration and central sensory disinteraction on postural sway and optokinetic reflex maintaining simultaneously body balance. *Acta Physiol. Pharmacol. Bulg.,* **26**: 177-180.

Iwanaga, K, Sakurai M, Minami, T and Kikuchi, Y (1993) Effect of restricted blood flow on intracellular pH threshold working muscle. *Ann. Physiol. Anthropol.*, **12**: 181-188.

Kimura, N, Katsumura, T, Hamaoka, T and Shimomitsu, T (1999) The estimation of oxygen availability in muscle during isometric exercise by near infrared spectroscopy. *J. Exer. Sport Physiol.*, **6**: 93-102.

Kitabayashi, T, Demura, S, Yamaji, S, Nakada, M, Noda, M and Imaoka, K (2002) Gender differences and relationships between physic and parameters evaluating the body center of pressure in static standing posture. *Equilib. Res.*, **61**: 56-62.

Kitabayashi, T, Demura, S and Noda, M (2003) Examination of the factor structure of center of foot pressure movement and cross-validity. *J. Physiol. Anthropol. Appl. Human Sci.*, **22**: 265-272.

Kopell, HP, Thompson, WA and Postel, AH (1962) Entrapment neuropathy of the ilioinguinal nerve. *N. Engl. J. Med.*, **266**: 16-19.

Kubo, T, Sakata, Y, Matsunaga, T, Koshimune, A, Sakai, S, Ameno, K and Ijiri, I (1989) Analysis of body-sway pattern after alcohol ingestion in human subjects. *Acta Oto. Laryngologica,* **468**: 247-252.

Laboratory of Physical Education, Tokyo Metropolitan University (2000) *New Physical fitness standards of Japanese people, 5th edition.* Fumaido, Tokyo: pp.98-101.

Ledin, T, Hafström, A, Fransson, PA and Magnusson, M (2003) Influence of neck proprioception on vibration-induced postural sway. *Acta Otolaryngol.*, **123**: 594-599.

Magnusson, M, Enbom, H, Johansson, R and Pyykkö, I (1990) Significance of pressor input from the human feet in anterior-posterior postural control. The effect of hypothermia on vibration-induced body-sway. *Acta Otolaryngol.*, **110**: 182-188.

Mangold, S, Laubli, T and Krueger, H (1996) Effects of a low alcohol dose on static balance, fine motor activity, and mental performance. *Neurotoxicol. Teratol.*, **18**: 547-54.

Markham, CH (1987) Vestibular control of muscular tone and posture. *Can. J. Neurol. Sci.*, **14**: 493-496.

Meyer, PF, Oddsson, LI, De Luca, CJ (2004) The role of plantar cutaneous sensation in unperturbed stance. *Exp. Brain Res.*, **156**: 505-512.

Mori, Y (1987) Upright setting and knee joint. *Orthop. Surg. Traumatol.*, **25**: 1912-1916.

Nieschalk, M, Ortmann, C, West, A, Schmal, F, Stoll, W and Fechner, G (1999) Effects of alcohol on body-sway patterns in human subjects. *Int. J. Legal. Med.*, **112**: 253-60.

Pollak, EW, Chavis, P and Wolfman, EF (1976) The effect of postural changes upon the ankle arterial perfusion pressure. *Vasc. Surg.*, **10**: 219-222.

Roebuck, TM, Simmons, RW, Mattson, SN and Riley, EP (1998) Prenatal exposure to alcohol affects the ability to maintain postural balance. *Alcohol Clin. Exp. Res.*, **22**: 252-258.

Rothwell, J (1994) Control of Human Voluntary Movement, Chapman & Hall, New York, pp 252-292.

Sakita, M, Takayanagi, K, Nakayama, A, Hanada, H and Kumagai, S (2006) The effect of cooling below the knees on dynamic standing postural control. *Rigakuryoho Kagaku*, **21**: 17-23.

Stål, F, Fransson, PA, Magnusson, M and Karlberg, M (2003) Effects of hypothermic anesthesia of the feet on vibration-induced body sway and adaptation. *J Vestib. Res.*, **13**: 39-52.

Sunderland, S (1990) The anatomy and physiology of nerve injury. *Muscle Nerve*, **13**: 771-784.

Traon, AP, Costes-Salon, MC, Galinier, M, Fourcade, J and Larrue, V (2002) Dynamics of cerebral blood flow autoregulation in hypertensive patients. *J. Neurol. Sci.*, **30**: 139-144.

Uimonen, S, Laitakari, K, Bloigu, R, Reinila, M and Sorri, M (1994) Static posturography and intravenous alcohol. *J. Vestib. Res.*, **4**: 277-283.

Vuillerme, N, Pinsault, N and Vaillant, J (2005) Postural control during quiet standing following cervical muscular fatigue: effects of changes in sensory inputs. *Neurosci. Lett.*, **378**: 135-139.

Wada, K, Sasaki, O (1990) The influence of the visual field on the static upright posture –The study on body sway, *Clin. Otolaryngol.*, **38**: 37-46.

Yamaji, S, Demura, S, Noda, M, Nagasawa, Y, Nakada, M and Kitabayashi, T (2001) The day-to-day reliability of parameters evaluating the body center of pressure in static standing posture. *Equilib. Res.*, **60**: 217-226.