Role of fatigue on proprioception and neuromuscular control

Pongsak Yukanandana *


One of the major causes of sports injury is muscle fatigue. Muscle fatigue leads to the inability of muscles, which control joint motion, to resist extreme motion during exposure of external force. Fatigue is defined as inability of the muscle to maintain power output. Fatigue is caused by central and peripheral factors. There are evidence of some metabolic changes in the fatigued muscle such as lactate accumulation and electrolyte imbalance. The central and peripheral causes of muscle output failure are still under investigation. Reflex inhibition and also EMG changes were found during muscle fatigue. The alteration of proprioceptive senses during fatigue may cause by changes in the input sensori-motor pathway. Motor control was also affected by fatigue, which demonstrated by alteration of electromechanical delayed (EMD) and muscle reaction time. Prevention and management of sports injury would improve if we know more about muscle fatigue and its affects.

Key words: Fatigue, Proprioception, Neuromuscular control.

Reprint request: Yukanandana P, Department of Orthopedic, Faculty of Medicine, Chulalongkorn University, Bangkok 10330, Thailand.

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* Department of Orthopedic, Faculty of Medicine, Chulalongkorn University
พลังศักดิ์ ดุษฎีนนท์. บทความความยืดหยุ่นของกล้ามเนื้อต่อการรับรู้ของข้อ และระบบการควบคุมการทำงานของระบบประสาทกล้ามเนื้อ. จุฬาลงกรณ์เวชสาร 2541 ก.ค.; 42(7): 531-39

ภาวะความยืดหยุ่นของกล้ามเนื้อก็เกิดขึ้นเมื่อกล้ามเนื้อไม่สามารถรักษาระดับกำลังในการทำงานอย่างต่อเนื่องของกล้ามเนื้อไว้ได้ ภาวะความยืดหยุ่นของกล้ามเนื้อเกิดขึ้นเร็วกว่าการเปลี่ยนแปลงทางมวลอย่างช้าๆ ในกล้ามเนื้อเอง หรืออาจเกิดจากปัจจัยอื่น ๆ ได้แก่ปัจจัยจากกล้ามเนื้อหรือปัจจัยจากระบบประสาท ส่วนปลายซึ่งมีผลทำให้กำลังของกล้ามเนื้อดลดลงไม่สามารถรักษาระดับการทำงานของกล้ามเนื้อไว้ได้ ในกรณีที่ภาวะความยืดหยุ่นที่เกิดระหว่างการล้มกิฬายิ่งปัจจัยน่าจะมีการลดลงเนื่องที่ทำให้การทำงานของระบบเกิดที่ไม่สอดคล้องกับทาง บทความนี้มีความน่าจะเป็นที่จะแสดงถึงการเปลี่ยนแปลงของการควบคุมการทำงานของกล้ามเนื้อในภาวะที่มีการยืดหยุ่นของกล้ามเนื้อต่อระบบการรับรู้และระบบส่งกำเพื่อให้เกิดความรู้สึกเช่นซ้ำ ๆ ถ้านั้นจะนำไปสู่การป้องกันการบาดเจ็บจากภัยอันเนื่องมาจากความยืดหยุ่นของกล้ามเนื้อดังกล่าวไปได้
Importance of Neuromuscular Fatigue

Fatigue is simply defined as an inability to maintain a power output or force during repeated muscle contraction which may be attributed to either metabolic or non-metabolic peripheral factors.\(^{(1,2)}\) Muscle fatigue can also be due to central factors such as psychological deterioration of the athletes so that they can not bring full use of their muscle power to perform activities. The result of fatigue may cause poor athletic performance or injuries. There are evidences that fatigue has been a participating factor in various kind of sports injuries\(^{(3,4)}\) such as swimming,\(^{(5)}\) gymnastic,\(^{(6)}\) ice hockey,\(^{(7)}\) running, \(^{(8)}\) baseball,\(^{(9,10)}\) golf\(^{(11)}\) etc. How fatigue can affect performance is an issue still being debated. Bigland-Ritchie, et al\(^{(12)}\) pointed out that the motor unit and neuromuscular control played a very important role in fatigue.

Muscle Fatigue

Although the concise definition of muscle fatigue is a failure to maintain a required or expected force output, it may be more appropriately represened as a failure in work performance.\(^{(13)}\) Winter\(^{(14)}\) has referred to muscle fatigue as occurring when the muscle tissue cannot supply the metabolism at the contractile element, because of either ischemia or local depletion of any of the metabolic substrates. Enoka\(^{(15)}\) has denoted muscle fatigue as a class of acute effects that impair performance which includes both an increase in the perceived effort necessary to exert a desired force and an eventual inability to produce this force.

Neuromuscular Fatigue: Central or Peripheral

The effect of fatigue on neuromuscular control has been extensively investigated for many years. There were evidences to support that effected fatigue on central neuromuscular control and also some evidences to support the peripheral effect. Setchenov\(^{(16)}\) observed that the exhausted muscles of one limb recover faster if the opposite limb was exercised moderately during the recovery period. Asmussen\(^{(2)}\) explained the effect of diverting activity during muscle fatigue as a feedback of nerve impulses from the fatigued muscles that impinges on the part of the reticular formation in the brain and caused an inhibition of the voluntary effort. This phenomenon was called "Setchenov phenomenon". Diverting activity produced an increased inflow of impulses from nonfatigued parts of the body to facilitate part of the reticular formation. The other evidence for central base fatigue effect is the effect of psychological stimulation in enhancement of performance during muscle fatigue. Ikai and Steinhaus\(^{(17,18)}\) showed that a simple shout during exertion could increase what was formerly believed to be maximal strength.

Merton\(^{(19)}\) showed no difference in tension development during voluntary contraction of the muscle compared to electrically induced maximal contraction. When the muscle was fatigued by voluntary contraction, electrical stimulation could not restore tension. He suggested that performance was not limited by the CNS but the “periphery” was the site of the muscle fatigue. Missiuro\(^{(20)}\) and DeVries et al\(^{(21,22)}\) showed the increasing of the EMG voltage during muscle exhaustion and suggested that central factors were not involved to any great extent in high-intensity fatigue.
Reflex Inhibition at the Motor Neuron Interface

Sherrington (23) in 1947 observed that the reflex arc was much more fatigable than conduction in the nerve fiber. He found that axons failed to respond to an artificial stimulus after several trials while a reflex became inelicitable after far fewer trials, and the more interneurons or synapse involved in the reflex arc, the quicker it became fatigued. Gandevia et al (26,27) believed that the motoneuron was the last site which any CNS (supraspinal, segmental, sensory feed back) command could be modified. Bigland-Ritchie et al (28,29) proposed that the reflex inhibition was the primary modulator of EMG changes during fatigue and reflex inhibition might have been caused by local muscular changes, such as lactate accumulation during fatigue.

Effect of Fatigue on Proprioception and Neuromuscular Control

Fatigue effects on joint proprioception

There is increasing concern about the correlation between proprioceptive senses and sports injuries. Many studies revealed the deficiency of proprioception and kinesthesia after injuries. In neuromuscular control pathway, proprioceptive signals give rise to an afferent impulse system. The afferent signal might stimulate monosynaptic spinal reflex, polysynaptic reflex or cortical feed-back control. Many programs necessary to improve proprioceptive senses were prescribed for injured athletes before returning to sports activities. Improvement of their neuromuscular control to would prevent reinjury and gain a good level of performance. It is still controversial whether sensory receptors in the joint, including capsuloligamentous tissue or sensory receptors in the surrounding muscles would play more important role in proprioceptive sense and kinesthesia. Some studies (30) have been reported that muscle sensory receptors were the prime mechanoreceptor for proprioception and kinesthesia. The mechanoreceptors in the skeletal muscle included muscle spindles and Golgi tendon organ (GTO). The specialized muscle spindle send afferent signals through alpha motor nerve fibers. The muscle spindles also contain intrafusal muscle fibers which are controlled by gamma motor neuron. Muscle spindle was believed to provide sensory information concerning the relative muscle length during stretching. GTO was located in the musculo-tendinous junction and was believed to provide inhibitory signals to control muscle contractions. During muscle fatigue, the extrafusal muscle fiber loss its contractile ability due to biochemical change and alteration of the neuromuscular control mechanism. The function of intrafusal muscle fibers during the fatigue state was still being studied. (30) There were some evidences of gamma motor neuron function during fatigue. Some researchers questioned that the intrafusal muscle fiber might not be fatigued and can still be function. (30)

There have been very few researches study about proprioception and fatigue in humans. Skinner et al (30) studied joint position sense of the knee before and after fatigue. They measured thresholds to detect passive motion and reproduction of passive positioning sense in eleven healthy male subjects. Their fatigue protocol consisted of running in the field or running on a treadmill, followed by workouts on isokinetic
machines until the work output decreased more than 10%, compared to pre-fatigue power output. They found that reproduction of passive positioning angle was significantly increased but the threshold to detect passive motion was not significantly changed. They found a correlation between reproduction of passive positioning and the threshold to detect passive motion at pre-fatigue, but not at the post-fatigue state. They concluded that joint position senses were affected by fatigue and they believed that the muscle receptors were prominent determinants of joint position.

Sharpe and Miles (31) studied joint position sense at the elbow after fatigue. They tested thirteen human subjects for ability to match the elbow angle of their arm with the contralateral arm before and after fatigue. Fatigue was induced by a series of maximal voluntary contractions of the elbow flexors of the dominant arm until maximum voluntary contraction force reduced to 30-60% of the pre-fatigue strength. They found inconsistent changes of the pattern of position matching after fatigue. And they found no reciprocal changed when the fatigued arm was the matching arm compared with when the non-fatigued arm was the matching arm. The authors believed that the central fatigue process might affect the ability to match the elbow position during contractile fatigue.

Voight et al (32) studied the effect of muscle fatigue on shoulder proprioception. They tested active and passive repositioning of the shoulder joint at 75 degrees of shoulder external rotation and 90 degrees of elbow flexion before and after fatigue. They fatigued the shoulder by using isokinetic internal and external rotation contraction at 180°/sec until peak torque output dropped below 50% of the maximum value for 3 consecutive repetitions. They found significant differences of the proprioception score between pre- and post-fatigue. They concluded that shoulder proprioception was diminished in the presence of the muscular fatigue. They proposed that clinical rehabilitation protocols must emphasize muscular endurance. Leiphart and Sterner (33) found different results for shoulder proprioception in the mid-range of shoulder motion after fatigue. They found no significant differences of proprioception and kinesthesia between before and after fatigue. They suggested that fatigue might not affect proprioception in the mid range of shoulder position compared to the extreme position of the shoulder which is more susceptible to injuries.

**Effect of Fatigue on Neuromuscular Control**

Muscle fatigue has been suggested to adversely affect the neuromuscular control of the joint. Electromyographical studies of fatigue muscles have demonstrated an extended latency of muscle firing (3) and less efficient muscle processes, (30) when compared to unfatigued muscle. Measurement of lower extremity balance, another means of quantifying the efferent neuromuscular pathway, also appears to be altered muscular fatigue. (35)

One of the most important factors for sports performance is reaction time which is the time, between stimulation and response. Reaction time is also very important in prevention of athletic injuries. When the athletes encounter a dangerous situation, how fast the muscle can generate force so that they can adjust to or move away from that situation is very important, and
when abnormal movements occur nearly to the point of anatomical strain, reaction time for muscle contractions to prevent anatomical disruption of the capsuloligamentous structure will prevent severe injuries. The response time has been reported to be related to the rate of force development. Muscle force generation is dependent on central and peripheral factors. Central factors include the proportion of the available total motor unit pool which is recruited during contraction, motor neuron excitability and the type of motor unit recruited during contraction. The peripheral factors include the cross-sectional area of the contracting muscle and the biochemical and the electrical events associate with the joining-sliding-relaxation of the contractile proteins. The response time delay consists of the time interval between stimulus and change in electrical activity in skeletal muscle and the time delay between the change in electrical activity and actual force generation by the muscle.

Electromechanical delay (EMD) is defined as the delay between onset of EMG activity and the onset of mechanical response. In the studies of human movement, electromyography (EMG) is being used widely to assess muscle firing patterns and recruitment of motor unit during various activities. Norman and Komi proposed that the length of the EMD is primarily affected by the time necessary to stretch the series of elastic components of a muscle to a point where muscle force can be detected, and factors such as initial muscle length, type of contraction and velocity of contraction influence this phase lag. However, the reported EMD values are still in a wide range even in the same muscle, same muscle length and same type of contraction. A weak point in this procedure is that one should define the level of both EMG onset and the threshold level of mechanical response. Most of the studies were predominantly focused on the rising limb of force production. Thus it is important to verify signal between muscle activation the and deactivation phase. In the EMG studies, EMD would be collected by shifting of the phase between EMG signal and torque signal. Vos et al. found longer relaxation delays in fatigue knee extension protocols in vastus medialis, rectus femoris and vastus lateralis muscles. Zhou et al. also found significant prolonged EMD of knee extensors in isometric contractions after fatiguing exercise. They stated that the mechanisms of EMD lengthening during fatigue could have been due to the deterioration in muscle conductive, contractile or elastic properties and require further study.

Wojtys et al. investigated the effect of quadriceps and hamstring muscle fatigue on anterior tibial translation and muscle reaction time in 10 healthy subject. They defined muscle reaction time as the time between the onset of passive anterior tibial translation and the onset of muscle firing. During fatigue they found an average increase of 32.5% in anterior tibial translation, and the response times in the gastrocnemius, hamstring and the quadriceps originating at the spinal cord and cortical level were slower. However the recruitment order of the lower extremity muscles in response to anterior tibial translation did not change with muscle fatigue. They proposed that the lengthening of muscle reaction time was in agreement with both Woods et al. and Biglan -Ritchie et al. who reported a decrease in motoneuron firing rates in the upper extremities during muscle fatigue and the reason for the outcome was
thought to be that as motoneuron firing rate decreased, muscle reaction time should lengthen. They believed that muscle fatigue affected the dynamic stability of the knee, altered the neuromuscular response to anterior tibial translation, and fatigue may play an important role in the pathomechanics of knee injuries in physically demanding sports.

Nyland et al \(^{43}\) investigated the effect of muscle fatigue on the muscle firing pattern of female athletes who performed a run and and stop maneuver. It was demonstrated that a longer time to the onset of muscle activation occurred following the fatigue test trials than during nonfatigue trials. A delayed rate of firing may create a moment when the knee joint is unable to stabilize against joint forces resulting in ligament injury. Additionally, decreased neuromuscular control, as determined through the assessment of balance, has been demonstrated in healthy individuals subsequent to the induction of knee joint muscular fatigue. \(^{33}\)

**Conclusion**

Muscle fatigue caused deterioration effect on sensori-motor system, which was shown by changes of the sensory input system, controlled system, feed back mechanism and also output unit. Endurance training, proprioceptive training and sensori-motor training as well as mental preparation may help preventing injuries from muscle fatigue.

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