UDC 612.743+612.884

SH. SHENOY,[1] H. BALACHANDER, [1] and J. S. SANDHU[1]

EFFECT OF PRIOR INSTRUCTIONS ON PREPROGRAMMED REACTIONS OF TRUNK MUSCLES IN INDIVIDUALS WITH AND WITHOUT CHRONIC LOW BACK PAIN

Received February 18, 2013.

The late component of the stretch reflex occurring in humans within a 40 to 120 msec interval following a loading perturbation is qualified as a preprogrammed muscle reaction (PPR). The PPR size can be significantly modulated with prior instructions. These modifications are significantly influenced by a number of factors, in particular by the presence of pain syndromes. The objective of our study was to compare the effect of prior instructions on the PPR amplitude in the trunk muscles in individuals with chronic low back pain (LBP) compared to healthy controls. LBP is a widespread syndrome, especially in athletes. Surface EMGs were recorded from superficial trunk muscles, *rectus abdominis* (RA) and *erector spinae* (ES), in athletes suffering from chronic LBP (n = 24) and asymptomatic (healthy) athletes (n = 25). Loading perturbations (induced by dropping a weight, application of 3 kg force, ≈ 30 N, to the outstretched hand from a 8 cm height) were introduced in standing at a known time with prior instructions to "*let go*" for the induced perturbation or to "*resist*" it. The root mean square (RMS) of the EMG amplitudes within the reaction duration were compared between the two groups. Statistically significant differences were obtained when the mean PPR EMG amplitudes were compared between the LBP and control groups for the above two task instructions; this was found for both examined muscles, RA and ES ($P < 0.05$). Therefore, individuals with chronic LBP exhibit poorly modulated PPR amplitudes according to prior task instructions. Changes in the networks controlling automatically regulated movements and excitability of the spinal pathways could be responsible for this specificity.

Keywords: preprogrammed muscle reactions, chronic low back pain, prior instructions, electromyography, trunk muscles.

INTRODUCTION

The spinal stretch reflex in humans is well known to include at least two components, a classic short-latency response (M1), occurring with an about 40 msec delay, and a more complex long-latency response (M2), occurring with a delay of 40-50 to 120 msec [1], also known as a preprogrammed muscle reaction (PPR). Although the time characteristics of the latter vary from muscle to muscle, it occurs in both the shortened and stretched muscles in response to perturbation in their length or loading [2].

It was proposed that the origin of the long-latency M2 response involves transcortical projection pathways [3, 4]. This makes the understanding of the long-latency response more complex than that of a simple stretch reflex. The M2 has been said to be task-dependent [5-7] and affected by various factors such as velocity, duration of perturbation [8], and pain [9]. The long-latency reflex and voluntary muscle responses exhibit a high functional similarity, probably because of a shared neural substrate [10, 11]. This explains its coordination between feedback gain and internal models during complex motor tasks [9]. Long-latency muscle responses function to counteract perturbations, to correct the direction of movement, and to regain stability [11, 12].

Modification in the size of M2 related to prior instructions has been observed [1, 13, 14]. When a command "*let go*" is given, so as not to "*resist*" the

[1] Faculty of Sports Medicine and Physiotherapy, Guru Nanak Dev University, Punjab, India
Correspondence should be addressed to:
Sh. Shenoy (e-mail: shwet1999@yahoo.com).
H. Balachander (e-mail: harini.bee@gmail.com).
J. S. Sandhu (e-mail: jssandhudr@gmail.com).

perturbation, the individual effectively suppresses realization of the long-latency response and decreases its amplitude [15]. However, this aspect is still under debate. Rothwell [16] and Capaday [17] concluded that the nature of the voluntary movement, and not of the reflex response, is influenced by prior instructions.

Chronic low back pain (LBP) is a common problem faced by vast human populations. Here, we do not discuss in detail questions on the etiology of this syndrome, as they are out of the topic of our communication. About 30% of athletes suffer from chronic LBP due to the repeated flexion and hyperextension demands sports game [18, 19]. Abnormal functioning of the motorneuronal loop causes reduced or absent firing of the trunk muscles [20, 21] and a delayed muscle reflex response [5] to trunk loading in individuals with chronic LBP.

Healthy individuals depend largely on neuromotor responses to maintain stability in dynamic loading situations, as no change in muscle recruitment is observed with anticipation [22]. Altered postural and neuromotor control in individuals with chronic LBP could blunt their stability and predispose them to recurrent injury.

Although the long-latency response of muscles of the hand largely involves the transcortical loop, postural muscles are often integrated with automated motor programs and mostly mediated by other spinal pathways [23]. To our knowledge, no study has been done to examine the effect of prior instructions on the long-latency response of trunk muscles. If the subject's intent could alter the long-latency response, it could prove beneficial in the rehabilitation of individuals with chronic LBP.

Hence, the purpose of our study was to examine the effect of a prior instruction on the long-latency reflex response of the trunk muscles (*rectus abdominis*, RA, and *erector spinae*, ES) during sudden trunk loading tasks in individuals with and without chronic LBP.

METHODS

Subjects. Athletes with nonspecific LBP (n = 24, 16 men and 8 women) were selected across various sporting bodies in and around Amritsar, Punjab, India. Athletes with chronic LBP having nonradiating pain for at least three months were included in the study. The presence of the mentioned syndrome was well documented by medical examination. The athletes were currently involved in the sport and had been playing for at least five days a week for the last three years. Cases with recent history of traumatic injury to back and lower limbs, history of CNS impairments, abdominal or back surgery, and recent history of systemic illnesses were excluded.

The control group consisted of athletes at a similar sporting level, with no history of LBP or injury (n = 25, 17 men and 8 women).

EMG Recording. Surface EMG data were collected using a Noraxon-MyoS 1200 set manufactured by Noraxon (USA). A Logitech Webcam videocamera was used in conjunction. Electromyograms were recorded from the superficial trunk muscles, *m. rectus abdominis* (RA) and *m. erector spinae* (ES), using bipolar disposable surface electrodes. Prior to the placement of the electrodes, the area was rubbed clean using an alcohol swab. For the RA, recording electrodes were placed parallel to the muscle, 3 cm apart and about 2 cm lateral to the umbilicus. For the ES, the electrodes were placed parallel to the spine, at the level of L3 and L4, 2 cm apart, over the muscle mass [24]. A reference electrode was placed on the lateral aspect of the trunk.

The electromyograph machine was set at a continuous recording mode, with sampling frequency 10^3 sec^{-1}, sensitivity 100 μV/div., filter setting 20 Hz–3 kHz, and sweep rate 50 msec/div [21].

Testing Procedure. A detailed history of sporting activity and assessment of LBP was taken before testing. Subjects filled the Visual Analogue Scale and Roland Morris Disability Questionnaire to assess the severity of pain. Individuals who did not fit the criteria were immediately excluded from the study.

The subjects were made to stand with the pelvis immobilized by support posteriorly and strapping anteriorly, to restrain any unnecessary movement [25, 26]. A cushioned weight of 3 kg force (about 30 N) was made to drop from predetermined height of 8 cm onto the subject's outstretched hand. The muscle reaction to this sudden anterior movement at the spine was recorded.

The subjects were informed of the testing procedure. The weight used for the trials for perturbation was adopted from a previous study by Ramprasad [21]. A prior command of "*let go*" or "*resist*" for the trunk muscles was explained and taught to the subject. The subject was made to stand relaxed with his/her arms outstretched. The exact time of drop was indicated to the subject by a metronome, and the EMG was recorded. Three trials were taken for each task, and a 10-min-long time interval between trials was given to

recuperate.

EMG Data Processing. For both muscle groups, the data computed were the mean window length of M2-M3 and the mean RMS amplitude of the M2 response.

To determine the RMS amplitude and window length, markers were introduced at the onset of M2, where the response size was 60% higher than M1 [27], and polyphasic M2-M3 waves were present within a 40 to 120 msec window. Raw EMGs were rectified and smoothed before analyzing. The mean RMS values for all three tasks and for RA and ES EMGs were calculated for both LBP and control groups.

Statistical Analysis. IBM SPSS Statistics (Version 20.0) was used for the analysis. The significance level for intergroup comparisons was set at 0.05. A general linear model and multivariate analysis were used to compare the mean PPR amplitudes between the LBA and control groups.

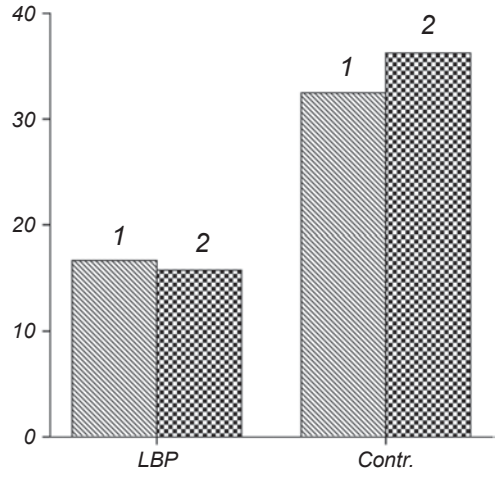
RESULTS

The mean RMSs of the EMG amplitudes for two muscles, RA and ES, were calculated. For the "*let go*" command, the mean RMS of the RA EMG amplitude was 16.68 ± 3.4 for the LBA group and 31.77 ± 11.77 for the control group. For the command "*resist,*" the mean values were 15.78 ± 3.6 and 35.38 ± 15.08 for the LBP and control groups, respectively.

For the "*let go*" task, the mean RMS of the ES amplitude was 59.40 ± 15.80 for the LBP group and 61.46 ± 14.42 for the control group. A mean value of 30.82 ± 13.26 was obtained for the "*resist*" command in the LBP subjects, while it was 64.75 ± 10.16 for the control group.

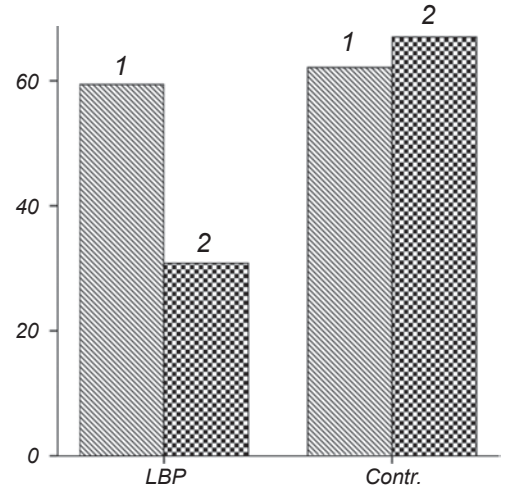
Statistical analysis was done using a general linear model and multivariate analysis. Further multiple comparison by the *post-hoc* Tukey test was done to compare the mean difference between groups. The results showed the existence of a statistically significant difference between LBP and control groups for both commands in the RA muscle group. A mean difference of 15.76 (P = 0.05*) was found between the "*let go*" amplitude in the LBP and control groups, while a mean difference of 20.46 (P = 0.05*) was found between the data related to the "*resist*" command in the LBP and control groups for the RA muscle group (Table 2). In the ES, no significant difference was obtained for the command "*let go*," while "*resist*" showed a significant mean difference of 38.18 (P = 0.05*) between the LBA and control groups.

Intragroup comparisons for both commands resulted in no significant difference except in the ES case (graphs 1 and 2). In the LBA group, the mean difference of EMG amplitudes between the commands "*let go*" and "*resist*" was statistically significant with a mean difference of 28.58 (P = 0.05*). All these values were significant at $P < 0.005$.



F i g. 1. Mean values of the RMS of the PPR amplitude of the *restus abdominis* muscles at instructions of "*let go*" (*1*) and "*resist*" (*2*) in the LBP and control groups.

Р и с. 1. Середні значення RMS амплітуди препрограмованих реакцій *m. rectus abdominis* при інструкціях «не опиратися» (*1*) та «опиратися» (*2*) пертурбації в досліджених групах.



F i g. 2. Mean values of the RMS of the PPR amplitude of the *erector spinae* muscles at instructions of "*let go*" (*1*) and "*resist*" (*2*) in the LBP and control groups.

Р и с. 2. Середні значення RMS амплітуди препрограмованих реакцій *m. erector spinae* при інструкціях «не опиратися» (*1*) та «опиратися» (*2*) пертурбації в досліджених групах.

Table 1. Anthropometric data and health state of individuals of the LBA and control groups.

Таблиця 1. Антропометричні дані та стан здоров'я в осіб досліджених груп.

Characteristics	Groups	
	LBA (n = 24)	Control (n = 25)
Age, years	24.26 ± 4.7	25.13 ± 5.05
Height, cm	174.42 ± 11.36	172.23 ± 9.63
Body mass, kg	69.76 ± 8.43	67.32 ± 7.84
VAS, cm	3.24 ± 1.62	0.0
Lifetime highest VAS	7.2 ± 2.19	0.0
RMDQ	4.20 ± 3.18	0.0
Activity reduction, %	8.96 ± 2.53	0.0

Footnotes: means ± s.d. are shown, VAS is Visual Analogue Scale, and RMDQ is Roland Morris Disability Questionnaire

Table 2. Results of *post hoc* Tukey testing of the variables in the LBA and control groups.

Таблиця 2. Результати *post-hoc*-тестування експериментальних даних, за Тьюкі, у двох досліджених групах.

Results	mean difference	s.d.	significance (p)
EMGs recorded from the *m. rectus abdominis*			
LBA 'Let Go' x LBA 'Resist'	.9040	2.72	0.987
Control 'Let Go' x Control 'Resist'	3.76	2.72	0.516
LBA 'Let Go' x Control 'Let Go'	5.76	2.72	0.05*
LBA 'Resist' x Control 'Resist'	20.46	2.72	0.05*
EMGs recorded from the *m. erector spinae*			
LBA 'Let Go' x LBA 'Resist'	28.58	5.11	0.05*
Control 'Let Go' x Control 'Resist'	4.88	5.11	0.775
LBA 'Let Go' x Control 'Let Go'	2.72	5.11	0.951
LBA 'Resist' x Control 'Resist'	36.18	5.11	0.05*

Footnote: * Significant at $P<0.005$

DISCUSSION

The purpose of our study was to determine the effect of prior instructions on the PPR amplitudes following trunk loading perturbations in individuals with and without chronic LBP. The mean PPR amplitudes of RA and ES EMGs with prior instruction to "*let go*" and "*resist*" were compared. For the RA, the mean amplitude for "*let go*" was approximately 48% lower in the LBP group compared to the control, and it was 57% lower for the "*resist*" command. For the ES muscle, no difference was found under "*let go*" conditions, but "*resist*" showed the 53% lower PPR EMG amplitude in LBP subjects when compared to controls. A previous study [21] demonstrated a lower PPR EMG amplitude in individuals with chronic LBP compared to controls. However, the results of our study show that individuals with chronic LBP poorly modulate PPR EMG responses upon prior instructions. Significantly lower EMG amplitudes, especially in the ES, were obtained in individuals with chronic LBP who intended to "*resist*" the induced perturbation when compared to an intent to "*let go.*"

Prior-task instructions exert noticeable effects on the PPR. Effective suppression of the M2 response upon intent to "*let go*" has been observed in several

previous studies [1, 13, 28]. This makes the PPR relate very closely to voluntary responses [9, 12], and the primary motor cortex modulates the PPR just like these responses [29, 30]. But this has been a topic of debate ever since, as several studies have shown no significant difference in the PPR amplitude following prior instructions [16, 17], especially in small muscles of the hand (such as *m. flexor pollicis longus*).

Postural muscles (in our case, the RA and ES) differ from the distal limb muscles functionally, as they rely more on automatically regulated movements. Studies provide evidence that, unlike muscles of the hand whose functions are effectively controlled by the transcortical pathways, postural trunk muscles are to a greater extent influenced by the spinal (segmental) pathways [3, 4, 23, 31]. A higher PPR amplitude in the RA and ES muscles in our study in normal individuals with a prior instruction to *resist* the perturbation could be due to an enhanced cortico-spinal excitability [32, 33] that is likely to arise from an overlap of multiple neural responses [34].

In the case of individuals with chronic LBP, two differences are evident: (i) The RA exhibits a lower mean PPR amplitude for both commands, i.e., "*let go*" and "*resist,*" (ii) In the ES, individuals presented better modulation for the intent to "*let go*" than to "*resist*." The "*resist*"-related amplitude was significantly lower for these individuals (Table 2).

Individuals with chronic LBP exhibit a few noticeable neuromotor changes, in particular, in proprioception, a reduced firing rate of motor units, and poor muscle stabilization [20, 35, 36]. This results in lower and even zero PPR amplitudes [21]. However, the reduced ES PPR amplitude on the intent to "*resist*" can be determined by chronic pain. The site of pain origin located rather close to the ES could induce a lower cortico-spinal excitability [37]. As was discussed previously, the intent and its effect on the amplitude of the PPR can vary due to changes in the supraspinal (cortico-spinal) excitability. Hence, chronic pain reducing the excitability of this system in individuals with chronic LBP can be responsible for difficulties of intended modulation of PPR.

Fatigue can also be a reason for reduced cortico-spinal excitability [38, 39]. Individuals suffering from chronic LBP often experience abnormally intense fatigue of postural muscles and poor postural control provided by trunk muscles [40, 41]. Long-lasting disturbances in the neuromotor system can induce plastic (both structural and functional) changes in the cortical networks [42]. A possibility for changes in the representation of the trunk muscles in the motor cortex under the above conditions has been hypothesized [43].

Thus, mild modulation of the PPR amplitude upon receiving prior instructions is obvious, especially in the ES, in individuals with chronic LBP. These changes could predispose LBP individuals to long-term manifestation of this syndrome. At the same time, the dynamic nature of the respective neural pathways points to an effective neuromotor exercise program that can help earlier recovery in these cases. The cause of changes in the PPR amplitude upon intent and perturbation should be qualified as multifactorial. It is likely that training-related modulation of cortico-spinal activation is one of the ways to improve the state of health of the respective contingent. Only few data are available in literature to help interpret the above-described results, and further studies may be required to support these findings.

The interpretation of our findings is limited by the "cross-sectional" design of the study. As was mentioned above, we did not elucidate precise medical reasons for the LBP occurrence. The way of stimulation (a weight dropped to induce perturbations) was manual. Hence, some variations in the perturbation force cannot be ruled out, although the weight and the height of drop were standardized. The subjects were well matched and, irrespective of their sport specialization, showed similar EMG amplitudes to the action of perturbation in the chronic LBP group, despite the fact that they were chosen from various sports (soccer, hockey, handball, or basketball).

Therefore, poor modulation of the PPR amplitude related to the type of prior instructions is evident in individuals with chronic LBP. Subjects with this syndrome demonstrate significantly lower perturbation-related EMG amplitudes on the intent to "*resist*" than to "*let go,*" especially in the ES muscle. The reason for this may be multifactorial, but the effect of reduced cortico-spinal excitability on prior task instructions with respect to the RA and ES muscles in individuals with chronic LBP can be an important reason, determining changes in automatically regulated (patterned) movements and reduced trunk muscle function.

All participants gave their informed consent before participation, and the testing procedures were explained to them. The study was approved by the Ethics Committee, Faculty of Sports Medicine and Physiotherapy, Guru Nanak Dev University, Amritsar, India.

The authors, Sh. Shenoy, H. Balachander, and J. S. Sandhu, have no conflict of interests.

Ш. Шеной[1], Х. Балачандер[1], Дж. С. Сандху[1]

ВПЛИВ ПОПЕРЕДНІХ ІНСТРУКЦІЙ НА ПРЕПРОГРАМОВАНІ РЕАКЦІЇ М'ЯЗІВ ТУЛУБА В ОСІБ З ХРОНІЧНИМ БОЛЕМ У ПОПЕРЕКУ ТА БЕЗ ТАКОГО СИНДРОМУ

[1] Університет Гуру Нанак Дев, Амрітсар, Пенджаб (Індія)

Р е з ю м е

Пізній компонент стретч-рефлексу, що розвивається у людей в інтервалі 40–120 мс після пертурбації (навантаження), кваліфікується як м'язова препрограмована реакція (ППР). Величина ППР може істотно модулюватися під дією попередніх інструкцій. На ці модифікації істотно впливають численні фактори, зокрема наявність больових синдромів. Ціллю нашого дослідження було порівняння амплітуд ППР м'язів тулуба в осіб, що страждали на біль у попереку (БП), та здорових контрольних тестованих. БП є виключно широко розповсюдженим синдромом, особливо у спортсменів. Ми відводили ЕМГ від поверхневих м'язів тулуба – *rectus abdominis* (*RA*) та *erectror spinae* (*ES*) – у спортсменів із синдромом хронічного БП (n = 24) та спортсменів без такого розладу (здорових, n = 25). Силові пертурбації (прикладання ваги 3 кгс до витягнутої руки з висоти 8 см) вводились у певний момент до тестованого в положенні стоячи з попередніми інструкціями «не опиратися» введеному навантаженню або «опиратися» йому. Порівнювалися значення RMS для амплітуд ЕМГ у межах реакції, що спостерігалася в двох групах. Було виявлено, що середні амплітуди ППР-ЕМГ у групах БП та контролю при двох вказаних вище попередніх інструкціях вірогідно відрізнялися; це було властиве для обох обстежених м'язів – *RA* та *ES* ($P < 0.05$). Таким чином, особи, що страждають на хронічний БП, демонструють обмежену модуляцію амплітуди ППР відповідно до попередніх інструкцій. Зміни в нейронних мережах, що контролюють автоматично регульовані рухи та збудливість спінальних шляхів, вірогідно є відповідальними за таку специфіку.

REFERENCES

1. P. H. Hammond, "Involuntary activity in biceps following the sudden application of velocity to the abducted forearm," *J. Physiol.*, **127**, 23-25 (1995).
2. P. W. Matthews, "The human reflex and the motor cortex," *Trends Neurosci.*, **14**, 87-91(1991).
3. G. C. Phillips, "Motor apparatus of the baboon's hand," *Proc. Roy. Soc. Lond., Ser. B, Biol. Sci.*, **173**, No. 31, 141-174(1969).
4. P. B. C. Matthews, S. F. Farmer, and D. A. Ingram, "On the localization of the stretch reflex of intrinsic hand muscles in a patient with mirror movements," *J. Physiol.*, **428**, 561-577 (1990).
5. J. Cholewicki, S. P. Silfies, R. A. Shah, et al., "Delayed trunk muscle reflex responses increase the risk of low back injuries," *Spine*, **30**, No. 23, 2614-2620 (2005).
6. A. K. Datta, L. M. Harrison, and J. A. Stephens, "Task-dependent changes in the size of response to magnetic brain stimulation in human first dorsal interosseous muscle," *J. Physiol.*, **418**, No. 1, 13-23 (1989).
7. V. Dietz, M. Discher, and M. Trippel, "Task-dependent modulation of short-and long-latency electromyographic responses in upper limb muscles," *Electroencephalogr. Clin. Neurophysiol.*, **93**, No. 1, 49-56 (1994).
8. G. N. Lewis, E. J Perreault, and C. D. MacKinnon, "The influence of perturbation duration and velocity on the long-latency response to stretch in the biceps muscle," *Exp. Brain Res.*, **163**, 361-369 (2005).
9. J. A. Hides, W. R. Stanton, S. J. Wilson, et al., "Retraining motor control of abdominal muscles among elite cricketers with low back pain," *Scand. J. Med. Sci. Sports*, **20**, No. 6, 834-842 (2010).
10. I. L. Kurtzer, J. A. Pruszynski, and S. H. Scott, "Long-latency reflexes of the human arm reflect an internal model of limb dynamics," *Current. Biol.*, **18**, 449-453 (2008).
11. E. Todorov and M. I. Jordan, "Optimal feedback control as a theory of motor coordination," *Nat. Neurosci.*, **5**, 1226-1235 (2002).
12. C. C. Gielen, L. Ramaekers, and E. J. Van Zuylen, "Long-latency stretch reflexes as co-ordinated functional responses in man," *J. Physiol.*, **407**, 275-292 (1988).
13. J. G. Colebatch, S. C. Gandevia, D. I. McCloskey, and E. K. Potter, "Subject instruction and long-latency reflex responses to muscle stretch," *J. Physiol.*, **29**, 527-534 (1979).
14. C. K. Loo and D. I. McCloskey, "Effects of prior instruction and anaesthesia on long-latency responses to stretch in the long flexor of the human thumb," *J. Physiol.*, **365**, No. 1, 285-296 (1985).
15. M. L. Latash, "Preprogramed reactions," in: *Neurophysiological Basis of Movement*, Human Kinetics Publ., Champaign (2008), pp. 113-218.
16. J. C. Rothwell, M. M. Traub, and C. D. Marsden, "Influence of voluntary intent on the human long-latency stretch reflex," *Nature*, **286**, 496-498 (1980).
17. C. Capaday, R. Forget, and T. Milner, "A re-examination of the effects of instruction on the long-latency stretch reflex response of the flexor *pollicis longus* muscle," *Exp. Brain Res.*, **100**, No. 3, 515-521 (1994).
18. T. Dreisinger and B. Nelson, "Management of back pain in athletes," *Sports Med.*, **21**, No. 4, 313-320 (2006).
19. K. L. Newcomer, T. D. Jacobson, D. A. Gabriel, et al., "Muscle activation patterns in subjects with and without low back pain," *Arch. Phys. Med. Rehabil.*, **83**, 816-821 (2002).
20. M. Ramprasad, D. S. Shenoy, S. J. Singh, et al., "The magnitude of pre-programmed reaction dysfunction in back pain patients: Experimental pilot electromyography study," *J. Back Musculoskelet. Rehabil.*, **23**, 77-86 (2010).
21. K. P. Granata, K. F. Orishimo, and A. H. Sanford, "Trunk muscle coactivation in preparation for sudden load," *J. Electromyogr. Kinesiol.*, **11**, No. 4, 247-254 (2001).
22. A. F. Thilmann, M. Schwarz, R. Töpper, et al., "Different mechanisms underlie the long-latency stretch reflex response of active human muscle at different joints," *J. Physiol.*, **444**, No. 1, 631-643 (1991).

23. J. R. Cram and E. Criswell, "Static assessment and clinical protocol," in: *Introduction to Surface Electromyography*, Jones a Bartlett Publ. (2010), pp 105-113.
24. J. H. Skotte, N. Fallentin, M. T. Pedersen, et al., "Adaptation to sudden unexpected loading of the low back – the effects of repeated trials," *J. Biomech.*, **37**, 1483-1489 (2004).
25. M. T. Pedersen, M. B. Randers, J. H. Skotte, and P. Krustrup, "Recreational soccer can improve the reflex response to sudden trunk loading among untrained women," *J. Strength Cond. Res.*, **23**, No. 9, 2621-2626 (2009).
26. J. Noth, M. Schwarz, K. Podoll, and F. Motamedi, "Evidence that low-threshold muscle afferents evoke long-latency stretch reflexes in human hand muscles," *J. Neurophysiol.*, **65**, 1089-1097 (1991).
27. E. V. Evarts and R. Granit, "Relations of reflexes and intended movements," *Prog. Brain Res.*, **44**, 1-14 (1976).
28. E. V. Evarts and J. Tanji, "Reflex and intended responses in motor cortex pyramidal tract neurons of monkey," *J. Neurophysiol.*, **39**, 1069-1080 (1976).
29. A. J. Suminski, S. M. Rao, K. M. Mosier, and R. A. Scheidt, "Neural and electromyographic correlates of wrist posture control," *J. Neurophysiol.*, **97**, 1527-1545 (2007).
30. C. Capaday, R. Fraser, R. Forget, and Y. Lamarre, "Evidence for a transcortical stretch reflex from the study of patients with mirror movements," *Soc. Neurosci. Abstr.*, **15**, 74.13 (1989).
31. H. Morita, N. Petersen, L. O. D. Christensen, et al., "Sensitivity of H-reflexes and stretch reflexes to presynaptic inhibition in humans," *J. Neurophysiol.*, **80**, No. 2, 610-620 (1998).
32. G. Lewis, M. Polych, and W. Byblow, "Proposed cortical and sub-cortical contributions to the long-latency stretch reflex in the forearm," *Exp. Brain Res.*, **156**, No. 1, 72-79 (2004).
33. G. N. Lewis, C. D. MacKinnon, and E. J. Perreault, "The effect of task instruction on the excitability of spinal and supraspinal reflex pathways projecting to the biceps muscle," *Exp. Brain Res.*, **174**, No. 3, 413-425 (2006).
34. P. W. Hodges and B. H. Bui, "A comparison of computer-based methods for the determination of onset of muscle contraction using electromyography," *Electroencephalogr. Clin. Neurophysiol.*, **101**, 511-519 (1996).
35. J. V. Jacobs, S. M. Henry, S. L. Jones, et al., "A history of low back pain associates with altered electromyographic activation patterns in response to perturbations of standing balance," *J. Neurophysiol.*, **106**, 2506-2514 (2011).
36. P. H. Strutton, S. Theodorou, M. Catley, et al., "Cortico-spinal excitability in patients with chronic low back pain," *J. Spinal Disord. Tech.*, **18**, No. 5, 420-424 (2005).
37. J. L. Taylor, J. E. Butler, G. M. Allen, and S. C. Gandevia, "Changes in motor cortical excitability during human muscle fatigue," *J. Neurophysiol.*, **490**, Part 2, 519-528 (1996).
38. J. B. Pitcher and T. S. Miles, "Alterations in cortico-spinal excitability with imposed vs. voluntary fatigue in human hand muscles," *J. Appl. Physiol.*, **92**, No. 5, 2131-2138 (2002).
39. A. Radebold, J. Cholewicki, G. K. Polzhofer, and H. S. Greene, "Impaired postural control of the lumbar spine is associated with delayed muscle response times in patients with chronic idiopathic low back pain," *Spine*, **26**, No. 7, 724-730 (2001).
40. S. H. Roy, C. J. De Luca, and D. A. Casavant, "Lumbar muscle fatigue and chronic lower back pain," *Spine*, **14**, No. 9, 992-1001 (1989).
41. B. M. Wand, L. Parkitny, N. E. O'Connell, et al., "Cortical changes in chronic low back pain: current state of the art and implications for clinical practice," *Man. Ther.*, **16**, No. 1, 15-20 (2011).
42. H. Tsao, M. P. Galea, and P. W. Hodges, "Reorganization of the motor cortex is associated with postural control deficits in recurrent low back pain," *Brain*, **131**, No. 8, 2161-2171 (2008).