Muscle Fatigue

Of the Arms and the Phalanges

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Introduction

- **Objective**

The purpose of the experiment was to observe muscle fatigue during exercises. For the first aspect of the experiment, the index finger (phalanx II) and the thumb (phalanx I) were repeatedly flexed in the squeezing of a spring enabled clothespin. For the second aspect of the experiment, the arm was extended straight and anteriorly to hold a textbook. Both exercises were timed to measure muscle fatigue.

Preliminary observations were that in the book holding exercise, the dominant arm would perform better as the muscles are more often used, and therefore better conditioned, and possibly slightly larger than the non-dominant arm. Only one hand was tested in the clothespin exercise, but it was predicted the dominant one would perform better than the non-dominant one as well for the same reasons. Also for the clothespin exercise, the number of squeezes was expected to decrease as the trials progressed.

In the book holding exercise, the primary muscles used include:

- Pectoralis major – Flexion of the humerus.
- Teres major - Extension of arm.
- Deltoid – Flexion of the humerus.
- Coracobrachialis – Flexion of arm.
- Triceps brachii – Extension at the elbow.

In the clothespin squeezing exercise, the primary muscles include:

- Flexor digitorum – Muscle in forearm that flexes the fingers (digits).
- Opponens pollicis – Functions to oppose the thumb (phalanx I) with the other phalanges.
- Flexor pollicis longus – Flexion of the thumb (phalanx I)

Other factors considered in the preliminary were the lifestyle and workout curriculum of the subject. It is hypothesized that a person that exercises regularly would perform better than a person that lives a sedentary lifestyle and does not work out regularly.

- **Background**

To first understand muscle fatigue, a base understanding of how muscles work is an important foundation. Muscle contraction is served by axons of nerves, called somatic motor fibers. Each of the nerve fibers branches out to a number of muscle fibers. One nerve fiber, combined with the multiple muscle fibers (from 3 to 200) that are innervated, is called a motor unit. When the muscle fibers are stimulated, contraction occurs (Saladin, 408).

At the point where the nerve fibers meet the target muscle fibers is a synapse called the neuromuscular junction. As with other synapses, there is a synaptic cleft where neurotransmitters come into play. With muscle contraction, Acetylcholine (ACh) is an important chemical neurotransmitter. The muscle fiber has ACh receptors, or proteins in the plasma membrane (sarcoplasm), that enable a response. The membrane contains an enzyme called Acetylcholinesterase (AChE) that breaks down the ACh after the ACh has stimulated the muscle, thus being important in the relaxation of a muscle after contraction. When the ACh molecules bind, sodium and potassium ion movements cause a wave of action potentials. The wave spreads down the sarcoplasm, allowing calcium to bind to the troponin of the thin filaments, exposing active sites of actin filaments and allowing binding of myosin heads. According to the sliding filament theory, the myosin head must also have an Adenosine Triphosphate (ATP) molecule bound to it. An enzyme in the head breaks down ATP into Adenosine Diphosphate (ADP) and phosphate (P_i), which releases energy to "cock" the head of the myosin in a high-energy position. The cocked myosin forms a cross-bridge between the myosin and actin, then releases the ADP and P_i into a low-energy position, which tugs the thin filament along with it. The tug is called a power stroke. The head remains bound until bonded with a new ATP. When that new ATP molecule arrives, the cross-bridge is broken, and attachment takes place at a further position down the filament. (Saladin, 409-412).

When the work, or contraction, is done, muscle relaxation occurs. Nerve signals stop arriving at the neuromuscular junction, causing the release of ACh to cease. The lack of ACh cause Calcium to pump back into the cisternae of the Sarcoplasmic Reticulum from which originally source from for storage, dissociating from the troponin the ions bonded with during contraction, causing the tropomyosin to move back into a position where myosin can no longer bind to actin, in turn causing the muscle fiber to ease tension, or relax (Saladin, 416).

Getting into fatigue, the sources of ATP, or energy, must be considered. Muscle contraction depends on ATP. The supply of ATP depends upon oxygen and organic energy sources such as glucose and fatty acids. There are two main pathways in which muscle manages the ATP budget:

1. Anaerobic fermentation – enables a cell to produce ATP in the absence of oxygen, but the ATP yield is very limited, and lactic acid is produced, which contributes to muscle fatigue. Only 2 ATP molecules are created per glucose.

2. Aerobic respiration – produces far more ATP than anaerobic fermentation, without the side effect of producing lactic acid, but a constant supply of oxygen is required. Typically 36 ATP molecules are created per glucose.

When the muscle first contracts, energy is needed immediately. Oxygen that has been stored in myoglobin will create the initial supply needed for aerobic respiration. The supply of oxygen quickly depletes, however. Until the respiratory and cardiovascular system catches up to increased demand for oxygen, the muscle borrows phosphate groups, transferring to ADP, to create ATP, which collectively is known as the phosphagen system. The phosphagen creates nearly all of the energy used in short bursts of muscle activity, and is typically enough to power up to a minute of brisk walking or six seconds of sprinting (Saladin, 423-424).

As the phosphogen system is exhausted, the muscles shift to anaerobic fermentation, the system which is not very efficient at creating ATP, and creates lactic acid contributing to muscle fatigue. The pathway from glycogen to lactic acid produces enough ATP for about 30 to 40 seconds of maximum activity. After 40 seconds or so, the respiratory and cardiovascular system will catch up and deliver oxygen to the muscles fast enough to meet most of the ATP demand.

After the exercise, the body will need to continue heavy breathing to replenish the oxygen reserves that were available and used up in the first minute of exercise, known as oxygen debt. The phosphogen system also needs replaced, which involves synthesizing ATP, and using some to donate back the phosphate groups originally borrowed from creatine. The lactic acid produced during exercise will enter the bloodstream, where the liver converts most of the acid to glucose (Saladin, 424).

Some muscles are more resistant to fatigue than others, depending on fiber makeup. Slow-twitch fibers, or type I fibers, are adapted to aerobic respiration and do not fatigue easily. Muscles composed mainly of slow-twitch fibers, also colloquially called red muscle, are suited for high-endurance, in example, the soleus muscle of the calf and the postural muscles of the back. In comparison, fast-twitch, or type II fibers, are adapted for quick responses but are not as resistant to fatigue resistance. Lacking in as many blood capillaries, myoglobin, and mitochondria, the type II fibers have a pale appearance, which explains the colloquial name “white fibers” attributed. Fast-twitch fibers have a subtype called intermediate fibers, which combine fast-twitch responses with aerobic fatigue-resistant metabolism. Such fibers are rare however, except in endurance trained athletes (Saladin, 426).

Materials and Methods

- **Methods and Materials for Clothespin portion of Experiment.**

  One clothespin, one timer, a subject to be tested, and a lab partner to keep time and record data were used. The subject was instructed to tip-pinch a spring type clothespin as many times as physically possible between the pads of phalanx I and phalanx II for (20) seconds. After the elapsed time, a break of (3) seconds was given, in which the lab partner took the opportunity to reset the timer and record with a pencil and piece of paper the number of times the subject successfully squeezed the clothespin. The process continued until the subject was tested for (10) trial iterations of (20) seconds, with (3) second breaks between each iteration.

- **Methods and Materials for Book Portion of Experiment.**

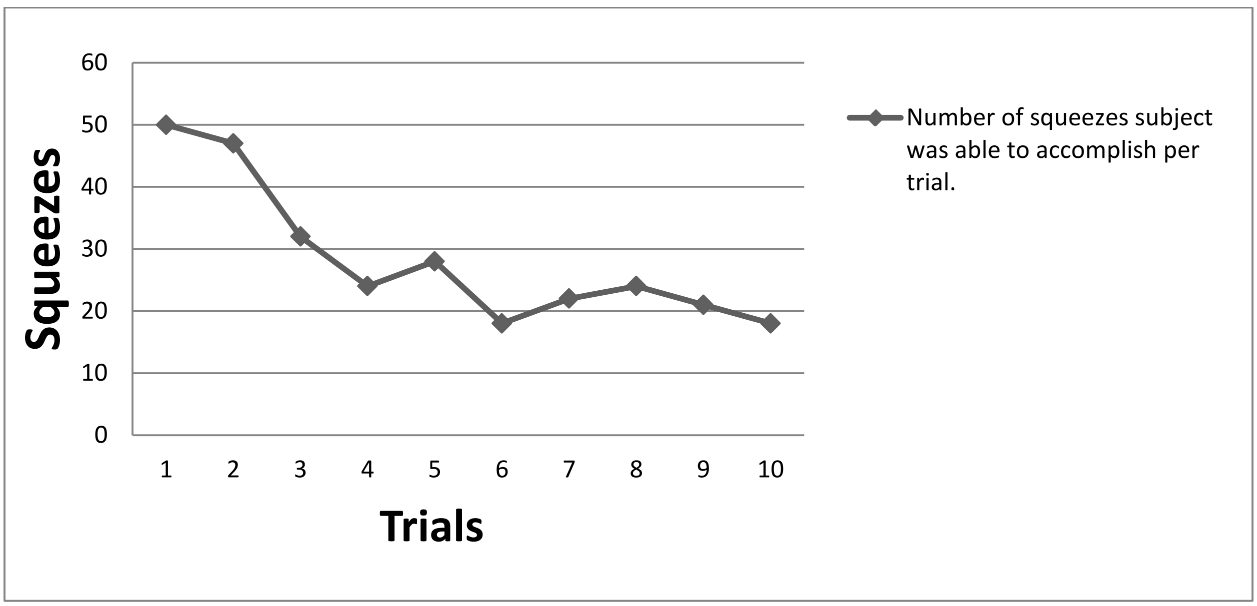
An Anatomy & Physiology textbook with binder, weighing approximately seven pounds, was designated as the weight in which a subject would hold anteriorly with the right arm for as long as the subject was physically able to maintain posture. At the beginning of the test, the lab partner instructed the subject to begin, and started a timer. After the subject either slouched to the point of no longer holding the book anteriorly and with a straight arm, or dropped the book holding arm, the experiment was over, and the time of muscle failure was recorded with pencil and paper by the lab partner. The process was repeated for the left arm.

Results

<u>Table 1.</u> – Ten trials of the clothespin exercise were orchestrated, with each trial lasting 20 seconds. The number of clothespin squeezes in each trial was recorded.

Trial	Number of Squeezes in 20 seconds.
1	50
2	47
3	32
4	24
5	28
6	18
7	22
8	24
9	21
10	18

<u>Figure 1.</u> – Graphed changes in amount of squeezes over iterations of trials.

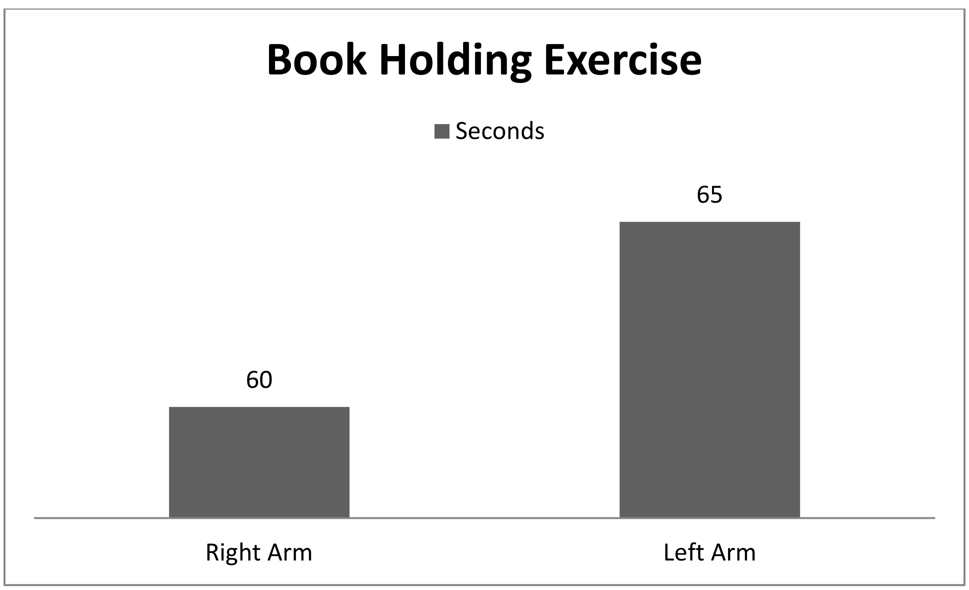


Verbal – Trend shows a decrease in Subject's squeezes as the trials progressed, with minor fluctuations.

Table 2. – The right and left arms were tested to see how long each was able to hold a book anteriorly.

	Right Arm	**Left Arm**
Seconds	60	65

Figure 2. – Graphed representation of the book holding exercise.



Verbal – The subject held the book with the left arm five seconds longer than with the right arm.

Discussion

- **Analysis**

In the clothespin exercise, the results have indicated that the number of squeezes the subject was able to accomplish decreased over time. In the beginning, the subject's muscles were rested and with plenty of oxygen storage that would be needed in the production of ATP. As the trials progressed, the immediate storage was used up, and the muscles began exhausting the phosphogen system, the muscles transferred to lactic acid fermentation. The lower ATP production combined with the lactic acid caused a reduction in performance and a cramping sensation in the muscles. The aerobic respiration helped to sustain the latter part of the experiment, however with the oxygen debt, the initial performance was not able to be achieved, showing only minor fluctuations in the trials.

In the book holding exercise, much of the same principles applied. The subject's arms were went through the same energy production cycles, first going through the stored energy in the muscles, and then going through the anaerobic fermentation phase. One change, however, was that at a certain point of exhaustion, the subject was not able to continue holding the book at all, whereas in the clothespin exercise, the subject's performance decreased but did not totally stop. Some variables to consider are the size of the muscles, the types of muscles involved and the muscle's susceptibility to lactic acid, and the subject's threshold in enduring the pain involved with the lactic acid.

The results for the clothespin exercise went according to the hypothesis, however in the book holding exercise, the non-dominant arm performed marginally better. It is possible that the dominant arm does not necessarily have larger muscles or have better conditioned muscles as predicted. Some activities that the non-dominant arm performs may actually make the arm the stronger and better conditioned arm. More tests would need to be done to create a more accurate average.

- **Interpretation**

The muscle fatigue experiments have shown some of the basic principles of how muscles use energy and go through different phases as the types of energy production changes. Some ambiguities that exist are what effect the weight of the book had, in that if a lighter weight was used, could the aerobic respiration process have kept up some performance rather than having complete muscle failure?

References

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Saladin, Kenneth S. (2011). *Anatomy & Physiology: The Unity of Form and Function* (6th ed.). New York, NY: McGraw-Hill.