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Review Article

# Electromyography and Muscle Fatigue: A Review

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## Abstract

Muscle fatigue is commonly pronounced in clinical aspects as exercise induced syndrome. In this review, we presents physical structure of muscle including actin and myosin arrangement for Excitation–contraction coupling and contribution of ions and other factors like $O_2$, ROS, pH, Blood flow, lactate, inorganic phosphate, Heat shock protein for muscle contraction and fatigue development. Finally the EMG power spectrum characteristics also discussed before and after the fatigue development.

**Keywords:** Sarcomere, Actin, myosin, ATP, Muscle fatigue and Electromyography.



## INTRODUCTION

The main function of the skeletal muscle is to provide force during walking, running and other everyday actions [1]. Muscles generate force during activities and produce movement. It is the only tissue in the human body having the properties of extensibility, elasticity, irritability and ability to make tension [2]. When performing task in long time or repetitive muscle gets tired and does not generates the required force to complete the task. This is because ions transportation of muscle is affected due to deficiency of energy present in muscle and metabolic products. Then end result is termed as muscle fatigue [3].

Neuromuscular fatigue is determined by a peripheral and a central component. Peripheral fatigue results from processes at or distal to the neuromuscular junction that lead to a reduction in force or power output in response to a given neural input. Central fatigue entails processes within the central nervous system (CNS) that reduce neural drive to the muscle and result in a decrease in voluntary muscle activation and therefore a decline in force or power and a compromised performance. Reductions in neural drive can result from changes at premotor regions of the brain and/or within the motor pathway [4].

Depends on duration of fatigue presence, the muscle fatigue is divided into acute fatigue and chronic fatigue. The acute fatigue recovered by rest in one week, chronic fatigue is not recovered from rest which presence more than one month. The detection of fatigue depends on physiological, electrical and emotional characteristics of human [5]. The monitoring of fatigue is important because it makes permanent damage to the muscle which leads endocrine disorder, immunity abnormal, organic diseases and enrichment of ergonomics. It is also affects the daily life activity of normal people and performance of athletics and sports people [6].

In the present review, we focus on selected contemporary factors related to muscle fatigue. This includes structure of muscles, motor neuron function with regard to the muscle contraction. The other factors which lead to fatigue are addressed here. This includes neural factors, potassium, calcium, Magnesium, blood flow level, oxygen present in blood, metabolic products like lactate, inorganic phosphate, reactive oxygen species (ROS), heat shock protein and orosomucoid. Insights into power spectrum of EMG changes due to fatigue are discussed.

### Physical structure of muscle fiber

Single muscle cell is called muscle fiber and which is surrounded by it is called sarcolemma. Muscle fiber Cytoplasm is called in the name of sarcoplasm. It consists of nuclei, mitochondria and more number of threadlike myofibrils. The Myofibrils are two types of proteins named by actin and myosin, which is patterned in parallel to one another. These proteins have important role during muscle contraction [7].

From the microscope view of muscle fiber the visible bands are characterized during muscle contraction. A basic unit of muscle fiber is named by sarcomere which is placed in between Z lines. Each sarcomere is divided by M line of equal length. It consists of A band and I band. The A band holds thick myosin filament and myosin filaments surrounded by six actin filaments. The I band contains only action filaments. In the Z line the protein filament are joined to couple the sarcolemma. The thick myosin filaments are contained in the H-Zone which is the middle of A band. When the muscle contracted the thin actin filaments in extreme of the sarcomere are moving towards each other and same time the Z lines move towards the A bands. I band get narrow to maintain their same size of before contraction during contraction and H Zone is removed in the sarcomere .The myosin and action filament are joined together to form cross-bridge during contraction. This cross bridge responsible for generation of muscle force and energy expenditure during contraction [8, 9].

**Motor neuron function**

Adult skeletal muscle fibers are each innervated in the spinal cord by a single branch of the axon arising from α -motoneuron. The α-motoneuron and all the muscle fibers it innervates constitute a motor unit and this is the functional unit of the muscle. α - Moto neurons differ in size and excitability and it is the recruitment of these cell bodies in the spinal cord that determine which fibers within the muscle are active during a movement. Small motor neurons, which are more readily activated, innervate relatively small numbers of muscle fibers while larger less excitable neurons have a greater number of axonal branches and thus control larger motor units. As a consequence of their different excitabilities the small motor units tend to be recruited early and frequently for activities such as walking and maintaining posture since these activities involve small forces while the large motor units are only involved when rapid or large contractions are required. The muscle fibers innervated by these different types of motor neurons have different contractile and metabolic characteristics, the small units being slow contracting, with a high oxidative capacity and fatigue resistance [10]. The large units tend to be fast, predominantly glycolytic and fatigue rapidly. The controlling action is done by neurotransmitter is a chemical messenger between two neuron cells then end result contract the muscle. The main messengers are glutamate, aspartate, D-serine, γ-aminobutyric acid (GABA), glycine, nitric oxide (NO), carbon monoxide (CO), hydrogen sulfide ($H_2S$), dopamine (DA), norepinephrine (noradrenaline; NE,NA), epinephrine (adrenaline), histamine, serotonin (SER, 5-HT). Out of these transmitters some have property of inhibit action and some of have exhibit action on performance of muscle contraction.

**Causes of muscle fatigue**

Muscle contraction is the flow of action from brain to muscle fiber. The factors which affect the flow of action potential from brain to muscle fiber are Neurotransmitter, ions, energy availability in the muscles, metabolic products like lactate, inorganic phosphate, reactive oxygen species (ROS), heat shock protein and orosomucoid. The cross bridge cycling is affected by presences and changes in above factors which lead to fatigue.

**Neural contribution**

The CNS, through a central neurotransmitter, generates many excitatory and inhibitory inputs on the spinal motoneurons, thus stimulating activating motor units (MUs) to get the force output. The strength and timing of contraction are controlled by the firing of the motoneurons [10]. When first recruited in a healthy system, MUs usually fire at 5–8 Hz. During brief non fatiguing voluntary contractions in humans, the mean MU firing rates are 50–60 Hz. MUs are recruited or derecruited in an orderly fashion on the basis of the motoneuron size, and they essentially control the amount of muscle tissue being activated.

Slowing or decreasing of MU firing contributes to the loss of force that leads to fatigue. Moto neuron firing is influenced by intrinsic changes in the motoneuron properties, descending drive and conducting feedback. During fatiguing maximal contractions, motoneuron firing rates decrease because of the following reasons: (1) Repetitive activation (repeated firing) of motoneurons leads to a decrease in their excitability to excitatory synaptic input (2) the excitatory drive from the motor cortex or other supraspinal area to the motoneurons is small. (3) the firing of group III/IV muscle afferents is increased, thus decreasing motoneuron firing; (4) the firing of muscle spindles (sensory receptors) is decreased, thus decreasing firing of group IA muscle afferents, increasing pre synaptic inhibition, and finally decreasing motoneuron firing.(5) specifically, group III/IV muscle afferents also exhibit feedback interaction with cardiovascular and respiratory processes via the autonomic nervous system, thereby improving muscle blood flow and oxygenation and consequently slowing the development of fatigue of the muscle itself [11].

**Energy availability in muscle fiber**

ATP (Adenosine triphosphate) is the protein and the main source of energy for muscle activation and which determine the muscle force [12]. The main ATPase which contributes muscle activity is $Na^+/K^+$-ATPase, myosin ATPase and $Ca^{2+}$ATPase.The energy required to move 3 ions of $Na^+$ to outside the of cell and 2 ions of $K^+$ inside of cell after action potential is 1 molecule use of ATP . The force generated by muscles depends on ATP usage of myosin and $Ca^{2+}$ATPase is necessary for re intake of calcium ion into SR for muscle in resting potential [13].

For ATP production, carbohydrate of glycogen is necessary which is stored in muscle cell. There are three different sub location of glycogen in muscles, the places of myofibrils and close to SR and mitocandria named inter myofibriller, Glycogen placed myofibrils and I-Band of Sarcomere named intramyofibriller and glycogen is located beneath of sarcolemma and primary next to mitochondria, lipids, neulai .There is linear relationship between glycogen content and muscle endurance during exercise since it is main fuel. Glycogen oxidation plays important role for ATP regeneration for long time exercise for more than hours and one of the important role of glycogen is tricarboxylic acid production to maintain oxidative metabolism [14]. SR $Ca^{2+}$ release, reuptake and $Na^{+}$/K+ pump function are affected by level of glycogen. Glycogen level is responsible for excitation-contraction coupling and relaxation of muscle [15].

**Flow of blood and oxygen level in muscles fiber**

High-intensity exercise can decrease arterial $O_2$ saturation by 5–15% from resting values (hemoglobin saturation 98%), a phenomenon referred to as exercise-induced arterial hypoxemia. Factors determining arterial desaturation during exercise include an inadequate compensatory hyperventilation, acid-induced and temperature-induced shifts in $O_2$ dissociation at any given arterial $PO_2$, and an excessive widening of the alveolar to arterial $O_2$ difference. The resultant arterial desaturation compromises $O_2$ delivery to the locomotors muscle and, given the importance of convective $O_2$ delivery in determining exercise-induced fatigue, promotes the development of peripheral fatigue [16, 29].

The blood is responsible for transportation of neutrician and oxygen to each and every cells of human body small intestine and collects byproduct of metabolic process. So the oxygen necessary for working muscle is carried by blood for ATP production in cell respiration [14, 15]. Previous result shows that due to decrease of oxygen, ATP production is affected because of ATP is the energy storage of muscle and increase in oxygen the energy supplied to muscle increases, thus increases muscle endurance time [11]. The prevention of blood flow in the working muscles decreases muscle endurance time and decaying the muscle force, same time blood flow in the muscle changes due to decreasing of MVC force. Normally oxygen intake and ATP utilization are raised when Voluntary oxygen maximum. During heavy exercise demand for large ATP cannot met by oxygen because already oxygen reached maximum, then the metabolic stability is not in balance state that goes to fatigue. These factors give the potential importance of blood flow in the fatigue prevention [17].

**Metabolic factors**

The cross bridge activities are affected by raise in intercellular metabolites like $H^{+}$, lactate [16], Pi and ROS [14] where generated in the muscle activation process. During hydrolysis of ATP muscle generate the force, ADP and Pi as result of contraction then ADP molecules combine with creatinephosphate to produce ATP and Pi again. ie,

CreatinePhosphate+AdenosineDiphosphate $\longrightarrow$ Creatine +ATP

The metabolic process also decomposes Creatine phosphate into creatine and Pi. During high intensity exercise the above action increases the amount of Pi generation as a result decreased muscle activation due to blocking action of Pi to $Ca^{2+}$ from SR [17]. Glycogen breakdown during metabolism generates intercellular accumulation of organic acids of which results raise in lactic acid. The lactic acid decomposed into lactic and $H^{+}$ ions. The increase of $H^{+}$ ions also interfering $ca^{2+}$ release from SR reduces which reflects deploying muscle contraction. Well known that mitochondria are power house of our body. During mitochondrial respiration ATP is generated with consumption of oxygen with byproduct of ROS is generated. The main ROS are superoxide, hydrogen peroxide and hydroxyl radicals. When muscles are contracted in heavy work, the consumption of ATP raises which reflects increasing of ROS and deficiency of ATP. The increase of ROS affects $Ca^{2+}$ sensitivity in SR, decrease $Ca^{2+}$release from SR, $Na^{+}/K^{+}$ pump action and group IV muscle afferents, these leads to direct inhibition of motor neuron[18]. The other metabolites like ADP, PCr and Mg has role with fatigue mainly ADP reduces the fiber velocity but increases muscle force because ADP rate directly proportional to cross bridge in high energy state. ADP does not create the disturbance in cross bridge cycling but related to inhibition of SR $Ca^{2+}$ release [19].

**Fatigue reactants**

Every organs of human body have different level of fatigue resistance due to presence of protein in their cells. Cortisol, catecholamine, IL-6 and HSPs are the most fatigue reactant which have role in the muscle function [20]. Among these HSP25 have vital role in muscle functioning because it regulates muscle structure, repair muscle protein and reduces apoptosis action of muscle [21]. ORM raises the muscle glycogen and increase the endurance time, but ORM deficiency results in reduction in endurance time so it is an anti-fatigue protein [22]. Further studies have demonstrated that ORM binds to C–C chemokine receptor type 5 (CCR5) on muscle cells and activates AMPK, thus promoting glycogen storage and enhancing muscle endurance, and representing a positive feedback mechanism for resisting fatigue and maintaining homeostasis. Modulation of the level of ORM and CCR5 signaling may be a novel strategy for the management of muscle fatigue [23].

**Role of Calcium**

The Calcium ion $Ca^{2+}$ plays a critical role as an initiator and preserving agent of the cross-bridge cycle in the force generation of skeletal muscle [15, 24]. A stimulus from CNS is passed to neuromuscular junction which is generated Acetylcholine of neurotransmitter strikes the muscle cell. In actual, $Na^{+}$ concentration is more extracellular fluid of cell and $k^{+}$ concentration is less inside the cell. When Acetylcholine strikes the cell more $Na^{+}$ ions move into the cell than $K^{+}$ ions. During repolarization period of action potential SR releases $ca^{2+}$ions into sarcolemma [25]. $Ca^{2+}$ and troponin forms tropomyosin which unblock the active sites of Actin. When myosin hydrolysis ATP, ADP and Pi are generated and Energy released, with result myosin power the actin filament inwards and shortening of sarcomere.

When $Ca^{2+}$ is no longer present on the thin filament, the tropomyosin changes conformation back to its previous state so as to block the binding sites again. The myosin ceases binding to the thin filament, and the muscle relaxes [26, 27].The causes of calcium release decreasing from SR have the following effect which leads to inhibition of cross bridge cycling. (1) The nature of $Na^{+}$ inwards and $K^{+}$ outward to cell happens when the action potential is applied, the action potential amplitude decreases due to high frequency stimulation [28]. (2) The inorganic phosphate makes decreasing of $ca^{2+}$ in SR because Pi can enter in SR and precipitate $ca^{2+}$. This action decreasing the free $ca^{2+}$ and quantity of $ca^{2+}$for release in SR. (3)The $Mg^{2+}$ is bound with ATP in the resting state of muscle fiber, when muscle contraction is more which reflects decreasing the ATP which increase the amount of free $Mg^{2+}$, the free $Mg^{2+}$ also blocks the release of $Ca^{2+}$ in SR[30].

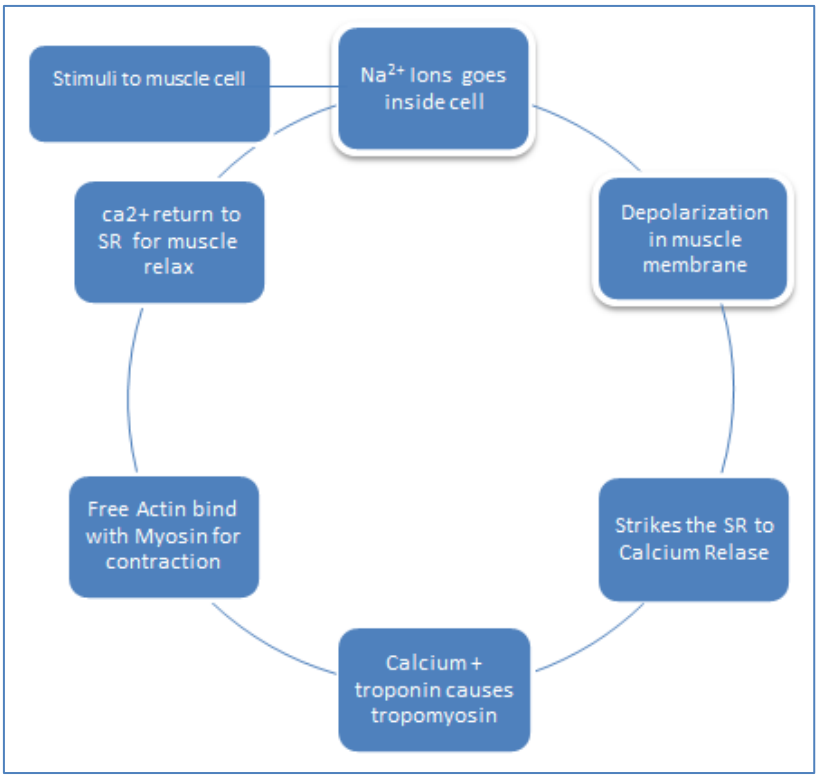


**Fig-1: Muscle Excitation and Contraction coupling**

**Effects in muscle conduction velocity due to fatigue and characteristics of emg power spectrum**

Muscle fiber conduction velocity (CV) is defined as the propagation velocity of action potentials along the membrane of a muscle fiber. As an important physiological parameter, CV is correlated with muscle fiber membrane properties, e.g., ion concentration, pH, muscle temperature and motor unit (MU) firing rate. The above said parameters of pH and ion concentration are changed in fatigue condition [31].

The motor unit denotes the basic functional element of the central nervous system and muscle that produces movement. It comprises a motor neuron in the ventral horn of the spinal cord, its axon, and the muscle fibers that the axon innervates. The central nervous system controls muscle force by varying the activity of the motor units in the muscle. The force exerted by each motor unit depends principally on the number of muscle fibers that are innervated by the motor neuron and the rate at which the motor neuron discharges action potentials [32]. The motor unit population that innervates a muscle is heterogeneous, due to systematic variations in the properties of both the motor neurons and the muscle fibers. These systematic variations in the components of the motor unit have been used to distinguish different types of motor units [33].

Intramuscular recordings have consistently found that the rate of increase in force during a submaximal voluntary contraction depends on the rate at which the activated motor units discharge action potentials. The discharging of action potential is depends on muscle conduction velocity and it is affected by change in muscle pH and ion concentration in muscle due to lactic acid accumulation [34]. So, in fatiguing condition muscle velocity decreases due to pH change which reflects EMG frequency decreases and muscle force increases to compensate load which reflect in EMG magnitude.

## CONCLUSION

The contraction of muscle has a series of events, starting from stimuli generated from brain, passes to neuromuscular junction then to motoneuron which initiates Excitation process of muscle cell. Action potential from muscle cell resultantly generates the contraction. Any inhibition like flow of calcium in Sarcolemma, Amount of ATP in cell, and oxygen level in this path way reduces the force generation capacity of muscle and lead to fatigue. Metabolic factors H+, lactate, ADP and Pi leads to raise in fatigue, Fatigue reactant like HSP25 and ORM resisting fatigue. Due to changes in pH and ion concentration in muscle, muscle the conduction velocity and firing rate of muscle fiber is decreased which reflects frequency of EMG decreases.

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