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Gain modulation of the middle latency cutaneous reflex in patients with chronic joint instability after ankle sprain

Genki Futatsubashi [a], Shusaku Sasada [b], Toshiki Tazoe [c], Tomoyoshi Komiyama [a,d,*]

[a] The United Graduate School of Education, Tokyo Gakugei University, Tokyo, Japan
[b] Department of Developmental Physiology, National Institute for Physiological Sciences, Myodaiji, Okazaki, Aichi, Japan
[c] Department of Physical Medicine and Rehabilitation, Center for the Neural Basis of Cognition, University of Pittsburgh, Pittsburgh, PA, USA
[d] Department of Health and Sports Sciences, Chiba University, Chiba, Japan

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HIGHLIGHTS

- We investigated the modulation of cutaneous reflexes in the lower limb muscles of subjects with ankle sprain and joint instability.
- The cutaneous afferent pathway was modified in the subjects with chronic ankle instability after ankle sprain.
- These new findings partly shed light on the problem of impaired ankle joint regulation in patients with chronic ankle instability.

ABSTRACT

Objective: To investigate the neural alteration of reflex pathways arising from cutaneous afferents in patients with chronic ankle instability.
Methods: Cutaneous reflexes were elicited by applying non-noxious electrical stimulation to the sural nerve of subjects with chronic ankle instability ($n = 17$) and control subjects ($n = 17$) while sitting. Electromyographic (EMG) signals were recorded from each ankle and thigh muscle. The middle latency response (MLR; latency: 70–120 ms) component was analyzed.
Results: In the peroneus longus (PL) and vastus lateralis (VL) muscles, linear regression analyses between the magnitude of the inhibitory MLR and background EMG activity showed that, compared to the uninjured side and the control subjects, the gain of the suppressive MLR was increased in the injured side. This was also confirmed by the pooled data for both groups. The degree of MLR alteration was significantly correlated to that of chronic ankle instability in the PL.
Conclusions: The excitability of middle latency cutaneous reflexes in the PL and VL is modulated in subjects with chronic ankle instability.
Significance: Cutaneous reflexes may be potential tools to investigate the pathological state of the neural system that controls the lower limbs in subjects with chronic ankle instability.

1. Introduction

Ankle sprain is one of the most common injuries in sports and tends to recur frequently if not given adequate and continuous treatment. Many individuals with ankle sprain experience degradation in the ability to control the ankle joint. A 7-year follow-up study reported that 32% of patients with ankle sprain had chronic complaints of pain, swelling, or recurrent sprain (Konradsen et al., 2002). Previous studies have shown that neuromuscular deficits related to chronic ankle instability were represented by the alteration of prolonged peroneus longus (PL) reaction time in response to sudden inversion stress (Konradsen and Ravn, 1990; Lofvenberg et al., 1995), postural control deficits (McKeon and Hertel, 2008;

* Corresponding author. Address: Department of Health and Sports Sciences, Chiba University, 1-33 Yayoicho, Inage-ku, Chiba City 263-8522, Japan. Tel.:/fax: +81 43 290 2621.
E-mail address: komiyama@faculty.chiba-u.jp (T. Komiyama).

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Tropp and Odenrick, 1988), and proprioceptive deficits (Freeman, 1965; Freeman and Wyke, 1965; Sjolander et al., 2002). However, the mechanisms underlying ankle sprain-related neuromuscular deficits are less clear.

One of the possible explanations for neural deficits related to chronic ankle instability after an ankle sprain is the occurrence of neural alterations due to ligament injury. Ligament injury in conjunction with ankle sprain would lead to loss or change in sensory feedback from the ligaments and other joint structures (Johansson et al., 1991; Melnyk et al., 2007; Sjolander et al., 2002). In a recent study, Palmieri-Smith et al. (2009) showed that the neuromuscular activity of the peroneal muscles leads to functionally unstable ankles. However, it is less clear to what extent the supraspinal and transcortical neural systems are modulated after an ankle sprain. Konradsen et al. (1997) suggested that the reaction pattern to sudden inversion stress may be mediated by spinal or cortical centers.

Cutaneous reflexes, which can be elicited by stimulating the skin surface in the leg, are known to play an important role in responding to obstacles during locomotion (Drew and Rossignol, 1987; Forssberg, 1979). In addition, cutaneous reflexes are an integral part of locomotor regulation, allowing the human gait to manage external perturbations (Dietz et al., 1987; Duysens et al., 1990, 1992, 1993; Zehr et al., 1997, 1998). Furthermore, Hundza and Zehr (2007) recently showed that modulation of the cutaneous reflex may be involved in changes to sensory afferents caused by shoulder instability. Therefore, it is crucial to understand how an ankle sprain affects the excitability of the cutaneous reflex pathways that impinge on the motoneurons acting around the ankle joint.

Zehr and Stein (1999) suggested that cutaneous reflexes may aid in preventing an ankle sprain if excessive inversion occurred and that these responses may occur in concert with reflexes arising from muscle afferents in the peroneal muscles (e.g., Konradsen et al., 1997). In addition, Duysens and Levin (2010) suggested that cutaneous reflexes after the stimulation of the sural nerve may be a way of testing for abnormality in subjects with ankle sprain. However, to date, the extent to which ankle sprains affect the neural control of lower limb muscles has not been substantiated.

We hypothesized that the cutaneous reflexes in the lower limb muscles show a plastic change in individuals with an ankle sprain, which would be tightly linked to chronic ankle instability. Therefore, we investigated the extent to which the cutaneous reflexes in the lower limb muscles are modified in subjects with chronic ankle instability attributable to an ankle sprain.

2. Methods

2.1. Subjects

Seventeen competition-level athletes (15 men and 2 women), who had sustained a unilateral chronic ankle sprain (incipient diagnosis was confined to grade I or II), participated in this study (age: 22.6 ± 2.9 years; height: 172.2 ± 6.9 cm; weight: 68.5 ± 10.0 kg). All the subjects claimed joint instability (11 right and 6 left) and fulfilled the following three criteria: (1) history of a minimum of two inversion sprains in one ankle; (2) no history of other fractures or neuromuscular deficiencies in the injured side; and a (3) history of "giving way" symptoms (feeling of wobble and abrupt unintended crumbling of the ankle joint during walking) and functional disability of the ankle joint during sports activities. The functional disability and symptoms were evaluated using Karlsson's scale in which the subjects reported <80 points (Fair or Poor) for the total score (Karlsson's score) and ⩽15 points (1–2 per month during exercise) for the Instability Symptom item (Karlsson and Peterson, 1991). For all the participating subjects, at least 1 month had passed since their most recent episode of ankle sprain. In addition, the pain with palpation in the anterior talofibular ligament was also evaluated using a visual analog scale (VAS; 0–100 mm). The right leg was the dominant leg in all the subjects. The subjects' characteristics and clinical and electrophysiological features are shown in Tables 1 and 2.

The control group consisted of 17 age-matched competition-level athletes (15 men and 2 women: soccer 2, rugby 1, basketball 2, badminton 1, lacrosse 1, baseball 4, volleyball 1, track & field 4, tennis 1; years of competition, 10.7 ± 2.2 years) without an ankle sprain or any other neurological deficits (age: 21.7 ± 2.3 years; height: 171.2 ± 6.9 cm; weight: 65.3 ± 5.0 kg; Karlsson's score on both sides: 100 points, Excellent). The right leg was also the dominant leg in all the control subjects.

The experimental procedure was approved by the Local Ethics Committee of the Faculty of Education, Chiba University. All the subjects gave their informed, written consent before the experiments, in accordance with the Declaration of Helsinki.

2.2. Electromyographic recordings

Surface electromyographic (EMG) signals were obtained from the tibialis anterior (TA), medial gastrocnemius (MG), PL, and VL muscles of the injured and uninjured sides by using bipolar Ag/

Table 1
Characteristics and clinical and electrophysiological features of the patients with chronic ankle sprain.

Injury subject	Gender	Age	Sport	Years of competition	Affected side	Karlsson score	PL MLR (reflex gain)		PT	
							Injured	Uninjured	Injured	Uninjured
1	Male	20	Rugby	10	Left	44	−0.622	−0.165	10.2	8.3
2	Male	22	Basketball	12	Left	52	−0.675	−0.525	9.4	7
3	Male	23	Rugby	10	Left	54	−0.843	−0.415	10.1	9.5
4	Male	20	Rugby	8	Right	57	−0.785	−0.522	14	13.3
5	Male	25	Ski	15	Right	62	−0.500	−0.322	7.7	8.2
6	Male	26	Soccer	14	Right	62	−0.663	−0.396	10.1	11.6
7	Male	21	Lacrosse	9	Left	67	−0.843	−0.535	11.7	11.4
8	Male	22	Soccer	12	Right	69	−0.580	−0.559	9.8	10.1
9	Male	25	Soccer	15	Left	69	−0.701	−0.544	9.3	9.6
10	Male	30	Rugby	14	Left	72	−0.601	−0.490	11.3	10.3
11	Male	20	Volleyball	8	Right	72	−0.626	−0.263	12.5	12.1
12	Female	20	Basketball	9	Right	74	−0.544	−0.420	10.4	10.9
13	Female	24	Basketball	12	Right	74	−0.465	−0.306	10.3	10.5
14	Male	21	Track & field	9	Right	75	−0.529	−0.612	11.2	10
15	Male	25	Soccer	13	Right	77	−0.696	−0.771	10	9.5
16	Male	19	Basketball	8	Right	77	−0.802	−0.600	11.7	11.5
17	Male	21	Soccer	11	Right	80	−0.505	−0.441	13	12.7