Sarcopenia and Locomotive Disorders in Sedentary City Lifestyle

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Abstract
Sarcopenia is an aging disorder that can associate many issues with the muscles, bones and the nervous system of a growing body. It is more prevalently accelerated in the city life set-up. This study gives an overall idea of how and when this process begins during the life. The article is aimed at giving an insight with scientific evidence, the manifestation and signs to watch in a progressing age. Over the years there are muscle lost and replaced by fats accumulation. The size of the cross-section of any limbs if compared with an active young and inactive old person, a clear mark can be seen where the muscle bulk has shrunk and there is more fats mass (Fig 1). When the fat mass percentage exceeds at 70% in aging body instability during ambulation gets distorted which needs to fall and subsequent injuries. The progress in Sarcopenia is affected by sluggish ionic exchange at the cellular level of the muscle fiber. As sarcopenia progresses due to lack of activities and with non-optimized end range of movement, the muscles fiber does not achieve a complete stretch to trigger an autonomous neurological potency to maintain the tone in the muscle fibers. This multiple end gross phenomenon brings about a progress in the aging process of the body. An ionic study of this statement needs further experiment in human body. My article is aimed to trigger and draw attention to research work to establish further understanding of Sarcopenia and perhaps retard aging processes in the muscles.

Lack of movement and lack of optimum stretch at the end of the muscles which are tendon in origin and the majority of them need attach to the bones will bring about weaknesses to the body structure due to lack of calcium (Ca+ ion) exchange activities. This would lead to osteopenia of the bones. In short, Sarcopenia will progress to osteopenia. An osteopenia will progress to osteoporosis. Though, all the processes are inevitable, however active activities with an equilibrium lifestyle can slow this process that could avoid instability, incoordination and fall injuries in the older age group.

Keywords: Sarcopenia, Muscular Diseases, Muscular Atrophy, Aging, Healthy Lifestyle

Sarcopenia
Sarcopenia is associated with aging process where the muscles bulk is lost due to lack of physical activities leading to various other conditions associated with biomechanics discomfort and musculo-skeletal pathologies [1, 2]. The body's skeletal muscles are composed of multinucleated cells called fibers. The number of fibers in every muscles are equal but the morphological change. As the age progresses, the muscle mass reduce whereas the fat mass increases [3].

Physiology of Muscle Bundle
Striated Skeletal Muscles that forms the majority of muscle bundle of our locomotive system that ambulates the body. They are covered externally by connective tissue called epimysium, which surrounds the entire muscle, holding it together. Epimysium is fiber bundles that wrap the fasciculi, in a sheath of connective tissue called perimysium. Finally, within the perimysium are the muscle fibers, which are the individual muscle cells [4]. The endomysium, is another sheath of connective tissue that surrounds each muscle fiber. So the epimysium, perimysium, and endomysium are connective structures that together form the tendon. Muscle fibers range in length from 1 mm to a maximum of 12 cm in the sartorius muscle. Their diameter ranges from a minimum of 10 μm to a maximum of 100-105 μm (average: 10–50 μm) [4].

Each cellular muscle fiber is made up by fusion of progenitor cells called myoblasts and thus form syncytia. The skeletal muscle fiber is a giant multinucleate cell. Skeletal muscle fibers are cylindrical in shape and contain many nuclei.
(even hundreds) located near the sarcolemma (the cell membrane of muscle cells)[5]. A gelatin-like substance fills the spaces between the myofibrils that runs the length of fiber. There can be hundreds of myofibrils in a single muscle fiber [6]. This is the sarcoplasm and it comprises the cytoplasm of muscle fiber. The sarcoplasm differs from true cytoplasm in that it contains a large quantity of stored glycogen as well as the oxygen-binding compound myoglobin, which is quite similar to hemoglobin. The myofibril consists of a linear array of sarcomeres (approximately 2.5 μ long and 1 μ in diameter), the structural units that carry out muscle contraction and relaxation. The number of sarcomeres in a fiber depends on the length of the fiber. Muscle contraction and relaxation are controlled by the intra-fiber free Ca++ concentration, [Ca++], in the myoplasm that bathes the muscle filaments of the sarcomeres. In turn, the [Ca++] is controlled by a network of membranes that regulate and coordinate the process in time and space [6].

Another special structure of the muscle fiber is the sarcoplasmic reticulum, which is the smooth endoplasmic reticulum. Its distinct shape can be recognized in every sarcomere. Both ion channels and ion transporters exist in the membranes of sarcoplasmic reticulum (ST) Ca2+-Mg2+-dependent ATPase is a major component of the sarcoplasmic reticulum (SR) membrane that accounts for more than 70% of the total protein. It serves as a translocator for Ca2+ as well as an energy transducer [6, 7]. However, the manner in which the movement of the enzyme molecule is coupled to the Ca2+ translocation across the membrane and associated with ATP hydrolysis is not clearly understood. To better understand the molecular basis of the active transport of Ca2+, information is needed concerning the structural aspects of the Ca2+, Mg2+-dependent ATPase molecules in the sarcoplasmic reticulum membrane. One of the most important findings in the kinetic studies on the Ca2+, Mg2+-dependent ATPase reaction in the presence of C12E8 is that the effect of C12E8 on the catalytic function of the sarcoplasmic reticulum ATPase is entirely reversible[8].

Muscle Tone

Muscle tone is fully determined by the monosynaptic stretch reflex, that tonic fusimotor activity is necessary for its production in normal humans, and that tonic muscle tone in antigravity leg muscles is responsible for the maintenance of posture [9]. Mechanical mechanisms are involved in the maintenance of resting muscle tone. These tones in the muscles are further maintained by the spinal cord reflex. This is further dependent upon building activates while exercising and ongoing activities that activates inter-neurons functions. Long-latency trans-cortical responses are elicited when a muscle is stretched, and these responses effectively deal with large displacements such as effectiveness and precision in movement [9].

Inactivity Affecting Muscle Tone

Exercises influence growth in the muscles. Growth of muscle can occur in three ways: (1) by an increase in muscle cell numbers, (2) by an increase in muscle fiber diameter, and (3) by an increase in fiber length. All three of these mechanisms are involved in muscle growth. However, growth in cell numbers is limited to genetic and fetal development and also in the prenatal and immediately postnatal period being born with or soon reaching their full complement of muscle cells. However actual growth occurs by either hypertrophy of the existing muscle fibers by adding additional myofibrils to increase the muscle mass or by adding new sarcomeres to the ends of the existing muscle fibers to increase their length[10]. Both of these mechanisms occur during the growth process. During this growth stretching and activities plays a vital part. Growth in the girth of the muscle fibers appears to take place by splitting of the myofibrils. This may be stimulated by development of stress creating an unequal pressure with splitting at the Z-band and development of additional SR and T-tubule systems especially in a regular exercise lifestyle. This adds to the diameter or girth of myofibrers without any hyperplasia. The growth in length occurs at either end of the fibers and results in addition of new sarcomeres. In both cases, new myofibrillar protein must be synthesized and deposited in the muscle cells. It is suggested that adaptation by adding or removing sarcomeres is physiologically determined by the degree of force a muscle can generate, especially in active muscles, that is in turn dependent on the degree of overlap of the thick and thin filaments. Thus, the amount of tension would control the number of in-series sarcomeres in a single muscle fiber. Nutrition is also known to play an important role in muscle building. A well balanced nutrition with adequate calorie diet is required to achieve optimum muscle growth. When the body is sedentary, the fat deposit the muscle fiber is the first to suffer followed by shrinking of sarcoplasm and ultimate the bone calcium deposit is affected which interrupts the nervous system. Brain and the eyes are the last system to be affected [11].
**Initial effects of Sarcopenia**

In a city lifestyle with sedentary and stereotype activities, sarcopenia is believed to start at the age of 30-35 years of age.

With advancing age, decrements occur in many physiological systems averaging on the order of 2% per year [12]. During the active age between 18 to 35, as healthy young adults, under normal circumstances [13], skeletal muscle protein synthesis and degradation is a balanced. Here the dynamic process is maintained in skeletal muscle mass [14]. During aging process and with sedentary lifestyle, however, muscle tissue is gradually lost, resulting in diminished mass and strength, hence developing Sarcopenia. The average adult can expect to gain approximately 500gms of fat every year between ages 30 to 60, and lose about a 250gms of muscle over that same time span; that change in body composition is equivalent to a 7Kgs loss of muscle and a 14Kgs gain in fat over 30 years[15]. This shift in body composition with advancing age is often masked by relative stability in overall body weight.

The consequences of sarcopenia include decreased strength [16, 17], metabolic rate[18], and maximal oxygen consumption [19]. The loss of aerobic capacity with age is predominantly due to a loss of muscle mass [20].

The progressing Sarcopenia, similar activities carried out will become sluggish; repetition counts of an activity will get prolonged. The most crucial point during aging process is when more effort is taken in climbing stairs and even lifting oneself from sitting on ground.

A Random study was conducted between two groups of individual. Group A of ages between 20 and 35, Group B between age 60 to 70.

A Timer was used to calculate time taken for the participant to stand up from a crossed-leg sitting position. All subjects taken in consideration were with normal average body built. Obese subject were not taken into consideration.

A set of 3 readings were taken after an interval of 2 minutes

Table 1 Random study showing an adverage time taken for the subjects to stand pushing themselves up from crossed leg sitting position to standing.

<table>
<thead>
<tr>
<th>Group</th>
<th>1st Set In Sec</th>
<th>2nd Set In Sec.</th>
<th>3rd Set In Sec</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>1.5</td>
<td>1.5</td>
<td>1.4</td>
</tr>
<tr>
<td>B</td>
<td>2.45</td>
<td>3</td>
<td>3.45</td>
</tr>
</tbody>
</table>

This Table shows that time taken for subject in Group B take longer time to push them up from a sitting position. When the same act was repeated, more time was taken for group B to push up whereas subjects in group A remained consistent. This short self-experiment can be a good indicator of any subject to spike up physical activities to improve posture and strength in the muscles.

With the decline in activities, the antigravity muscles of the body are the first to be affected with obvious wasting process that indicates enhancing sarcopenia. In men there will be loss of muscle mass at the Gluteus Muscles (Buttocks). The next muscles involved will be hamstrings followed by the calves.

All this process then alters the posture and the gait pattern. The heel-strike phase in gait pattern gets altered. Shuffling gait becomes more and more apparent as we age. With the lack of optimum stretching of the locomotive muscles, the optimum stretch required to walk gets altered and this evidently leads to stress on the joints especially the knees, hips and the lumbar. Medical conditions like mechanical arthritis of the knee and hips can arise. Lower back pain becomes evident.

**Other Factors affecting Sarcopenia**

Apart from inactivity in sedentary lifestyle, there are other activities that can bring about changes in total muscle mass:

1. A lack of regular physical activity plays a major role.
2. A change in protein metabolism (a deficit between protein synthesis versus degradation).
3. Alterations in the endocrine milieu (decrease in growth hormone (GH) and testosterone and an increase in cortisol and cytokines),
4. A loss of neuromuscular function (denervation versus re-innervation),
5. Altered gene or genetic expression,
6. Apoptosis, dying of cells due to autoimmune diseases and other ailment and being bed ridden

**Conclusion**

Exercise is beneficial and will decrease body fat, improve reserve capacity, and increase muscle strength (and maybe muscle mass), but why is exercise compliance low in elderly people? Are there factors that prevent older individuals from benefiting from exercise? It could be that sarcopenia has both physiologic factors, as has
been discussed, in combination with social issues resulting in older persons not taking up exercise.

Exercises like in the gym, running, jogging, cardio-based exercises, skipping; physical activities, like gardening, cleaning, scrubbing floors, washing cloths, climbing up and down the stairs, farming; sport activities of all kind keep the muscles optimized and gives them a good stretch at the muscle ending [21]. This gives strength to the bones they are attached to[22]. All this retard sarcopenia and osteopenia.

Sarcopenia is a process whereby a loss of reserve capacity in the muscles will result in an increased sense of effort for a given exercise intensity. This forces older individuals to exercise at a greater percentage of their maximal capacity[23]. If this is perceived by the elderly group, that they need more effort to exercise then they will more likely avoid exercises. A vicious cycle then begins in that if one avoids exercise, then future performance will continue to decrease, as cardiovascular function and VO2 max will diminish, again going back to the perception that exercises take most effort, thus exacerbating sarcopenia.

The physiological and psychological factors that contribute to the process of sarcopenia are multifactorial, occurring over a prolonged time period, possibly with no identifiable single cause or mechanism, potentially explaining this age-related loss of mass and strength in and of itself. Therefore, our goal as clinicians should be to advocate exercise yet be aware of the issues that limit compliance (Figure 1). Our goal as researchers should be to gain an improved understanding of the complex biological factors leading to age-related muscle loss beyond those attributable to a simple decrease in physical activity.

Figure 1: Comparison of Normal Muscle Bulk with Elderly Muscle Bulk (cross section of thigh muscle bulk)[24]

References


6. Fleischer, S., *Sarcoplasmic reticulum and other membranes in the regulation of skeletal muscle contraction and relaxation, a multilevel approach*, in *Structure and function*


