

Neuromuscular Control and Performance during Sudden Acceleration in Subjects with and without Unilateral Acute Ankle Sprains

M. Qorbani

Abstract—Neuromuscular control of posture as understood through studies of responses to mechanical sudden acceleration automatically has been previously demonstrated in individuals with chronic ankle instability (CAI), but the presence of acute condition has not been previously explored specially in a sudden acceleration. The aim of this study was to determine neuromuscular control pattern in those with and without unilateral acute ankle sprains. Design: Case - control. Setting: University research laboratory. The sinker-card protocol with surface translation was used as a sudden acceleration protocol with study of EMG upon 4 posture stabilizer muscles in two sides of the body in response to sudden acceleration in forward and backward directions. 20 young adult women in two groups (10 LAS; 23.9 ± 2.03 yrs and 10 normal; 26.4 ± 3.2 yrs). The data of EMG were assessed by using multivariate test and one-way repeated measures $2 \times 2 \times 4$ ANOVA ($P < 0.05$). The results showed a significant muscle by direction interaction. Higher TA activity of left and right side in LAS group than normal group in forward direction significantly be showed. Higher MGR activity in normal group than LAS group in backward direction significantly showed. These findings suggest that compared two sides of the body in two directions for 4 muscles EMG activities between and within group for neuromuscular control of posture in avoiding fall. EMG activations of two sides of the body in lateral ankle sprain (LAS) patients were symmetric significantly. Acute ankle instability following once ankle sprains caused to coordinated temporal spatial patterns and strategy selection.

Keywords—Neuromuscular response, sEMG, Lateral Ankle Sprain, posture.

I. INTRODUCTION

ANKLE sprains are the most common acute injuries treated in general practitioners' clinics, emergency departments of public hospitals and in physiotherapy clinics [1], [2] and caused to constraints in their postural control system. Once an individual has suffered an ankle sprain, he or she is susceptible to recurrent sprains [3], [4].

Acute sprains may result in permanent disruption of the mechanoreceptors in the injured ligaments, [5] and these changes may lead to neuromuscular alterations in the lower limb. According evidence chronic ankle instability is associated with neuromuscular changes at the involved ankle, as well as proximally in the ipsilateral limb [6], [7] and in the contralateral limb [8]. Freeman et al [9]-[13] suggested that alterations in postural control could be attributed to deficits in

the afferent input arising from mechanoreceptors residing in the ankle ligaments and capsule (articular deafferentation). In addition to providing a basis for explaining the source of chronic ankle instability (CAI) as a subsequent effect of the recurrence of lateral ankle sprains (LAS), articular deafferentation has also been expanded to explain the source of chronic instability existing at other joints [14], [15]. Balance performance requires the interaction of the nervous and musculoskeletal systems. The role of the nerves system is to detect and predict instability and produce the appropriate muscle forces that will complement and coordinate with all the other forces acting on the body so that the COM is well controlled and balance is maintained. Balance deficits have been implicated as contributing to poor balance performance following lower limb musculoskeletal injury [16].

According to evidence demonstrates proximal neuromuscular changes in those with ankle instability. Such changes have been identified in both the ipsilateral and contralateral limbs of individuals with unilateral ankle injuries. Proximal changes ipsilaterally after acute ankle sprain have been shown in altered electromyographic and strength measures of the hip extensors [17] Contralateral changes associated with acute ankle sprain have been illustrated via altered activation patterns of the hip extensors [17] and impaired postural control in the limb contralateral to the acutely sprained ankle [18]. Two or more discrete strategy in control of posture exist that could either be used separately or be combined by the nervous system to produce adaptable control of the horizontal position of the center of mass (COM) in the sagittal plane [19]. The ankle strategy repositioned the COM by moving the whole body as a single-segment inverted pendulum by production of torque at the ankle. The hip strategy, in contrast, moved the body as a double-segment inverted pendulum with counter phase motion at the ankle and hip. They further suggested that hip strategy should be observed in situations that limit the effectiveness of ankle torque at producing whole-body motion (e.g. compliant or shortened support surfaces). The experimental observations that followed were consistent with this hypothesis, showing that ankle strategy was used to respond to translations during stance on a flat support surface; while hip strategy was observed during responses to backward translations during stance on a narrow (10 cm) beam [20]. A strategy selection is best described by what the central nervous system (CNS) is attempting to control.

M. Qorbani has an MS of Sport Biomechanics, Young Researchers and Elite Club, Boroujerd Branch, Islamic Azad University, Boroujerd, Iran (e-mail: mehrqorbani1@gmail.com). Cell number: +98 9183702639.

The goal of this study was to determine neuromuscular control in those with and without unilateral acute ankle sprains. Our hypothesis was that, in comparison with healthy subjects, and subjects with unilateral acute ankle sprains would exhibit different muscle synergy patterns and then different balance performance in sudden acceleration in a sudden acceleration protocol.

II. MATERIAL AND METHOD

20 young adult women in two groups (10 LAS; age = 23.9 ± 2.03 yrs; height = 170.3 ± 6 cm; mass = 61.6 ± 11.6 kg) with once history of unilateral ankle sprain in 3 weak to 3 month after primary treatment were participated in the experimental protocol and (10 normal; age = 26.4 ± 3.2 yrs; height = 168.5 ± 5.2 cm; mass = 59.4 ± 9.4 kg) with no significant history of lower extremity conditions, knee injury, or low back pain within the last year; no history of surgery in the lower extremity; and no neurologic deficits comprised the normal group. All subjects read and signed informed consent forms before initiation of testing. The informed consent form and protocol were approved by the Human Investigations Committee at our university. Subjects completed 2 ankle questionnaires, base of the Ankle Instability Instrument (AII) and the Foot and Ankle Instability Disability index (FADI), [21] including the FADI Sport Subscale, to determine inclusion criteria.

A. Instrument

We used a 16-channel EMG system (MA-300, Motion Lab System, D.T.U model) to record muscle activity. Ag/AgCl bipolar surface electrodes were placed on the middle portion of the respective muscles includes of tibialis anterior (TA), vastus medialis (VM), medialis gastrocnemius (MG), and erector spinae in (L3). The ground electrode was placed on the head of clavicle. The body mass in kilograms (kg) and the height in meters (m) of the participants were measured using a calibrated physician's scale.

A four-camera motion analysis system of Vicon T20s motion capture system (250 Hz, Vicon Motion Analysis Inc., Oxford, UK) with 2000 Hz of cut off frequency was used to obtain the three dimensional kinematics of the movement pattern of participants during the testing and to determine of starting up of the singer- card with the software of Nexus 1.8.2, Vicon, Oxford, UK.

B. Subject Preparation

The hair of skin over the tibialis anterior, gastrocnemius medialis, vastus medialis and erector spinae (L3) was shaved and stimulation sites cleaned with alcohol before electrode placement. Electrode placed basis of SENIM order [22]. To stimulate the muscles, were applied with a thin layer of conducting gel. Reflective anatomical and 16 tracking markers [23] (Plug-In Gait Marker Set, Vicon Peak, Oxford, UK) were pasted on both the legs of the subject during the testing. The anatomical markers placed on the following locations bilaterally: anterior, posterior iliac crests, greater trochanters, lateral and medial femoral epicondyles, lateral malleoli,

lateral, and post of heels, and the first metatarsal heads. A cluster of 2 tracking markers were also placed on both right and left sides of body. We start experiment trails after calibration.

C. Protocol

The singer-card complex was used for sudden acceleration protocol with surface displacement in this study. The reliability and validity of this test accepted of other resources [24], [25].the subject was secured in an upright stance position in the static card with waist and shoulder free in calibrated area. The feet were apart equal width of shoulder. The singer-Card complex once moved in forward and then backward direction apiece 3 trails under effect of the force produced shoot and hang of singers were equivalent 20% of subject's weight to product sudden acceleration and horizontal translation, without notice of subject as blinding situation.

D. Data and Statistical Analysis

Raw EMG data were filtered with notch filter of 60Hz band pass at 65–500 Hz using Datapac software (motion lab system), stored on a PC computer, and analyzed using biomechanical method of Visual C3D. The first of 100 ms of the beginning of card movement for normalization was considered because the initial responses to surface perturbations are called "automatic postural responses" because postural latencies (70-180 milliseconds) are much longer than stretch reflex latencies (40-30 milliseconds), but shorter than voluntary reaction times (180-250 milliseconds) [26]. The RMS of data was rectified with regulation of 180 ms of windows. The EMG data was normalized with peak dynamic method:

$$X_{norm} = X_{cure} - X_{min} / X_{max} - X_{min} \times 100$$

The films of video camera were considered to observe of control movement's pattern. After normalization EMG data it was assessed by spss 16 statistical software.

The between-subjects factor was group (LAS, normal), direction (forward, backward) and the within-subjects factor was side (involved, uninvolved as left and right). The data of EMG were assessed by using multivariate test and one-way repeated measures 2×2×4 (direction × side × muscle) ANOVA (P< 0.05).

III. RESULT

We observed that the LAS and normal groups were significantly different within group for TA, VM, MG and L3 in intensity of EMG activity during sudden acceleration; F=24.06, P=0.00 and between group; F=6.75, P=0.04. The LAS and normal groups were significantly different in EMG activity and muscle activity pattern in two directions; F=19.18, P=0.00 (Figs. 1 and 2). These figures illustrate significantly different in backward direction between two groups in MGR; F=7.32, P=0.015 and in TAL; F=6.47, P=0.021. In two groups were significantly different muscle activity pattern for four muscles; F=6.7, P=0.004. There is significantly different in

direction and function in two side of the body in two groups; $F=7, P = 0.017$. In two groups the difference of muscle activity in two sides was asymmetric; $F= 1.02, P = 0.41$.

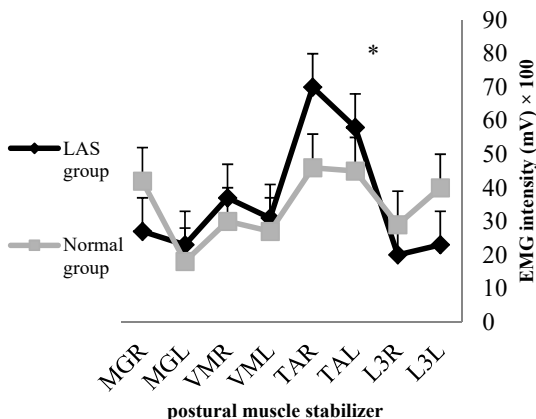


Fig. 1 The mean of EMG activation of posture muscle stabilizers; TA, VM, MG and L3 and standard error in two side of the body in forward direction. *The α level was set as $P < 0.05$

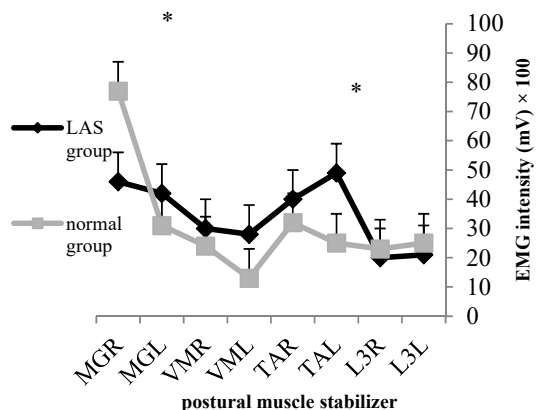


Fig. 2 The mean of EMG activation of posture muscle stabilizers; TA, VM, MG and L3 and standard error in two side of the body in backward direction. *The α level was set as $P < 0.05$

IV.DISCUSSION

We investigated the neuromuscular control and performance with the strategy selection of balance recovery in subjects with and without unilateral acute ankle sprains against fall, during sudden acceleration due to singer – card complex movement as a dynamic balance test. In accord with patterns of EMG activity of posture stabilizer muscles bilaterally to response of sudden acceleration of card movement in two groups in forward and backward direction we found two different strategy of balance recovery. Balance control system often includes inhibitory and compensatory movements that quickly and automatically occur after perturbation or instability momentum [27]. Therefore evaluation of dynamic balance control provides better perspectives of balance control strategies under influence of injuries and because of ankle strategy occurred in little

perturbations [28] so we expected occurrence of other strategies as; hip strategy, stepping strategy and suspensory strategy [28] due to high speed and sudden movement. These responses are termed "mixtures" against single strategy as ankle or hip strategies. It is proposed that such mixtures are the combinations of more than one elemental synergy and that timing and latency variations of the responses are due to segmental reciprocal delays, "a lower level segment' by segment interaction between individual muscle control" [29]. Loading and unloading segments were attended according to muscle pattern activation in two sides of two groups in two directions. Stepping strategy used for prevention against falling with straitening of base of support (BOS) and translation of COM as hip strategy (Horak et al 1997). Suspensory strategy is a situation that feet shaped as squat condition with descending COM (constant BOS) and without COM translation as ankle strategy [30], [31]. Higher EMG activation in TA and MG illustrated in comparison with VM and L3 (Figs. 1 and 2). Muscle EMG pattern of subjects compared with EMG activity in ankle strategy. In this strategy, the body swings as an inverse single segmental pendulum about the ankle. In the mechanism of response plantar flexors act to balance recovery and sequence recruitment is distal to proximal limbs [31]. As an example in forth sway when dorsiflexors contract simultaneously, the person's ability reduced to balance recovery. In another word with activation of an antagonist muscle it is necessary to produce an extra momentum via antagonist muscles [32]. This extra momentum produced extremely and quickly and used to maintenance of COM within the base of support [31]. Body sway about ankle joint complex in strategy of ankle in back sway attend with more EMG activity in; gastrocnemius, hamstrings, and spinae; and in forth sway attend with more EMG activity in tibialis anterior, quadriceps, and abdominal muscles [32]. Co-contraction of antagonist muscles illustrated in aging peoples [31]. The belief is that co-contraction strategy was be used as a method to production of stability in weaker joint [32]. In forward direction with comparison of two sides of the body in two groups (Fig. 1) EMG activation of TA, and VM muscles significantly in LAS group was be more than normal group ($P < 0.05$). On the other hand the EMG activation of L3 in left and right side adversely was more than in normal group similarly in hip strategy [30]. In backward direction with comparison of two sides of the body in two groups (Fig. 2) EMG activation of MGR muscle significantly in normal group was be more than LAS group ($P < 0.05$). TA and MG muscles had the most EMG intensity and the least latency respect to other muscles in two groups. According to muscle responses the most simultaneity was in TA and VM in LAS group and MG and L3 in normal group. In normal group all muscles had symmetric function in two sides except in MG (significantly) due to loading and unloading limbs and in LAS group there was symmetry in all of the muscles of two sides. So we found that occurring stepping strategy in normal group and suspensory strategy in LAS group in two directions.

As a clinical implication; changes in our basic understanding of how the CNS controls postural stability have useful implications for physical therapy practice design.

Further research is needed to assess the functional consequences of such deficits. Lastly, the potential limitation of the assessment of motoneuron pool excitability in selection of balance strategy recovery.

In conclusion, we demonstrated different muscle synergy patterns and then different dynamic performance in sudden acceleration in those with unilateral acute LAS.

REFERENCES

- [1] Lynch, S. A., & Renstrom, P. (1999). Treatment of acute lateral ankle ligament ruptures in the athlete- Conservative versus surgical treatment. *Sports Medicine*, 27(1), 61-71.
- [2] Garrick JG, Requa RK. The epidemiology of foot and ankle injuries in sports. *Clin Sports Med*. 1988; 7:29-36.
- [3] Hertel J. Functional anatomy, pathomechanics, and pathophysiology of lateral ankle instability. *J Athl Train*. 2002; 37:364-375.
- [4] McKay GD, Goldie PA, Payne WR, Oakes BW. Ankle injuries in basketball: injury rate and risk factors. *Br J Sports Med*. 2001; 35:103-108.
- [5] Freeman MA, Dean MR, Hanham IW. The etiology of functional instability of the foot. *J Bone Joint Surg Br*. 1965; 47:678-685.
- [6] Gribble PA, Hertel J, Denegar CR, Buckley WE. The effects of fatigue and chronic ankle instability on dynamic postural control. *J Athl Train*. 2004; 39:321-329.
- [7] Caulfield BM, Garrett M. Functional instability of the ankle: differences in patterns of ankle and knee movement prior to and post landing in a single leg jump. *Int J Sports Med*. 2002; 23:64-68.
- [8] Hertel J, Olmsted-Kramer LC. Deficits in time-to-boundary measures of postural control with chronic ankle instability. *Gait Posture*. 2007; 25:33-39.
- [9] Freeman MAR. Instability of the foot after injuries to the lateral ligament of the ankle. *J Bone Joint Surg Br*. 1965; 47:669-677.
- [10] Freeman MAR, Wyke B. Articular contributions to limb muscle reflexes: the effects of partial neurectomy of the knee joint on postural reflexes. *Br J Surg*. 1966; 53:61-68.
- [11] Freeman MAR, Wyke B. The innervation of the ankle joint: an anatomical and histological study in the cat. *Acta Anat (Basel)*. 1967; 68:321-333.
- [12] Freeman MAR, Wyke BD. Articular reflexes at the ankle joint: an electromyographic study of normal and abnormal influences of ankle-joint mechanoreceptors upon reflex activity in the leg muscles. *Br J Surg*. 1967; 54:990-1001.
- [13] Lephart SM, Warner JP, Borsa PA, Fu FH. Proprioception of the shoulder joint in healthy, unstable and surgically repaired shoulders. *J Shoulder Elbow Surg*. 1994; 3:371-380.
- [14] Lephart SM, Kocher MS, Fu FH. Proprioception following ACL reconstruction. *J Sport Rehabil*. 1992; 1:186-196.
- [15] Lentell, R. 1990. The relationship between muscle function and ankle stability. *Journal of Orthopedic Sports and Physical Therapy* 11: 605-611.
- [16] Bullock-Saxton JE, Janda V, Bullock MI. The influence of ankle sprain injury on muscle activation during hip extension. *Int J Sports Med*. 1994; 15:330-334.
- [17] Evans T, Hertel J, Sebastianelli W. Bilateral deficits in postural control following lateral ankle sprain. *Foot Ankle Int*. 2004; 25:833-839.
- [18] Horak F, Nashner L. Central programming of postural movements: adaptation to altered support-surface configurations. *J Neurophysiol* 1986; 55(6):1369-81.
- [19] Docherty CL, Gansneder BM, Arnold BL, Hurwitz SR. Development and reliability of the Ankle Instability Instrument. *J Athl Train*. 2006; 41:154-158.
- [20] Retrieved from www.seniam.org.
- [21] Retrieved from www.Vicon.com.
- [22] Mansfield A, Peters AL, Liu BA, Maki BE. A perturbation-based balance training program for older adults: study protocol for a randomized controlled trial. *BMC Geriatr*. 2007; 7: 12.
- [23] Mansfield A, Peters AL, Liu BA, Maki BE. Effect of a Perturbation-Based Balance Training Program on Compensatory Stepping and Grasping Reactions in Older Adults: A Randomized Controlled Trial. *PHYS THER*. 2010; 90: 476-491.
- [24] Nashner LM, Cordo PJ. Relation of automatic postural responses and reaction-time voluntary movements of human leg muscles. *Exp Brain tLT*. 1981; 43: 395-405.
- [25] Plisky PJ, Rauh MJ, Kaminski TW, Underwood FB. Star Excursion Balance Test as a predictor of lower extremity injury in high school basketball players. *J Orthop Sports Phys Ther*. 2006; 36(12): 911-919.
- [26] Hwang S. The Balance Recovery Mechanisms Against Unexpected Forward Perturbation. *Annals of Biomedical Engineering*. 2009; 37: 8: 1629-1637.
- [27] McCollum G, Horak FB, Nashner LM. Parsimony in neural calculations for postural movement. In: *Bloedel J, Dichgans J, Precht Worlds. Springer-Verlag*; 1984:52-66.
- [28] Horak FB, Henry SM, Shumway-Cook A. Postural Perturbations: New Insights for Treatment of Balance Disorders. *PHYS THER*. 1997; 77:517-533.
- [29] Nashner L.M, G McCollum. The Organization of human postural movements: A formal basis and Experimental Synthesis. *The behavioral and brain sciences*. 1985.8, 135-172.
- [30] Macaluso A, Nimmo M, Foster J, Cockburn M, McMillan N, Vito G. Contractile Muscle Volume and Agonist/Antagonist Coactivation Account for Differences in Torque between Young and Older Women. *Muscle Nerve*. 2002; 25: 858-863.
- [31] Okada S, Hirakawa K, Takada Y, Kinoshita H. Age-related differences in postural control in humans in response to sudden deceleration generated by postural disturbance. *Eur J Appl. Physiol*. 2001; 18: 10-18.
- [32] Pijnappels M, Bobbert MF, van Dieen JH. EMG modulation in anticipation of a possible trip during walking in young and older adults. *J of Electromyography and Kinesiol* 16 .2006: 137-143.

Mehrangiz Qorbani was born in Iran (Bijar) in 1986. She became a member of World Academy of Science, Engineering and Technology in 2014. She earned BS of physical education from Al-Zahra University (Tehran) in 2009 and MS of sport biomechanics in Azad University (Boroujerd) Aug 2013. She is PhD candidate in Virginia University (USA) now.

Mehr is a researcher in sport biomechanics and had more articles in "balance control system in patients with acute lateral ankle sprains".

Ms Qorbani became member of Young Researchers and Elite Club, Islamic Azad University from September 2011 until now.