ESTABLISHING ACCESSORY MUSCLE ACTIVATION IN HEALTHY
INDIVIDUALS DURING A GRADED EXERCISE TEST.

by

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ABSTRACT

Introduction:
Hyperinflation impairs diaphragm function and presumably places increased demands on accessory respiratory muscles in people with COPD. The purpose of this study was to develop a new application of electromyography(EMG) to assess accessory muscle activation and apply it during a graded exercise test(GXT).

Methods:
Muscle activation, ventilatory parameters and dyspnea were collected from twenty participants during the GXT. The ratio between the inspiratory muscle activation during exercise and maximal muscle activation was used to quantify activation.

Results:
Accessory muscle activation progressively increased during the GXT with the amplitude of scalenes exceeding that of the sternocleidomastoid (maximal activation 29.5% and 12.7%(%MVIC), respectively). Activation was moderately associated with IRV and weakly with dyspnea.

Conclusion:
Healthy individuals who fail to achieve a VO_{2MAX} used minimal accessory muscle activation to increase ventilation. These findings lay the groundwork to investigate accessory muscle activation in individuals with COPD and assess the benefits of interventions.

LIST OF ABBREVIATIONS USED

BMI	Body Mass Index
COPD	Chronic Obstructive Pulmonary Disease
EELV	End Expiratory Lung Volume
EX	Exercise
EMG	Electromyography
FEV$_1$	Forced Expiratory Volume in One Second
FRC	Functional Residual Capacity
FVC	Forced Vital Capacity
GXT	Graded Exercise Test
Hz	Hertz
IC	Inspiratory Capacity
IRV	Inspiratory Reserve Volume
MEP	Maximal Expiratory Pressure
MIP	Maximal Inspiratory Pressure
MUAP	Motor Unit Action Potential
MVIC	Maximal Voluntary Isometric Contraction
R	Airway Resistance
RER	Respiratory Exchange Ratio
RR	Respiratory Rate
SCM	Sternocleidomastoid
TLC	Total Lung Capacity
VC	Vital Capacity
VCO$_2$	Carbon Dioxide Production
V$_E$	Minute Ventilation
VO$_2$	Oxygen Consumption
VO$_{2MAX}$	Maximal Oxygen Consumption
VO$_{2PEAK}$	Peak Oxygen Consumption
V$_T$	Tidal Volume
VT	Ventilatory Threshold

ACKNOWLEDGEMENTS

CHAPTER 1 – INTRODUCTION

Chronic Obstructive Pulmonary Disease (COPD) is an umbrella term used to describe lung diseases, such as emphysema and chronic bronchitis that cause expiratory airflow limitation.[1] Currently, COPD affects approximately 700 000 adults in Canada and despite being largely under-diagnosed and under-treated it has become one of the leading causes of mortality worldwide.[1,2] The Canadian Thoracic Society states that COPD is a common respiratory disorder; characterized by progressive, partially reversible airflow limitation and lung hyperinflation.[1] A decrease in maximal expiratory flow rate is the hallmark physiological abnormality associated with COPD.[3,4] Research demonstrates that chronic exposure to toxic particles or gases and resultant airway inflammation lead to this airflow limitation, hyperinflation, and to ventilation and gas exchange disruption, which are typical symptoms of COPD.[5]

The elastic properties of the respiratory system assist in generating the pressure difference needed to move air in and out of the airways.[4] In individuals with COPD, inflammation degrades the lung elastic fibers, reducing the expiratory driving force, which results in airflow limitation and in a decrease in minute ventilation (V_E). Consequently, there may be insufficient time to allow end expiratory lung volume (EELV) to decline to the natural relaxation volume.[4,6,7] As a result inspiration is initiated before expiration is complete and progressive air trapping occurs, inevitably leading to hyperinflation of the lungs, which contributes to dyspnea.[4,6,8] This is problematic during exercise because the increase in respiratory rate further exacerbates dynamic hyperinflation due to the preexisting airway narrowing and increased airway resistance.

Ultimately this increase in dynamic hyperinflation further limits the ability for tidal volume expansion, which can be assessed by performing an inspiratory capacity (IC) maneuver.[9,10]

There are numerous muscles that participate in ventilation. In individuals with COPD the primary concern lies with the inspiratory muscles. The diaphragm is the most important muscle of inspiration and accounts for approximately 70% of the tidal volume in healthy individuals.[11] In COPD, hyperinflation permanently changes the shape of the diaphragm, placing it in a mechanically disadvantaged position and thus limiting its force generating capacity.[12] It has been hypothesized that as a result, the respiratory system places greater demand on the scalenes and recruits accessory respiratory muscles such as the sternocleidomastoid (SCM) to assist in ventilation.[12] This becomes important during situations of increased ventilatory demand such as exercise. During times of increased ventilatory demand, an individual needs to generate higher flow rates in order to fulfill the increase in minute ventilation. Generating these higher flows is a power activity, which is typically accomplished by type II muscle fibers, which is believed to be the muscle fiber composition of the scalenes and SCM.[13] Therefore it is believed that in addition to the decrease in IC, muscle activation of the scalenes and SCM would increase and these things combined would increase the sensation of breathing discomfort.

An early manifestation of COPD is exercise intolerance, resulting from abnormal pulmonary function, impaired respiratory muscle function, breathing discomfort and leg muscle discomfort.[8,14] In a clinical trial conducted by O'Donnell and colleagues, 61% of participants terminated exercise because of breathing discomfort, 19% stopped due to a combination of breathing and leg discomfort and 18% were limited due to leg discomfort

alone.[14] Not only are people with COPD challenged with exercise tasks, but also their respiratory impairment leads to a difficulty with their activities of daily living.[15] Ironically, exercise is one of the most effective methods of reducing hyperinflation in individuals with COPD and therefore could possibly improve respiratory muscle function. In a study of COPD participants, aerobic training at or above anaerobic threshold was shown to significantly decrease lactic acid production.[16,17] This change was associated with a decrease in respiratory rate (RR) and hyperinflation as well as increased exercise capacity.[18] Presumably, this would also be associated with an improvement in their ability to perform normal tasks of daily living.[15] Unfortunately, little is known about the effects of exercise training on accessory respiratory muscle function and whether or not it improves with exercise training.

A recent study conducted by Duiverman et al.[19] assessed respiratory muscle activation during a graded exercise test (GXT). The researchers used surface electromyography (EMG) in both healthy and COPD participants, and found that increasing exercise intensity was associated with progressive increase in scalene muscle activation in both populations. The muscle activation in the participants with COPD started to significantly increase early in the exercise test, while that of the healthy population was delayed until the later stages. This progressive respiratory muscle activation suggests that exercise intensity has an influence on respiratory muscle activation in both healthy and COPD populations. Knowing the recruitment and amplitude of accessory respiratory muscle activation in response to increased workloads would be useful, particularly in people with respiratory disease, as it would allow researchers to assess the effects of interventions to decrease the work of breathing. The

study by Duiverman and colleagues normalized EMG activity using a logarithmic transformation of the ratio of the peak EMG activity during exercise and the peak activation at rest. One weakness of this technique is that it does not quantify the activation with respect to the muscle's maximal capacity. Another weakness of the Duiverman study is that the investigators did not describe the methodology in enough detail for it to be reproducible nor did they cite sources that would provide that information.

Alternatively, the amount of muscle activation with respect to the muscle's maximal capacity can be determined by normalizing the EMG with respect to the maximum voluntary isometric contraction (MVIC). This has been shown to be a reliable method that can be used to compare activation between muscles, tests, and individuals.[20-22] The MVIC has been used to assess other peripheral muscles,[23,24] but it has not been used to assess respiratory muscle activation. In order to be able to evaluate the role these muscles play in individuals with COPD, we need to first establish the amplitude of activation required of the SCM and the scalene during a graded exercise test and how this changes as exercise intensity increases. For the purpose of this study, the pattern of activation will be described in terms of, muscle activation amplitude. The methodology and results of this study can be used as a template to understand how accessory muscle function changes in individuals with respiratory disease and how the muscle activation changes with response to interventions.

1.1   Research Objective

The goal of the study was to use surface EMG to determine if increasing exercise intensity results in changes in accessory respiratory muscle (scalene and SCM) activation, described by EMG signal amplitude in healthy individuals. The main objectives investigated are as follows:

A. To determine if the %MVIC muscle activation for both muscles increase with an increase in exercise intensity.

B. To determine if there were differences in the %MVIC within each muscle and between rest, the ventilatory threshold (VT) and end exercise (EX).

C. To determine if there was a relationship between the %MVIC for each muscle and the participant's IC.

D. To determine if there was a relationship between the %MVIC for each muscle and the participant's perceived breathing exertion.

1.2   Research Hypothesis

A highly controlled exercise test was used to test whether accessory respiratory muscles demonstrated an increase in activation with an increase in exercise intensity. It was hypothesized that accessory respiratory muscle activation would increase with exercise intensity and the scalenes would demonstrate higher muscle activation amplitude compared to the SCM. Additionally, it was hypothesized that there would not be a relationship between the IC maneuver and the muscle activation in these healthy individuals. Typically we only see that relationship in individuals with COPD in whom the IC decreases with hyperinflation. The decrease in IC suggests more air trapping, which places greater stress on the diaphragm, which may result in the increase in

accessory respiratory muscle recruitment in order to fulfill the increase in ventilatory demand. However we do anticipate a relationship between the participants' perceived breathing exertion and muscle activation.

CHAPTER 2 – REVIEW OF THE LITERATURE

Chronic Obstructive Pulmonary Disease is a multidimensional disease, with several systemic manifestations and associations with a number of co-morbid diseases.[3,25] The structural changes occurring in the lungs, depending on the disease severity, place the respiratory system at a mechanical disadvantage making quiet breathing a difficult task. Consequently, in order to meet the ventilatory demand, the respiratory system places greater demand on certain respiratory muscles and recruits accessory respiratory muscles to assist in ventilation. This respiratory muscle recruitment and activation can be assessed using surface EMG.

Symptoms experienced by individuals with COPD, such as dyspnea, fatigue and chest tightness during rest and during activities of daily living can lead to an increasingly sedentary lifestyle and progressive deterioration in functional capacity.[3] With progressive inactivity, these individuals begin to experience poor cardiovascular function and loss of muscle strength.[3] This deterioration creates a continuous decline in quality of life and overall function.[3] Ironically, exercise is one of the most effective methods of reducing dyspnea in people with COPD. With an increase in exercise intensity, respiratory muscle activation increases, however little is known about the effects of exercise training on these muscles in people with COPD. Changes in respiratory muscle activation as a result of exercise training may be indicative of a decrease in energy expenditure and indirectly of less hyperinflation. Determining the most appropriate way to quantify the amplitude of respiratory muscle activation, will allow future research to assess the effects of interventions to improve respiratory muscle function and activity tolerance. Therefore, the purpose of this review is to discuss the mechanical function of the lungs associated

with COPD, how the mechanical dysfunction affects activity in individuals with COPD, the recruitment of accessory respiratory muscles during increased ventilatory demand and the ways to assess muscle recruitment and activation.

2.1 Healthy Lung: Structure and Mechanics

2.1.1 Lung Structure

The lung is an arrangement of complex, structures that interact to accomplish ventilation and gas exchange. Quiet breathing is defined as breathing during resting conditions. Air is directed from the atmosphere to the gas exchange region of the lung through a series of branching airways which become narrower, shorter and more numerous as they penetrate deeper into the lungs. The inspired air travels through the successive generations of bronchi from the conducting zone to the respiratory zone (Figure 1).[26] The conducting airways are structurally supportive and lead air to the gas-exchange region. The conducting airways have secretory capabilities and enlarge and narrow passively in response to lung volume change.[26,27] The respiratory zone includes the respiratory bronchioles and the alveoli. The alveoli participate in gas exchange, which allows the transfer of oxygen and carbon dioxide into and out of the blood.
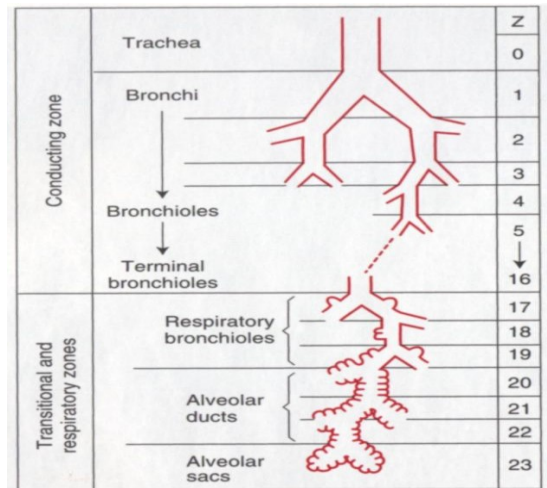
Figure 1. Airways in the human lung.[6]

The airways are supported by a sheath of connective tissue made up of elastic and collagen fibers. This sheath ends at the terminal bronchioles, at which point the bronchioles become directly attached to the lung parenchyma.[26,27] The parenchyma is an intricate mesh of interconnected elastic and collagen tissue that surrounds the alveoli and peripheral airways and contributes to the elastic recoil of the lung.[6]
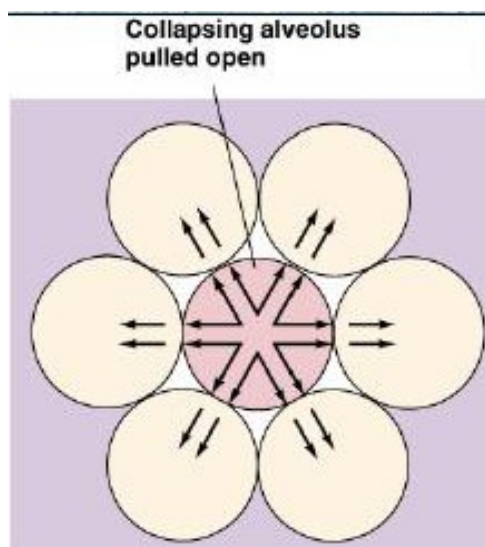
Figure 2. Mechanical tethering of the alveoli to maintain airway patency. (from http://images.slideplayer.us/1/221466/slides/slide_24.jpg)

The parenchyma works to oppose non-uniform changes in lung volume through a phenomenon known as interdependence or mechanical tethering: a tension that is transmitted through the airways to increase their diameter and prevent collapse (Figure 2).[6] When the pressure in one of the alveoli decreases, the surrounding alveoli begin to deform, stretching the elastic structures in the parenchyma in order to maintain the airway patency. The overall result is an outward force generated by the parenchyma acting to hold the alveoli open.[6]

2.1.2 Lung Mechanics: Resistive Properties of the Lungs

Impedance to airflow in the lungs has a resistive and elastic component. Typically the resistive component is dominated by the airways, while the elastic is dominated by a lung volume component. During quiet breathing airflow in the peripheral airways is laminar, which means that it is parallel to the airway walls. According to Poiseuille's law, laminar airflow ($\dot{V}$) is directly proportional to the pressure difference (P) between the airways, times the fourth power of its internal radius (r), and inversely proportional to eight times the airway length (l) multiplied by the viscosity of the air (n). Pi (π) is a constant of 3.14.

$$\dot{V} = \frac{P\pi r^4}{8nl}$$

Equation 1. Poiseuille's law.

Resistance is the measure of impedance to airflow and indirectly determines the force required to breathe. Rearranging equation 1 demonstrates that airway resistance (R) can be calculated as the pressure change divided by the flow and is markedly affected by changes in the airway radius (r), viscosity of the air (n) and length of the airways (l).

$$R = \frac{8nl}{\pi r^4}$$

Equation 2. Rearrangement of Poiseuille's law.

Since length and viscosity do not usually change, airway resistance is very sensitive to airway radius. In health the major contribution to airway resistance lies in the larger airways. Respiratory bronchioles have smaller individual radii, but the parallel arrangement of these small airways results in a larger total cross sectional area creating little resistance to airflow. In COPD, the smaller airways contribute more to airflow resistance because of a reduction in their luminal size.

2.1.3 Elastic Properties of the Lung

The elastic properties of the lung are described in terms of elastance, which refers to the elastic recoil of the lung. Elastic recoil is the tendency of the lung to recoil to its original dimensions after distension and reflects the relationship between pressure and volume.[26,27] In health there is a great deal of elastic recoil in the lung and often it is

described in terms of compliance. Compliance is the ease with which the lungs can be stretched or inflated and is inversely related to elastance (equation 3). An analogy of this relationship is a coiled spring, the more difficult it is to stretch the spring the greater the ability to snap back (greater elastic recoil or low compliance). Therefore a lung that is stiff is more difficult to inflate however, has a greater ability to recoil.

$$C_L = \frac{\Delta V}{\Delta P}$$

Equation 3. Lung compliance.

The pressure-volume curve (Figure 3) can be used to describe the relationship between the changes in pressure needed to achieve a given change in volume in the respiratory system or its components; the lung and chest wall.[4] At 0-pressure of the respiratory system (P_{rs}) or functional residual capacity (FRC) there is no air moving in and out of the lungs, because the chest wall and lung forces are equal and opposite. Therefore, there is no tendency for the respiratory system to inflate or deflate. As the air moves into the lungs there is an increase in elastic recoil. For volumes between 20-80% vital capacity (VC) in figure 3, the lung is very compliant, so a small pressure change results in a large increase in volume. As the lung approaches total lung capacity (TLC) the pressure required to change the volume increases markedly.
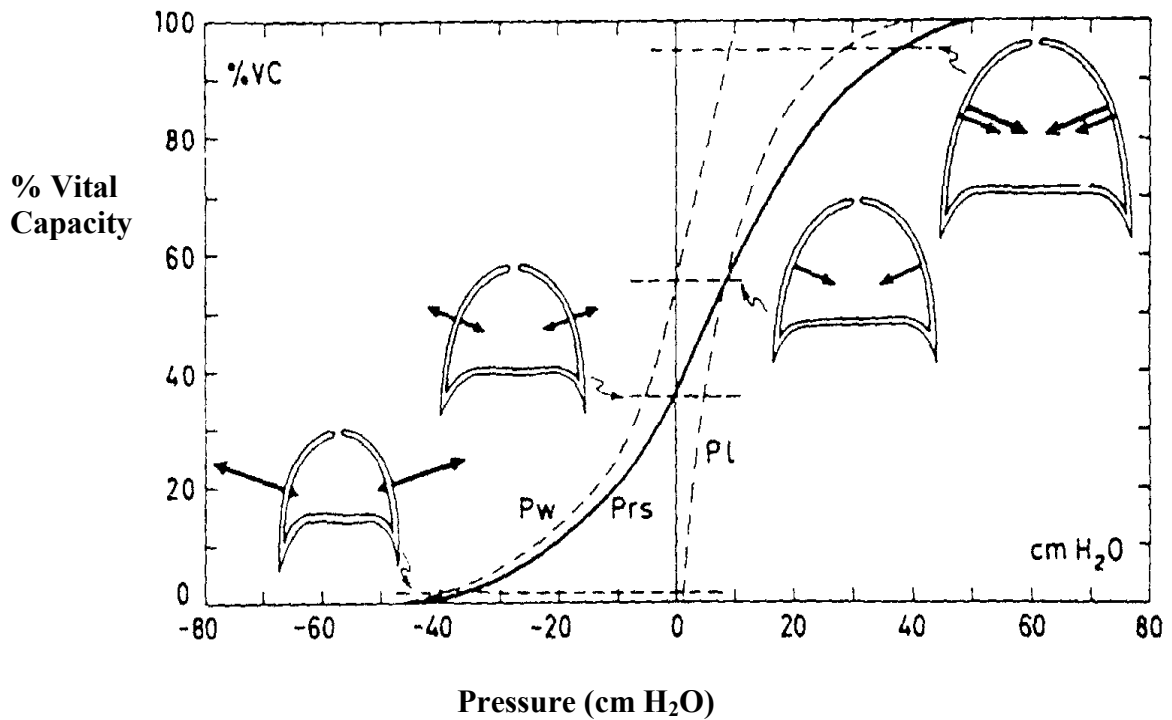
Figure 3. Pressure-volume curve of the respiratory system. [P_{rs} – pressure of the respiratory system, P_w – pressure of the chest wall, P_l – pressure of the lung] (from http://or7.chat.ru/Image5.gif)

2.2 Chronic Obstructive Pulmonary Disease

2.2.1 Pathology

The Canadian Thoracic Society defines COPD as a common respiratory disorder; characterized by progressive, partially reversible airflow limitation and lung hyperinflation.[1] Airflow limitation may result from an increase in airway resistance, which causes a decrease in maximal expiratory flow rate; the hallmark physiological abnormality associated with this disease.[3,4] Chronic Obstructive Pulmonary Disease includes both emphysema and chronic bronchitis. Emphysema is a condition defined by enlargement of the airspaces distal to the terminal bronchioles, which decreases lung compliance.[3,5] Chronic bronchitis is a condition in which airway inflammation increases mucus secretion and results in a persistent cough. This inflammation ultimately increases

the resistance through the airways, making airflow more difficult.[5,28] Thus, both pathologies impair normal function of the respiratory system.

Chronic exposure to toxic particles or gases and resultant airway inflammation leads to airflow limitation and poor ventilation and gas exchange, and is considered the leading cause of COPD.[5] This exposure results in the release of toxic oxygen radicals, proteases and elastases that damage tissue.[2,29,30] Gradually, elastases destroy the elastin of the parenchyma.[29,31] Loss of elastin reduces parenchymal attachments, which makes the small airways more amenable to collapse.[32] Particulate exposure also leads to chronic inflammation, which promotes mucus hypersecretion and smooth muscle hypertrophy,[5,28] both of which contribute to airway narrowing. In addition, the inflammatory response also triggers an abnormal tissue repair process in the small airways. This results in scar tissue and a significant increase in airway smooth muscle, ultimately decreasing airway diameter, which increases airway resistance.[33-35] The resultant response to the foreign gases and particles, thereby limits airflow, which consequently impacts lung mechanics.

2.3   Mechanical Abnormalities Associated with COPD

2.3.1   Airflow Limitation

Expiratory airflow limitation is the principal mechanical dysfunction in individuals suffering from COPD.[3,4] Given that the airway radius decreases during exhalation, the addition of smooth muscle hypertrophy and accumulation of mucus effectively further decreases the airway diameter and increases the resistance to flow in COPD.[3-5,31] The increase in expiratory flow resistance leads to hyperinflation that affects the inspiratory phase of breathing.

2.3.2 Hyperinflation

Structural instability of the airway walls, combined with smooth muscle hypertrophy and mucus hypersecretion, impedes expiratory flow so there is insufficient time to allow the end expiratory lung volume (EELV) to return to its natural relaxation volume.[4,6,7] This increase in the EELV is called hyperinflation. As a result subsequent inspiration begins at a higher lung volume and the IC is reduced. Hyperinflation places the lung on a less compliant portion of the P-V curve. The hyperinflated lung also puts the chest wall in a more expanded position, causing it to move to a less compliant portion on its P-V curve. This makes the respiratory system as a whole less compliant, requiring higher expanding pressures, generated by the respiratory muscles, to accomplish the same tidal volume change.

2.4 Exercise Intolerance and Dynamic Hyperinflation

The decline in lung function in COPD is associated with physical inactivity, which is related to increased rates of hospitalization and mortality.[36] Individuals with higher levels of physical activity generally report better health-related quality of life compare to an inactive individual.[37] The presence of lung hyperinflation at rest means that the individual's ability to increase ventilation in a demanding situation such as exercise is greatly reduced because there is a decreased ability to increase tidal volume, which is required to increase minute ventilation.[10] Consequently, an early manifestation of COPD is exercise intolerance, resulting from interactions between pulmonary function, gas exchange limitations, and muscle fatigue.[8,14] During activity the individual experiences dynamic hyperinflation, defined as a temporary and variable increase in an

individual's $EELV.^{38}$ Minute ventilation (V_E) must increase to support gas exchange demand. The increase in V_E is typically accomplished by an increase in both RR and V_T. In COPD, a reduced IC during exercise limits the increase in V_T; therefore the only way to increase V_E is to increase RR. Inspiratory and expiratory time decrease in order to accommodate the increase in RR and this accentuates the hyperinflation until the individual approaches their TLC, at which point no increase in V_T can occur. Dynamic hyperinflation resolves once the activity has been stopped and the RR decreases.

2.5   Respiratory Muscles in Healthy Conditions

The respiratory muscles generate the force needed to expand the thoracic cavity, creating a pressure difference that allows air to move in and out of the lungs.[6,26,27] The primary respiratory muscles, the diaphragm and intercostals, and the accessory respiratory muscles, the scalenes and SCM accomplish ventilation. The diaphragm is the most important muscle of inspiration, generating approximately 70% of the force needed to accomplish inspiration in healthy individuals at rest.[11] The intercostals connect adjacent ribs and assist in ribcage expansion when they contract. The accessory respiratory muscles assist the primary respiratory muscles during periods of increased ventilatory demand. The scalene and SCM elevate the first two ribs and the sternum, respectively. There is some controversy; however, as to whether these muscles should be considered "accessory" and this issue will be discussed in the next section.

2.5.1 Diaphragm

The diaphragm is described as an endurance muscle contracting rhythmically for low tension, high repetition activity.[11] It is dome shaped and separates the thoracic cavity from the abdominal cavity (Figure 4).[27] The diaphragm attaches to the lumbar vertebrae (L1-L3) of the spine as well as the lower six ribs (rib 7 – rib 12).[27,39] The zone of apposition is the point where the diaphragm comes into direct contact with the internal aspect of the ribcage.[27] At this point there is no lung present, just the parietal pleura. The zone of apposition has anatomic importance because it directs diaphragmatic tension and helps with chest wall expansion.
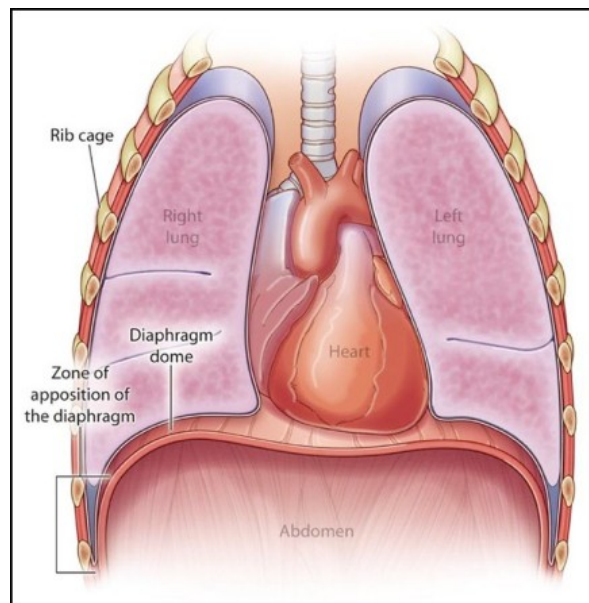
Figure 4. Anatomy of the thoracic cavity. Highlighting the diaphragm and the zone of apposition. (from http://thorax.highwire.org/content/54/5/458/F3.large.jpg)

During inspiration the diaphragm descends like a piston in the thoracic cavity, which acts as the cylinder.[27] As inspiration occurs the diaphragm pushes downward into the abdominal cavity, resulting in outward displacement of the abdominal wall and a

decrease in the height of the zone of apposition.[12,40] This contraction generates negative pressure in the thoracic cavity consequently drawing air into the lungs.[6,11] Abdominal muscle tension increases and opposes descent of the diaphragm. The resulting increase in intra-abdominal pressure is transferred to the chest wall via the zone of apposition and results in expansion of the ribcage.[26,27] Contraction of the diaphragm without the intercostal muscles produces expansion of the distal aspect of the thoracic cavity.[27]
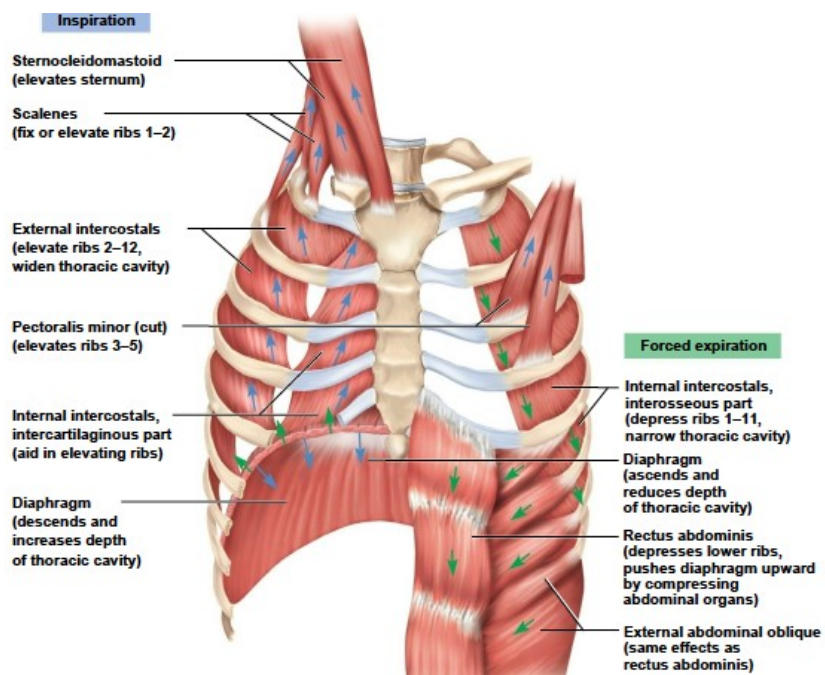
Figure 5. Muscles of Respiration (from http://classconnection.s3.amazonaws.com)

2.5.2   Intercostal Muscles

The intercostals muscles run between the ribs and when active they create uniform rib cage expansion.[41,42] The external intercostals run from the tubercules of the inferior border of the ribs to the chondrocostal junction of the superior border of the rib below.[39] When they contract the ribs elevate, increasing the dimensions of the thoracic

cavity.[27,39,43] In contrast, the internal intercostals extend from the sternocostal junctions of the superior border of the rib to the inferior border of the rib above.[39] They contract during exhalation to pull adjacent ribs together to decrease the dimensions of the thoracic cavity.[39,43] The parasternal intercostals are located between the sternum and the chondrocostal junctions, their action assists inspiration.[39,43] The parasternal intercostals stabilize the rib cage, opposing its tendency to collapse slightly under the influence of the negative pressure generated by the movement of the diaphragm.

2.5.3   Accessory Respiratory Muscles

The accessory respiratory muscles include the SCM, scalene, pectoralis, and trapezius.[27,43] These muscles assist the diaphragm during times of increased ventilatory demand.[27,43] The SCM originates from the manubrium of the sternum and the medial third of the clavicle.[39] It inserts at the mastoid process of the temporal bone and lateral half of the occipital bone.[27,39] The primary function of the SCM is lateral flexion and rotation of the neck and head.[39]

The scalenes are a group of three pairs of muscles that originate from the transverse processes of the cervical vertebrae of C2-C7 and insert onto the first and second ribs.[39,44] Traditionally the scalene muscles were seen as an accessory respiratory muscle, assisting only during times of ventilatory demand, however EMG studies have established that in healthy humans these muscles consistently contract during inspiration and may in fact be a primary respiratory muscle.[44] In 1996 De Troyer and colleagues used EMG to demonstrate that the scalene muscles were active throughout quiet inspiration in humans and that their activation was increased with ventilatory demand.[41]

A later study conducted by Legrand et al. determined that the scalene muscles have a greater ability to change pleural pressure during increased ventilatory demand than the SCM, which supports the assertion that the scalenes are primary muscles of respiration.[42]

2.6   The Effects of COPD on the Respiratory Muscles

Changes in the lung structure associate with COPD, particularly hyperinflation, result in changes in the mechanical function of the respiratory muscles. The presence of lung hyperinflation forces the inspiratory muscles to work at shorter than normal length, putting them at a less than optimal point on the force-length curve.[40] This reduces their ability to generate enough pressure change to inflate the lungs and requires a greater than normal muscle excitation.[27] The increased EELV associated with COPD decreases the zone of apposition and increases the area of the rib cages.[6,12,40,45] Since the diaphragm works like a piston, a shorter zone of apposition results in a decreased ability for the zone to transmit the tension from the diaphragm into the chest wall for expansion.[46,47] At this shortened length, the tension the diaphragm is able to produce is much less than normal.[46] This decreases the diaphragm's ability to generate pressure change and expand the thoracic cavity.[45] The mechanical consequences of hyperinflation decreases the force generating capacity of the diaphragm and at the same time increases the force generating demands on the accessory respiratory muscles.[43,46,47] As a result accessory respiratory muscles are recruited sequentially to augment respiratory muscle force.[46]

2.6.1 Recruiting Respiratory Muscles

During increased ventilatory demand, such as exercise, there is progressive recruitment of accessory inspiratory muscles.[27,43,47] These accessory respiratory muscles tend to be recruited according to their ability to generate a pressure change.[48] A study by Legrand et al. determined that the scalene muscles have a greater ability to create a pressure change than the other well-known accessory muscle of respiration, the SCM.[42] Electrical stimulation of the scalenes in dogs produced a large cranial displacement of the ribs and sternum, an increase in anteroposterior diameter of the rib cage and an increase in lung volume. [49] Others have suggested that the muscles would have the same function in humans.[27,46,47,50] Traditionally the scalenes were considered accessory respiratory muscles, however EMG studies using needle electrodes have established that in healthy humans these muscles contract even during quiet breathing and are further activated during increased ventilatory demand.[19,42,51]

The SCM activity has only been reported during situations of increased ventilatory demand. When the SCM is recruited as an accessory muscle of respiration, the head is held fixed by the upper fibers of the trapezius muscle, allowing the SCM to contract and elevate the upper portion of the rib cage.[44,52] This rib and sternum elevation has been demonstrated in animals[49] and in humans who take larger than normal resting tidal volume breaths.[53] The SCM has also been shown to be active in individuals with high quadriplegia, who do not have a functioning diaphragm,[54,55] meaning these individuals need to recruit accessory respiratory muscles such as the SCM in order to breathe. Minimal assessment of SCM activity has been performed due to the small size of the muscle and the possibility of crosstalk. In fact only one study has assessed SCM

activity using EMG during increased ventilatory demand.[21] Duiverman and colleagues however, suggested that, during increased exercise intensity, the scalene muscle activity in their participants might have contained SCM activity, which encourages research to investigate the influence that the SCM has during ventilatory demand.[19]

2.7   Assessment of Respiratory Muscle Force

Respiratory muscles generate pressure differences that drive ventilation. A variable that influences pressure is lung volume, which affects the respiratory muscles length-force relationship.[47,56] In the case of the diaphragm, at lung volumes above FRC, the diaphragm and other inspiratory muscles are shortened and their contractile force is reduced. In contrast, expiratory muscle force generation is reduced at low lung volumes. Respiratory muscle strength is commonly measured by assessing the pressure changes generated during the Muller and Valsalva Maneuvers.[47,56,57] The Muller Maneuver measures the maximal inspiratory pressure (MIP) at the mouth. Typically it is measured at residual volume against a closed airway. The Valsalva Maneuver assesses the maximal expiratory pressure (MEP). Mouth pressure is measured at TLC as the individual forcefully exhales against a closed shutter. The maneuvers are performed at the extreme lung volumes because they represent the optimal length-tension ratio for the respective respiratory muscles.[56]

Currently MIP and the MEP are the most widely applied methods for assessing respiratory muscle strength.[57,58] These non-invasive tests are easy to perform, highly reproducible, and normative values are available for all age ranges.[57,58] They are; however, effort dependent and low values may reflect lack of motivation rather than poor

muscle strength.[58] These tests are also unable to differentiate each muscle's strength or determine which muscles are activated during the maneuver.

2.8   Electromyography: Evaluation of Muscle Activation

Muscle contraction depends on electrical activation of the muscles, which can be assessed using EMG from surface or indwelling electrodes.[21] While electromyography is not widely used clinically, it is a common method for understanding and analyzing muscle responses during fundamental movements.[59] An advantage of using EMG as compared to a MIP or MEP is that EMG can provide information on the contributions of each muscle, while MIP or MEP can only assess the sum of the contributions of all respiratory muscles. The electrical activity recorded is the sum of the motor unit action potentials (MUAP) in the pick-up zone, which can then be processed and analyzed to evaluate the neuromuscular activity occurring in the muscle.

2.8.1   Generation of the Electrical Potential:

Excitation-contraction coupling describes the physiological process where an electrical discharge at the muscle initiates a chemical event and ultimately a muscle contraction. This is generated via a complex series of events. During a resting state there is an ionic equilibrium between the intra and extra cellular space called a resting potential. A concentration gradient is maintained through an ion pump with a greater concentration of sodium outside the cell and a greater concentration of potassium inside, creating a negative potential across the cell membrane of approximately -80 to -90mV intracellular. When a motor nerve is stimulated and the associated electrical impulse

arrives at the motor endplate, a voltage-activated channel is opened. This opening increases membrane permeability to sodium (Na+), creating an intracellular influx of sodium and a depolarization of the muscle membrane. Depolarization does not last long and quickly the muscle membrane begins repolarization. It is during this stage that the sodium channels are no longer being activated and additional potassium (K+) channels are opened to move the potassium out of the cell. The resting potential is restored via an active sodium/potassium ion pump.

As the nerve action potential reaches the motor end plates, acetylcholine is released into the synaptic cleft and binds to their adjacent receptors on the post-synaptic muscle fiber, which triggers a new wave of electrical activity. The action potential continues along the muscle cell membrane and into invaginations of the membrane called transverse tubules where depolarization stimulates the release of Calcium ions (Ca++) from the sarcoplasmic reticulum into the sarcoplasm. The calcium binds to the troponin, changing its conformation and causes a shift in the tropomyosin. The tropomyosin movement uncovers the binding site on the actin head, facilitating a cross-bridge formation between the actin and the myosin filaments and a subsequent contraction of the muscle fiber. In order to stop the muscle contraction, active transport moves the calcium ions back into the sarcoplasmic reticulum.

Electromyography is an evaluation of the electrical signals being emitted during muscle activation. It provides a view of the changes in membrane potentials associated with propagation of a muscle unit action potential (MUAP). The electrodes measure the summation of all MUAPs (figure 6) that occur within the pick-up region between the two

electrodes.[21] The MUAP propagates an electrical signal across the muscle fibers and it is that electrical signal that is recorded by the EMG.[21]
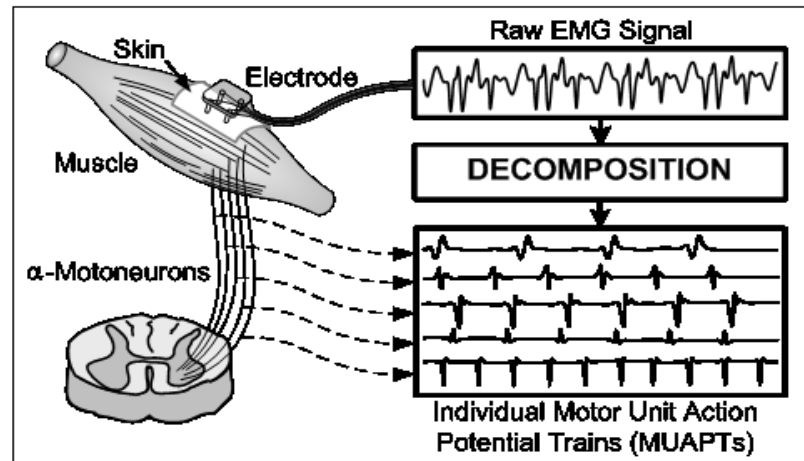
Figure 6. The pick-up MUAP by the electrodes. (from http://www.bu.edu/iss/files/2010/12/EMG_signal_decomposition.png**)**

2.8.2 EMG-Force Relationship:

Previous work using EMG to investigate respiratory muscle activation has used muscle force to determine a correlation between EMG and force, in an attempt to determine the extent to which each muscle participated in the change in pressures in the thoracic cavity.[60] The EMG literature however is conflicting, with some research supporting linear relationships and other research supporting nonlinear relationships between the EMG signal and the force generation.[61-64] This inconsistency may be due to differences in the size of the muscle being assessed,[64] the fiber composition of the muscle,[62] and selective recruitment of the fiber types[62] to name a few. For the purpose of this study we will not be collecting data that will allow us to determine if one muscle is doing more work than the other, because work is the force used to move an object

through a distance. Rather we want to establish how much muscle activation is required to achieve a given ventilatory demand. For instance, if the scalenes have a higher %MVIC at end EX compared to the SCM, we cannot say that it is contributing more force to meet the ventilatory demand; instead, we can say the muscle is more activated to meet the ventilatory demand.

2.9   Why Investigate Impaired Respiratory Muscles Function?

In individuals with COPD respiratory muscle function is impaired, which undoubtedly contributes to exercise intolerance.[8,14] Not only are these individuals challenged by exercise tasks, but also their respiratory impairment leads to a decrease in their activities of daily living, which is significantly correlated with a decrease in quality of life.[15,37,65] There are a number of interventions that can be used to decrease hyperinflation and possibly improve respiratory muscle function. Exercise and medication are the most common. Aerobic exercise is one of the most effective methods of reducing hyperinflation in individuals with COPD and could possibly improve respiratory muscle function. Exercise training, has been shown to decrease lactic acid.[16,17] This change was associated with a decrease in RR and hyperinflation as well as increased exercise capacity.[18] Presumably this would also be associated with an improvement in a person's ability to perform normal tasks of daily living.[15] Unfortunately, little is known about the effects of exercise training on the accessory respiratory muscle function and whether or not it improves with exercise training or other medical interventions.

Studies by Chiti et al., and Breslin et al. have shown that EMG is an effective measurement technique to assess accessory respiratory muscle activity in healthy

individuals during increased ventilatory demand. Specifically, their research[19,66,67] has suggested that as exercise intensity increases, so does the muscle activation of the scalenes in healthy individuals. Duiverman and colleagues were able to demonstrate similar results in individuals with COPD.[19]

Duiverman et al.[19] assessed accessory respiratory muscle activation during a graded cycle ergometer exercise test. Using surface EMG in both healthy and COPD participants, the researchers found that with increased exercise intensity there was a progressive increase in scalene and intercostal activation in both populations. The muscle activation in the participants with COPD started to significantly increase after stage two (stages increased by 5 or 10watts/min) of the exercise test, while activation in the healthy population was delayed until the later stages (stages increased by 25watts/min). This progressive accessory respiratory muscle activation suggests that exercise intensity has an influence on the recruitment of respiratory muscle activation in both a healthy and COPD population. Knowing the pattern of accessory respiratory muscles activation in response to increased workloads would be useful, particularly in people with respiratory disease, as it would allow researchers to assess the effects of interventions to decrease the work of breathing. The study by Duiverman and colleagues assessed EMG activity using a normalization technique that does not relate to the amount of activation relative to a maximum. Instead the group performed a logarithmic transformation of the ratio of the peak EMG activity during exercise and the peak activation at rest. Unfortunately the investigators did not describe the methodology in enough detail for it to be reproducible nor did they cite sources that would provide that information. This is particularly problematic because the technique does not quantify the activation with respect to a

known maximum and therefore it is not possible to describe how much the muscle is activating to complete the given exercise stage.

2.9.1 Normalization:

Electromyography data is highly sensitive to factors such as electrode placement, electrode application, subcutaneous fat, and muscle fatigue, just to name a few.[68,69] Normalization is the process that attempts to control for this variability by expressing the EMG signal in terms of a signal obtained during a standardized and reproducible condition[68,70-72] There are different normalization approaches that can be used to express the EMG signal, and choosing this normalization approach is to a large extent influenced by the outcome variable or what the researcher is trying to investigate.[22,69] Yang and Winter[22] compared four different normalization approaches to see the effects of each approach on intersubject variability. These approaches included normalizing to: average force recorded during an isometric contraction, the peak EMG signal during the task, mean EMG signal during the task, and 50% of the MVIC. They determined that although the peak and mean EMG signal during the task reduced the inter-subject variability, it does not provide information on the degree of muscle activation with respect to the individual's capacity that is required for the task.[22] Therefore it is unknown whether the muscle needs to activate to 15% or 95% of its maximum capacity in order to complete the task, which would be determined with an MVIC normalization approach.

To account for this unknown amount of activation one can perform a maximum voluntary contraction where a participant attempts to recruit all motor units by performing a standardized task.[20,21,59,68,73,74] By normalizing the EMG values as a

percentage of a maximum one can make comparisons between individuals and muscle sites. The validity of this technique has been questioned with researchers believing that some populations may have a difficulty maximally recruiting all motor units.[22,75] However, it is difficult to interpret absolute EMG measures. Thus, guidelines suggest that some normalization protocol be performed.[76] The MVIC is an accepted normalization procedure and has been shown to increase intra-class correlation coefficient between trials, increasing the reproducibility of the data compared to the peak or mean normalization approach.[59,68,70] Maximal voluntary isometric contractions are also feasible for older adults.[77,78]

Standardized positions for multiple muscles have been developed from previous research and muscle function testing procedures to ensure that a maximal contraction can be produced.[79] Research conducted by Vera-Garcia et al in 2009 investigated the effectiveness of several MVIC exercises for trunk muscle activation, and found that no single test was superior for obtaining maximal activity.[80] The trunk muscles lie between the proximal chest and the distal pelvis and are responsible for spinal stability, as well as provide the ability to flex, bend and rotate. Although neck muscles provide the same functionality, they are not responsible for maintaining and supporting the central part of the body. Vera-Garcia found that the position of the pelvis played a major role in creating the maximal MVIC contraction, because the muscles are attached to the pelvis.[80] In contrast to the trunk muscles; the neck muscles are not affected by distal portions of the body.[81] No previous work was found that has used an MVIC to normalize scalene and SCM activation, therefore it unknown what type of contraction would elicit a maximum. It has been encouraged that in attempts to elicit a maximal contraction, a series of

different exercises for each muscle should be used, unless there is a previously determined superior MVIC. This has not been determined for the scalenes and SCM.[80,82,83]

We know that muscle activation in the scalene and SCM increases with exercise intensity in both healthy individuals and those with COPD[19]; however, this activity has been described with respect to resting levels of activation. It does not express activation with respect to the muscles' capacity. Therefore the aim of this study was to qualify muscle activation amplitude of the scalenes and SCM, in healthy individuals during a symptom limited GXT. This groundwork can be used to understand changes in muscle activation in people with COPD. For instance, hyperinflation in people with COPD impairs diaphragm function, which may require the accessory muscles of respiration to increase their activity earlier and to a greater extent than is seen in healthy individuals. Such changes can only be properly appreciated when compared to function in healthy people. Furthermore, characterizing accessory muscle activation in people with COPD will allow us to assess the effect of interventions that might improve their function.

CHAPTER 3 – METHODS

3.1 Participants and Recruitment

The study population included 20 healthy adults (convenient sample). Participants were recruited from the COLD Study (healthy participants) during May of 2015. The researcher recruited participants from a respirology database of participants who agreed to be contacted for future research studies. The recruitment process is shown in Figure 7.
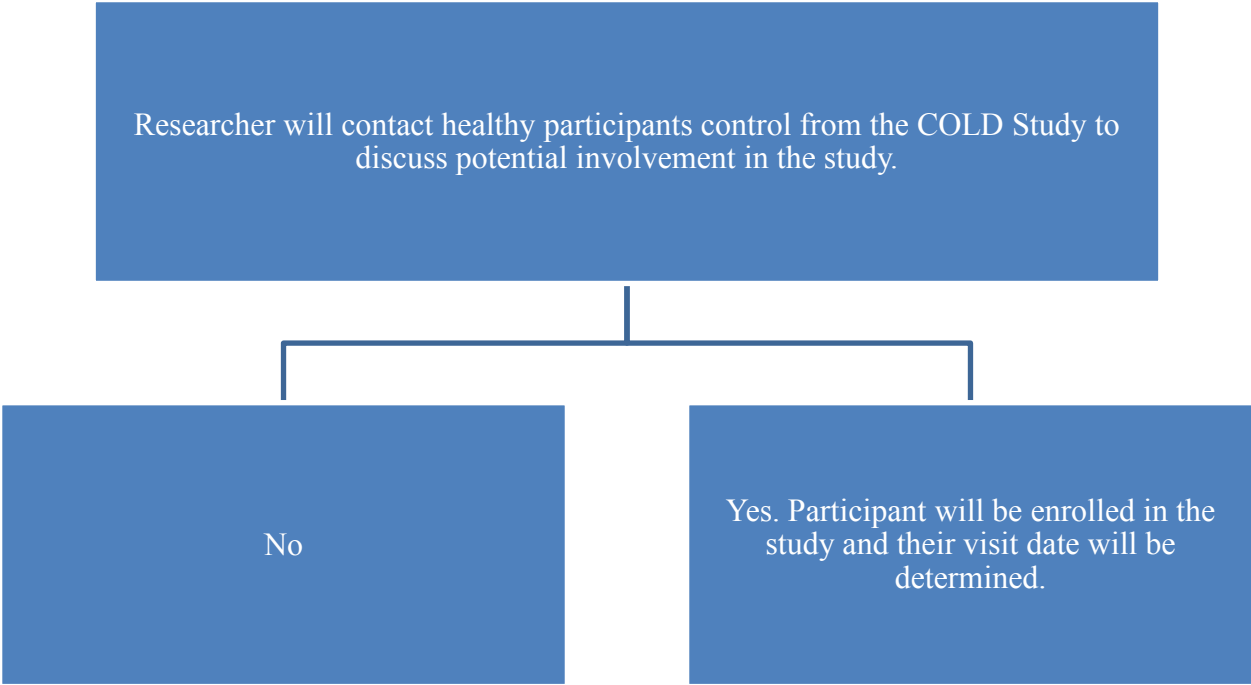
Figure 7. Recruitment flow chart for the participants.

Inclusion and exclusion criteria were addressed and confirmed during the initial discussion with the potential participants (Appendix A). The inclusion and exclusion criteria are as follows:

Inclusion:
- Male or female
- ≥ 40 years of age

Exclusion:
- Comorbidities that would limit their ability to:
    - Exercise on a recumbent bike
    - Perform a maximal contraction of their neck muscles
    - Make the exercise unsafe
- Use of supplemental oxygen

3.2 Ethics, Recruitment and Consent

The study was approved by the NSHA Research Ethics Board at the QEII Hospital prior to recruitment (NSHA-RS/2016/018). The researcher discussed the project with potential participants over the phone and answered any questions or concerns. If the potential participant was interested in the study, an appointment was scheduled for their study visit. Consent forms were signed at the study visit prior to beginning any data collection.

3.3   Procedures

Participants attended one study visit. An outline of the experimental procedures is shown in Figure 8.
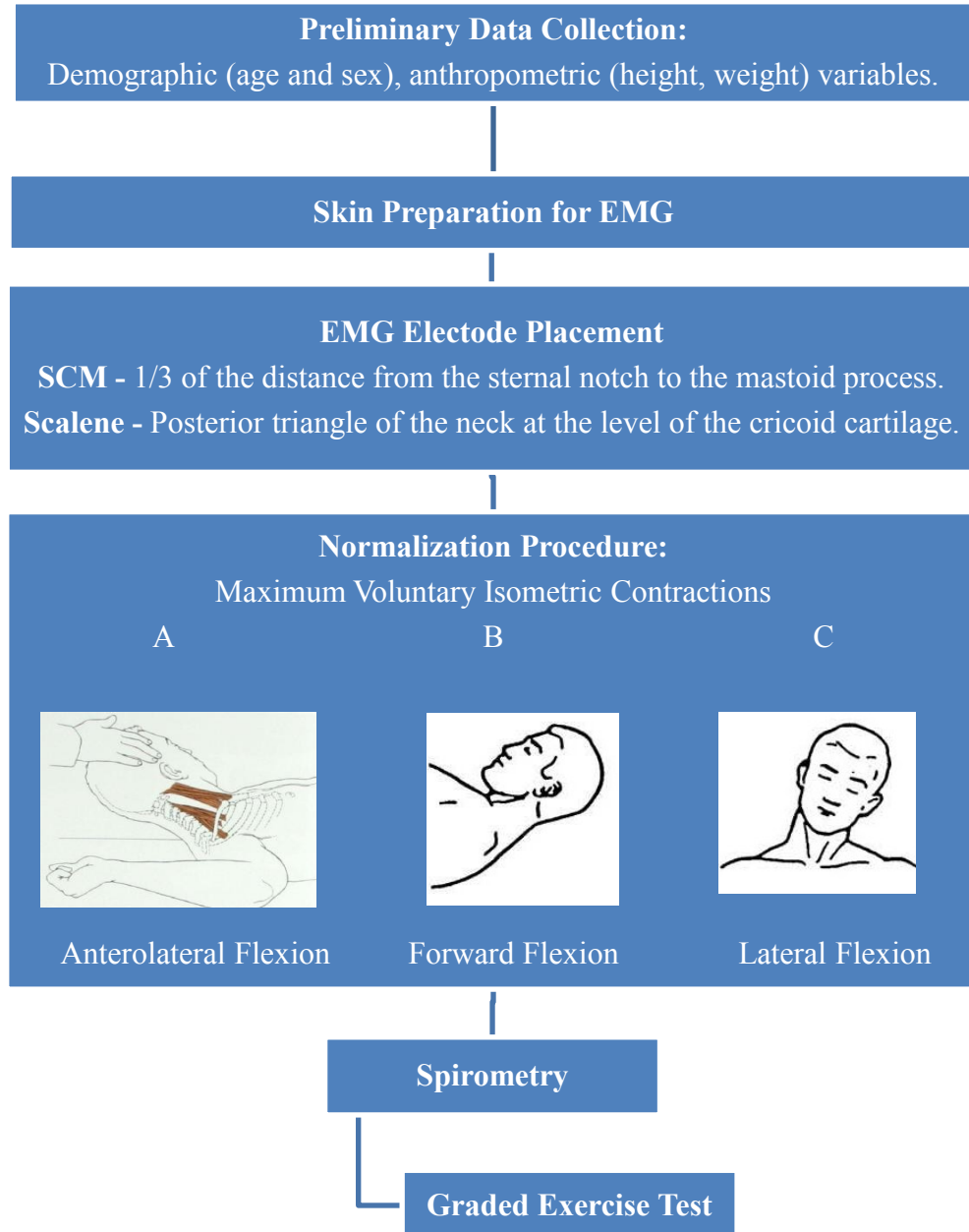
Figure 8. Visit Outline

Table 1. Exercise Test Procedure and Data Collection Points

Tasks	EMG: Skin Prep	EMG: Electrode Placement	EMG: MVIC	GXT: Spirometry	GXT: Seated rest	GXT: Quiet breathing	GXT: Unloaded Pedaling	Stage 1 (0-30 sec)	Stage 1 (30-60 sec)	Stage 2 (0-30 sec)	Stage 2 (30-60 sec)	Stage 3 (0-30 sec)	Stage 3 (30-60 sec)	Stage 4 (0-30 sec)	Stage 4 (30-60 sec)	Stage …until peak (0-30 sec)	Stage …until peak (30-60 sec)	Recovery (2min unloaded pedaling)
Time (min)	5	5	20	10	2	3	1	1		1		1		1		< 14		10
Ventilatory Parameters					X	X	X	X	X	X	X	X	X	X	X	X	X	Stop at 5 minutes
IC Maneuver **						X X X					X				X		X	X
Breathing Exertion Borg Scale (Appendix B)						X			X		X		X		X		X	X
Leg Discomfort Borg Scale (Appendix B)						X			X		X		X		X		X	X
EMG (20sec data collection)					X		X		X		X		X		X		X	X
Reason for stopping the test (Appendix C)																		X

Notes: X – time point of data capture; X X X – three maneuvers; ** – performed on even stages
Abbreviations: EMG, electromyography; IC, inspiratory capacity; MVIC, maximal voluntary isometric contraction

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3.3.1 EMG Measurements

Ag/AgCl single use disposable surface electrodes (5mm diameter, Duo-Trode) were used. The electrodes were placed on the SCM and the scalene using standardized placements established in the preliminary research for this study (Appendix E) (Table 2) and adjusted based on individual anthropometrics, palpation and a series of resistance exercises isolating the muscles.[84] The muscles on the right side of the neck were used and the electrodes were placed in a bipolar configuration, in line with the muscle fiber orientation. Once the electrode position was determined, standard skin preparation was performed by cleaning the electrode site using an alcohol/water solution to reduce skin impedance and therefore improve signal conduction.[84,85]

Table 2. Electrode Placement [81]

SCM	Scalene
1/3 of the distance from the sternal notch to the mastoid process.	Posterior triangle of the neck at the level of the cricoid cartilage.

No data was collected in the 10 minutes immediately after electrode placement to ensure that the ratio of skin-electrode impedance to the input impedance of the amplifier is as small as possible.[85] In accordance with the SENIAM guidelines, it is recommended that this ratio be less than 1%.[84,85] A Bortec amplifier with a resistance of 10 GΩ was used. This is deemed sufficient to produce the desired ratio. Prior to any data collection, a subject bias and a noise trial were performed, where the subject was supine and completely relaxed.
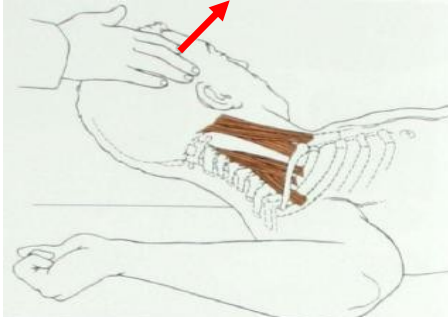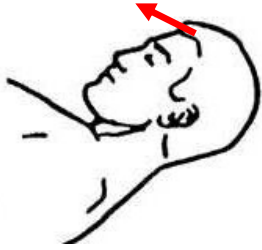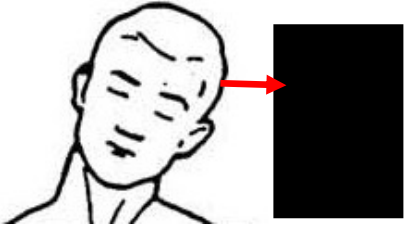
Sampling rate refers to the frequency with which the signal is collected and it is expressed in Hz. The sampling rate needs to be high enough so as to provide a signal

without any attenuation or aliasing.[86] According to the Nyquist Theorem a signal must be sampled at a frequency that is greater than twice the highest frequency of the signal of interest.[86] The highest frequency content for EMG is 500Hz; a sampling rate of at least 1000Hz is required to avoid signal loss.[21,73] Previous research investigating respiratory muscle activity with surface EMG used a sampling rate of 2000Hz, which provided a good representation of the signal in both healthy individuals and individuals with COPD during conditions of increased ventilatory demand.[19,87]

3.3.2   EMG Normalization Procedure

Prior to the exercise test the participants were asked to perform a series of standardized MVIC maneuvers for EMG normalization purposes.[20] For the purpose of this study, based on my preliminary research, three MVIC maneuvers were chosen from previous work[81] and muscle function-testing procedures.[79] The MVIC maneuvers for the scalene and SCM muscle included anterolateral neck flexion (Figure 8-A),[79] forward flexion (Figure 8-B) and lateral flexion (Figure 8-C). For all the MVIC maneuvers the participant was supine on a table, with their elbows bent and hands beside their head. To ensure safety and for resistance, the participants were stabilized with non-elastic straps across their forehead, hips and ankles. The researcher also provided feedback on the direction in which the participant should resist by placing their hand on the participants forehead. Table 3 shows each of the MVIC maneuver.

Table 3. MVIC Maneuvers, with the direction of the contraction.

Anterolateral Neck Flexion		The participant rotated their neck and laterally flexed, with the non-testing side of their neck on the table. The individual resisted against the non-elastic strap in forward flexion.
Neck Flexion		The participant resisted against the non-elastic straps in forward flexion.
Lateral Neck Flexion		The participant pushed against a block in lateral flexion.

One practice subMVIC for each exercise was performed, followed by two additional maximum MVIC maneuvers for each exercise. While performing the maximum voluntary contractions the standard instructions were, "ready, set, go 1001, 1002, 1003, relax." This standardized verbal encouragement has been shown to improve reliability of the MVIC procedure.[88] The maximum EMG value for each muscle, regardless of the movement (forward, lateral or anterolateral flexion) was denoted as the true MVIC. In order to minimize muscle fatigue, a 1-minute rest was provided between each MVIC.

3.3.3 Exercise

The participants wore an Oxycon Mobile^{TM} (VIASYS Healthcare, GmBH, Germany), throughout the GXT. The Oxycon Mobile^{TM} is a portable metabolic unit that collects breath-by-breath data, while the participant breathes through the mask. Ventilatory parameters collected throughout the exercise included: V_E, V_T, RR and IC.

Prior to the exercise test, spirometry measures were also collected. The GXT was performed on a recumbent cycle ergometer (Vision Fitness, HRT R2750, USA). After sitting on the cycle ergometer for 2-minutes, baseline measurements including: ventilatory parameters, three IC maneuvers, EMG, and breathing and leg exertion were collected (Table 2). Participants began the GXT with 1-minute of loadless pedaling, followed by increases in resistance every minute (Appendix D). During the exercise test the participant maintained a pedaling rate between 60-64rpm. EMG collection began 20 seconds into the minute followed by the participant being asked to rate the magnitude of their perceived breathing exertion and leg discomfort by pointing to the 10-point modified Borg Scale (Appendix B). During the even stages, in the last 10 seconds of the exercise stage an IC maneuver was performed. During the test each subject was verbally encouraged to continue exercising as long as possible. The exercise test was terminated at the point of symptom limitation or if the subject was unable to maintain the 60-64rpm for at least 20 seconds. Exercise test duration was recorded as the duration of loaded pedaling. Peak work rate was recorded as the highest work rate that was maintained for at least 30 seconds. At the end of the test the participants were asked to state their reason for stopping the test. Data recorded manually included: breathing exertion, leg discomfort, and reason for stopping the test (Appendix C).

3.4   EMG Collection and Processing

The raw EMG signal was pre-amplified 200-times and then additional amplification (bandpass 10-1000Hz, CMRR=115db at 60Hz, input impedance approximately $10G\Omega$, $\pm$ 4V) was performed using an eight channel surface EMG system (AMT-8 EMG, Bortec Inc., Calgary, Alberta). The raw EMG signal was digitized at 2000Hz using an A/D converter (National Instruments BNC-2090 16-bit $\pm$ 10V).

All EMG data were corrected for the subject bias and gain and converted to μV. The data were full-wave rectified and then conditioned using a non-recursive, low-pass 2nd order Butterworth filter with a cutoff frequency of 2Hz. The filter was built using a custom Excel template[1], where Butterworth coefficients were calculated in using standard equations.[89] The filtered data provided a profile of the myoelectrical activity of the muscles over the breathing cycles.

3.4.1   Determining the maximum MVIC and normalizing the EMG data

The MVIC data was full wave rectified, corrected for the subject and system bias. A lowpass 2th order Butterworth filter with a cutoff frequency of 2Hz was used, and a linear envelope was performed (processed as above). The maximum EMG amplitude for the muscles during the MVIC exercises was calculated by using a 100 millisecond moving average. Once the EMG data for the MVIC maneuvers were processed, the difference between the two trials was assessed for each maneuver following the study visit. If the difference between the two trials were greater than 10% then the study visit

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[1] Custom Excel template written by Dr. John Kozey, School of Biomedical Engineering, School of Health and Human Performance & Department of Industrial Engineering

data for that participant was not used. The highest peak value from all the MVIC trials was deemed their highest maximal contraction and was used in the data analysis.[21] The processed exercise EMG data were normalized to the highest MVIC trial so the muscle activation could be expressed as a %MVIC (Figure 11). Muscle activation was quantified as a percent of the baseline MVIC and calculated according to Equation 4. The peak of each breathing cycle during inspiration was defined as the point where the preceding sample was lower and the proceeding sample was also lower. Once the peak was identified for each of the breathing cycles, then the %MVIC peak was averaged for each muscle collected during each stage of the GXT to determine the activation required for each exercise stage. For the purpose of this study, the muscle activation amplitude required to meet the increase in ventilatory demand for each exercise stage was the main variable of interest. See equation 4 for calculation of the %MVIC.

$$\%MVIC = \frac{ES_x}{ES} \times 100$$

Equation 4. % MVIC calculation for each muscle site with the peak amplitude in the numerator.

3.5 Data Analysis & Statistical Analysis

3.5.1 Descriptive Statistics

Descriptive statistics (sample mean and sample standard deviation) were used to describe the participants' demographical characteristics (including age, sex, weight, height, and % FEV_1) and response measurements (including breathing exertion, leg

discomfort, V_E, IC, maximal exercise stage and %MVIC muscle activation) at rest, the VT and end EX. Homogeneity of variance and normality of distributions were checked for demographic data, response variables and muscle activation. The F-test was used to test for equal variances and the Shapiro-wilks test along with an assessment of the skewness and kurtosis was used to test for normal distribution. Means and standard deviations for all continuous variables were calculated and frequency counts were calculated for categorical data. Differences between the time points (rest, VT and End EX) for the responses variables were assessed using student t-tests ($\alpha = 0.05$). All tests were performed using SPSS.

3.5.2 Objective A

The primary objective of this thesis was to determine if increasing exercise intensity resulted in changes in accessory respiratory muscle activation in healthy individuals, and sub-objective A was to determine if this increase was linear. These objectives were addressed by plotting the %MVIC for each of the exercise stages for each muscle. A line of best fit for a linear and polynomial model was plotted for each of the muscles and R^2 values were generated, to determine which model had the stronger fit with each muscle. This statistical analysis was performed using Excel.

3.5.3 Objective B

Objective B was to determine if there were differences in the %MVIC muscle activation between the muscles and at different time points throughout the exercise. A mixed model ANOVA was used to assess the differences. In this study, we have three

repeated measures, which are the time points (rest, VT and end EX), and the two different groups are the muscles (scalenes and SCM). The muscle activation represents the dependent variable. The ANOVA was used to test for main effects and interactions between and within the variables. Pair-wise comparisons were made using the Post-Hoc Tukey for any significant differences in the interactions. The p-value or α was set to 0.05. This statistical analysis was performed using SPSS.

3.5.4   Objective C and D

The final objectives were to determine if there was a relationship between the %MVIC and participants' IC and the %MVIC and the participants' perceived breathing exertion. This was addressed by assessing the correlation between the variables. A correlation coefficient (r) measures the strength and the direction of a relationship between two variables. The r-value is between -1 and 1, which indicates the strength of the association. For the purpose of this study, the relationship between %MVIC and IC and %MVIC IRV were determined by performing a Pearson's Product Moment Correlation and the relationship between the %MVIC and breathing exertion was determine by the Spearman correlation. This statistical analysis was performed using Excel.

CHAPTER 4 – RESULTS

4.1   Participant Recruitment and Study Visit:

The recruitment period for this study ran from May 2015 to June 2015 (Figure 9). There were 23 potential participants contacted for the study; 20 were eligible to participate in the study. Reasons for exclusion were: the participant did not meet the inclusion criteria (n=1), unable to meet time commitment (n=2).

Individuals contacted to participate in the study (n=23)

Excluded from study   (n=3)
Did not meet inclusion criteria (n=1)
Unable to meet time commitment (n=2)

Participants who were interested and eligible to participate (n=20)

Participants who completed the full visit (n=20)

Data analyzed (n=20)
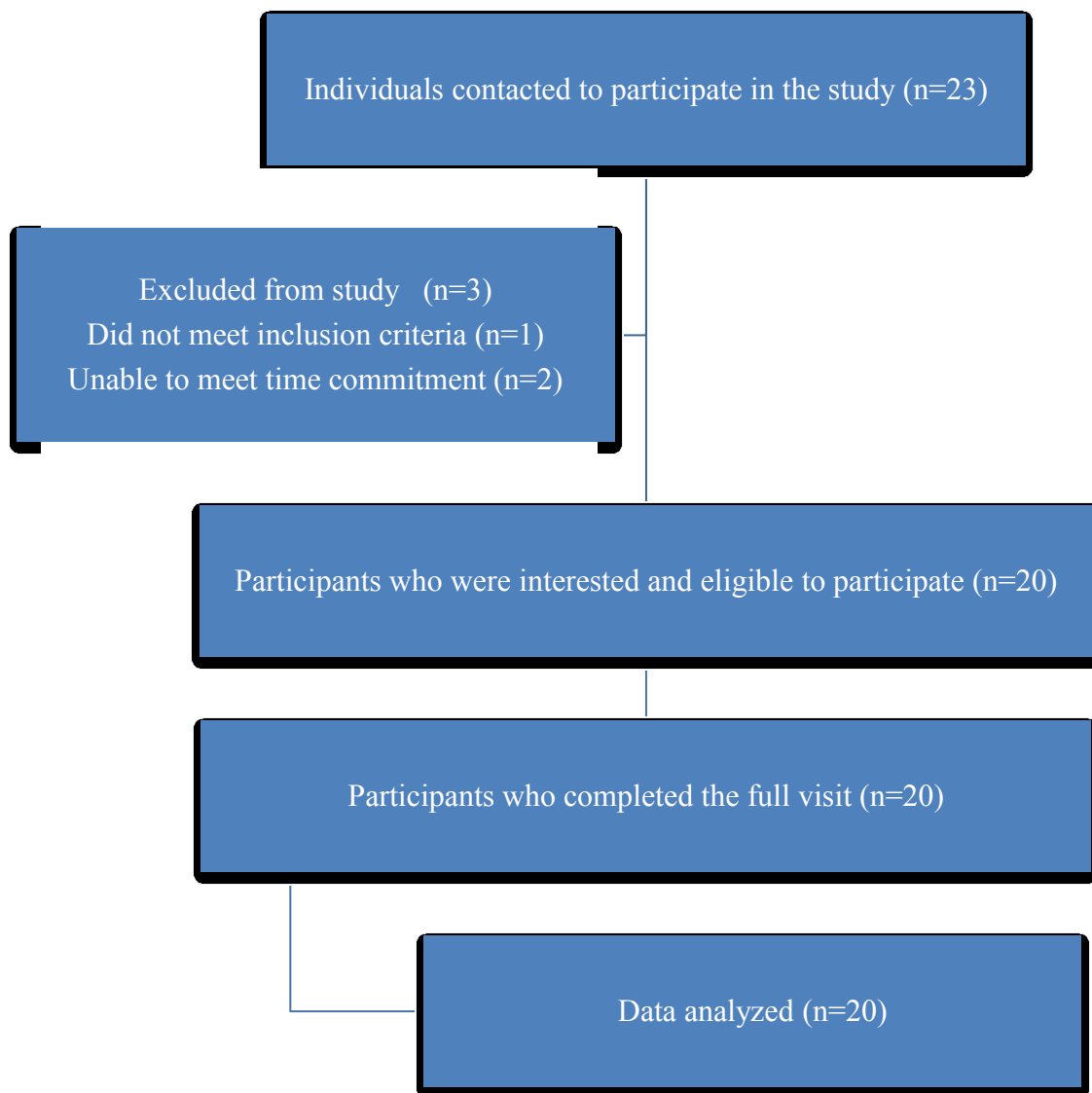
Figure 9. Flow chart of participant recruitment and study visit.

4.2   Participant Characteristics and Response Variable Statistics

Complete study data was obtained for all 20 participants that were recruited for this study. Participant demographics are displayed in Table 4. Participant data has been presented as frequencies and means with standard deviations. Fourteen participants who completed the study were between 50-59 years old, with an overall participant age average of 57 years. Men and women were equally represented. Physical activity characteristics for the participants are presented in Table 5. Eighty-five percent of the participants reported performing moderate intensity exercise (walking at least 30 minutes or equivalent) less than 1 to 3 times a month. Fifteen percent of the participants reported performing exercise (equivalent to walking 30 minutes) less than 2 times/week. Lung function was within normal limits as determined by the Global Initiative for Chronic Obstructive Pulmonary Lung Disease (Table 4). According to these guidelines lung function is considered to be healthy when it falls within the 95% confidence intervals for the various breathing parameters.

The variables displayed in Table 6 were collected by the Oxycon Mobile™ Metabolic Unit during the GXT. The metabolic unit measures V_T and RR directly and uses this information to calculate V_E. Similarly, the metabolic unit also measures exhaled oxygen and carbon dioxide and uses the ratio to calculate the respiratory exchange ratio (RER). The GXT consisted of 1-minute stages during which participants maintained a constant pedaling rate of 60-65rpm. Exercise times ranged from 6 to 16 minutes, with a mean duration of 10 minutes, corresponding to a workload of 141.9 watts. The participants' VT stage was determined as the point where the VCO_2 was greater than the VO_2 as described by the V-slope method.[90] The participants reached their VT between 4

and 11 minutes, with a mean VT time of 7 minutes, which is equivalent to 97.4 watts (Table 6). At the end of the GXT participants were asked their reason for stopping, by choosing from the following options: leg discomfort, breathing exertion or other. All participants responded that they stopped their exercise test due to leg discomfort.

Table 4. Participant Characteristics: Demographic and Anthropometric Characteristics

Characteristics	Frequency (n=20)
Sex	
Female	10
Male	10
	Mean (standard deviation)
Age (years)	57.4 (5.1)
Height (cm)	169.6 (10.1)
Weight (kg)	83.8 (15.6)
BMI (kg/m^2)	29.0 (4.2)
Pulmonary Function	
Predicted FEV_1 (L)	2.9 (0.81)
Predicted FVC (L)	3.5 (0.88)
Best FEV_1 (L)	3.2 (0.83)
Best FVC (L)	3.9 (0.94)
FEV_1/FVC (L)	80.6 (4.90)

BMI – Body mass index; FEV_1 – Forced expiratory volume in one second; FVC – Forced vital capacity

Table 5. Physical Activity Level of the Participants

Physical Activity Level	Frequency (n=20)	Percentage
No exercise	8	40%
Sporadic exercise (less than 1-3 times/month)	9	45%
Exercise less than 2 times/week	3	15%
Exercise more often or equal than 2 times/week	0	0%

Table 6 displays the mean and standard deviation for each of the response variables during the exercise, which were assessed at rest, VT and end EX. The data in

Table 6 demonstrates that there was a significant difference between VT and rest and between VT and end EX (p<0.05).

Table 6. Response variables assessed during the exercise test.

Response Variables	Mean (standard deviation)				
	Rest	VT	End EX	Δ Rest & VT	Δ VT & End EX
Exercise Workload (watts)	0.00(0.00)	97.4(26.6)	142(41.5)	97.4*	44.5*
Minute Ventilation (L/min)	9.00(2.50)	40.3(11.1)	62.4 (27.7)	31.3*	53.4*
VO_2 (mL/kg*min)	3.50(2.70)	17.6(5.80)	24.6(7.20)	14.1*	7.00*
IC (L)	2.79(0.90)	-	2.72(0.90)	-	-
IRV (L)	2.00(0.79)	-	0.68(0.39)	-	-
Breathing Exertion (0-10 scale)	0(0)	3.20 (0.90)	5.90(1.70)	3.20*	5.90*
Leg Discomfort (0-10 scale)	0(0)	3.20 (1.40)	5.70(2.30)	3.20*	5.70*

IC – inspiratory capacity; IRV – inspiratory reserve volume; RER – respiratory exchange ratio; VCO_2 – maximal carbon dioxide production; VO_2 – maximal oxygen consumption; L – liters; min - minute
* Significant difference between (p<0.05)

All the exercise tests were considered to be maximal in the sense that the participant's stated that they could not continue exercising; however it is important to know whether or not the participants achieved a VO_2 maximum during the exercise test. VO_2 maximum is defined as the maximum amount of oxygen the body can use during an incremental exercise test. The gold standard for determining if an individual has achieved a maximal exercise test is to confirm that there is less than a $150\text{mL}\cdot\text{min}^{-1}$ difference increase in VO_2 between the final two work rates of a progressive exercise test.[91,92] To

assess this in our participants, the mean VO_2 during the last 30 seconds in the final two exercise stages were calculated. If the difference between these two means was less than 150mL·min^{-1} then the participant was deemed to have achieved a maximal exercise test. Based on this criterion none of the 20 participants achieved a maximal exercise test.

4.3   MVIC Muscle Activation Response

Three maneuvers were used to stimulate maximum activation of the scalene and SCM muscles. These were: forward, anterolateral, and lateral neck flexion. After completing the assessment of all three MVICs it was clear that forward flexion provided the greatest maximal contraction in all participants (Table 7). Mean normalization amplitudes in %MVIC for each exercise and muscle are shown in Table 8. For both the scalene and SCM, the greatest activation was elicited in the forward neck flexion. All participants also found this maneuver the easiest to perform.

Table 7. Participant breakdown of the MVIC exercises that produced maximal activations for each muscle. The number of participants is shown in the table.

Exercise	Muscle	
	Scalenes	SCM
Rotational Neck Flexion	0	0
Forward Neck Flexion	20	20
Lateral Flexion	0	0

Table 8. Muscle activations during each MVIC maneuver normalized to the maximal MVIC amplitude for each exercise and muscle [mean(standard deviation)]. Greatest maximal activations for each muscle are bolded ($p<0.05$).

Exercise	Muscle	
	Scalenes	**SCM**
Rotational Neck Flexion	49 (16.5)	56 (12.8)
Forward Neck Flexion	**100 (0)**	**100 (0)**
Lateral Flexion	23 (11.2)	26 (14.2)

4.4 Muscle Activation Response to Exercise Intensity

The primary objective of this study was to determine if increasing exercise intensity resulted in changes in accessory respiratory muscle activation amplitude in healthy individuals. Furthermore we wanted to determine whether or not the increase was linear. Figure 10 presents that activation data for the scalene muscle group as a mean (+SD) of %MVIC. The activation progressively increased from 9.9(6.7) to, 24.1(5.6) and, 29.5(9.5) at rest, VT and end EX, respectively. Activation data for the SCM appears in Figure 10. Again, activation progressively increased during the exercise test. The figures for rest, VT and end EX were 0.5(0.7), 8.3(4.7), 12.7(5.6)% MVIC, respectively. The scalenes tripled in peak activation throughout the test while the SCM failed to double in peak muscle activation. Activity in the scalene group was present at rest in all participants (Figure 10). Sternocleidomastoid activity was detected in 4 participants at rest. Progressive recruitment occurred during the exercise test so that 5 participants demonstrated activation at 20%; 15 participants at 40%, 19 participants at 60% of exercise duration. It was not until 80% of the workload that all participants had activated their SCM.
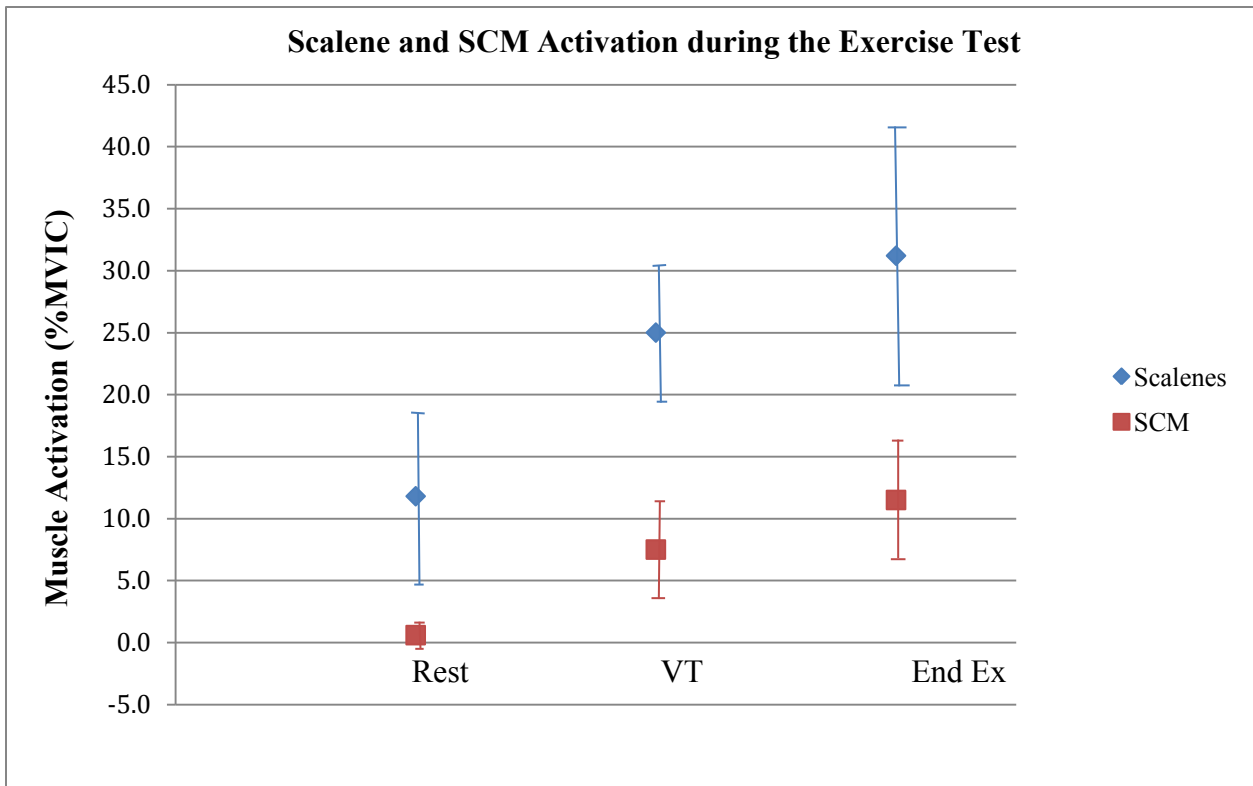
Figure 10. Mean and standard deviation of the scalenes and SCM activation plotted at rest, VT and end EX. Data presented in the symbol is the mean and the vertical lines represent the standard deviation.

A Shapiro-Wilks test ($p > 0.05$) and visual inspection of histograms and normal Q-Q plots showed that the data were normally distributed. The skewness and kurtosis also had z-values between -1.96 and +1.96. An F-test was also performed to test for equal variance ($p > 0.05$). These results indicate that the data was normally distributed with equal variances.

A mixed model ANOVA was performed to assess the difference between scalene and SCM activation at different time points in the GXT. Results from the analysis demonstrated significant time*muscle interaction ($p < 0.05$). Post-hoc Tukey's test analysis was used to investigate the interaction in more detail. The results of the Tukey analysis demonstrated a significant difference in muscle activation between the two

muscle groups at each time point. At rest, VT and end EX there was a significant difference in scalene activation compared to SCM activation ($p < 0.05$).

Further analysis was performed to determine if the increase in muscle activation during the test was linear. For this analysis the exercise test duration was separated into 20% intervals and the muscle activation for each participant was plotted. Once the activations were plotted a linear model was fit to the data for the scalene and SCM muscle activation. The data did not represent a good fit, with an R^2 value of 0.32 and 0.35 for the scalenes and SCM, respectively. After looking at the muscle activation throughout the exercise test it was clear that graphically the pattern of muscle activation for both muscles was curvilinear (Figure 11). Having made this observation the data was fit to a polynomial model, representing a stronger fit, with R^2 values of 0.52 ($p < 0.05$) and 0.61 ($p < 0.05$) respectively for the scalenes and SCM. This analysis of the activations throughout the exercise test for both muscles demonstrated a curvilinear increase as exercise intensity increases (Figure 11).

Scalenes Muscle Activation Throughout Exercise Test

$y = 0.0003x^2 + 0.1695x + 9.5477$
$R^2 = 0.5175$

SCM Muscle Activation Throughout Exercise Test

$y = 0.001x^2 + 0.0318x + 0.2698$
$R^2 = 0.6113$
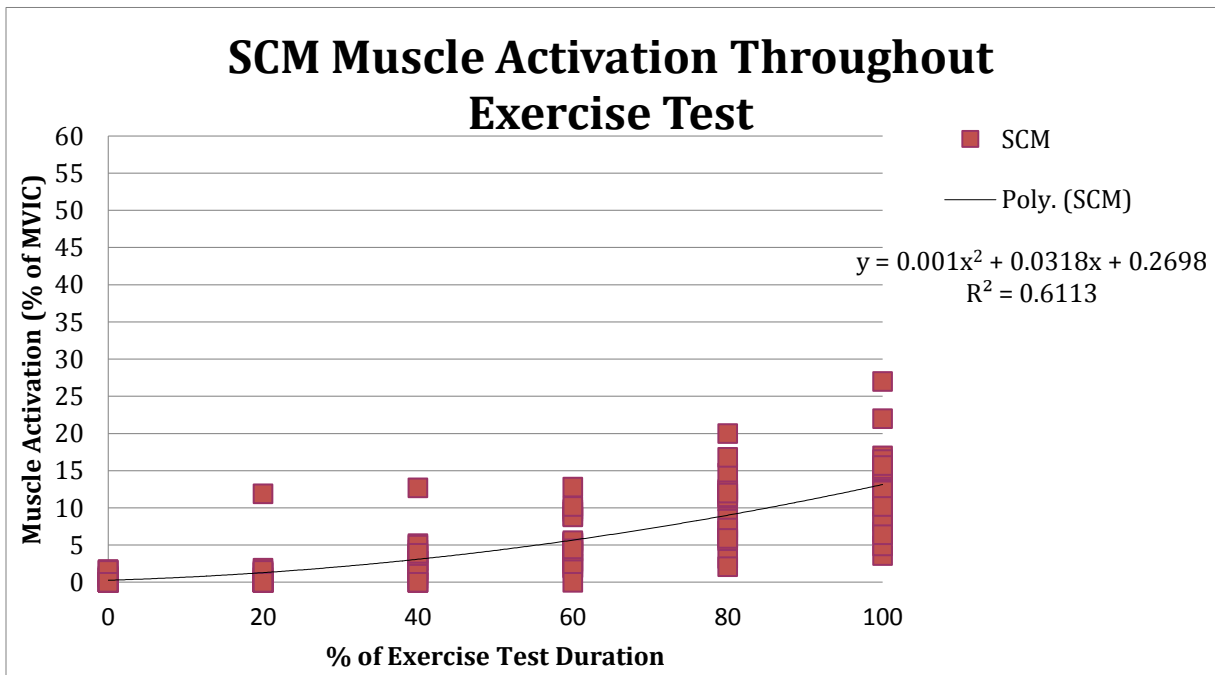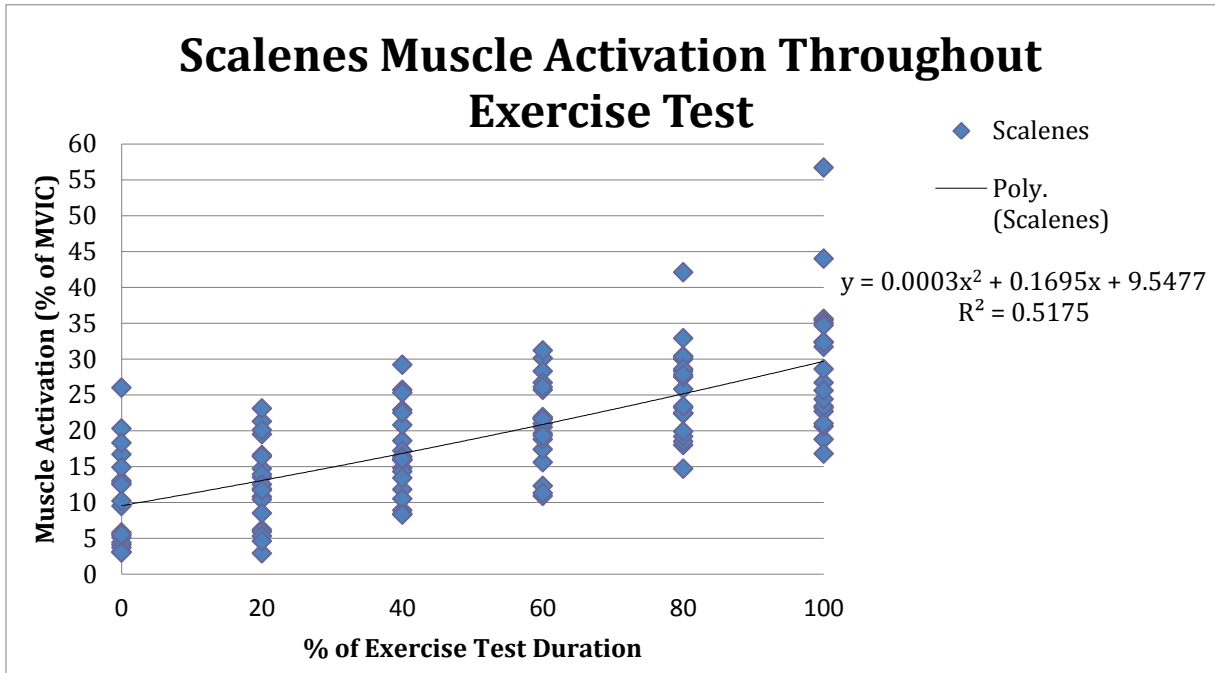
Figure 11. Muscle activations for each muscle plotted for each participant at 20% intervals of their maximal exercise workload.

4.5 Correlations between Response Variables

The final objective of this study was to determine the relationship between the following variables 1) muscle activation and IC, 2) muscle activation and perceived breathing exertion, and 3) muscle activation and perceived leg discomfort. A guide set out by Evans in 1996 can be used to describe the strength of the correlation (Table 9).[93]

Table 9. Strength descriptions for correlation values.

r value	Description of Strength
0.00-0.19	Very weak
0.20-0.39	Weak
0.40-0.59	Moderate
0.60-0.79	Strong
0.80-1.0	Very strong

The IC maneuvers were conducted during the even stages of the exercise test as well as at end exercise. Collecting the IC measurements during the exercise test was difficult due to a delay in the metabolic unit screen, which did not allow for appropriate identification of end expiration. The inability to accurately assess end expiration during later stages of the exercise test when the RR was high also made measuring the IC difficult. We did not know a priori, the point at which the participants' VT would occur. As VT could have occurred during an odd stage of the exercise test, we were unable to record the IC at the VT for each participant. VT occurred during an even test stage in only four participants. Therefore we only assessed the correlation between the muscle activation and IC at end EX. The correlation between these two variables was performed using the Pearson Product Moment correlation. Based on Evan's guidelines the correlation between muscle activation and the IC for the scalenes (r=0.38) and SCM (r=0.38) were weak. IC is a measure to assess dynamic hyperinflation, which is typically

associated with an increase in dyspnea, which in our case we call breathing exertion. For the purpose of this study we assessed the relationship between IC and breathing exertion and found a negative weak correlation (r= -0.35).

Increase ventilation during the exercise test was accomplished by elevation of the RR and V_T. An increase in V_T is associated with a fall in the inspiratory reserve volume (IRV). O'Donnell has stated that the IRV relates better to dyspnea than the IC;[18] therefore, we also assessed the correlation between IRV and breathing exertion in our data. The Pearson Product Moment correlation was used to assess the correlation between the muscle activation and the participants' IRV. The scalene (r=0.52) and SCM (r=0.49) muscle activation both demonstrated a moderate correlation with IRV. When we assessed the relationship between IRV and breathing exertion we found a moderate correlation (r=0.49).

Participants used the Borg (CR-10) scale to rate both perceived breathing exertion and leg discomfort. The association between the muscle activation and the perceived exertion variables were assessed at VT and end EX and were analyzed using the spearman rank correlation. The correlations are presented below in Table 10.

Table 10. Correlations between muscle activation and perceived exertion variables

Correlation between: perceived breathing exertion and muscle activation			
VT	Scalenes	0.19	Very weak
	SCM	0.46*	Moderate
End EX	Scalenes	0.24	Weak
	SCM	0.30	Weak
Correlation between: leg discomfort and muscle activation			
VT	Scalenes	0.10	Very weak
	SCM	0.22	Weak
End EX	Scalenes	0.10	Very weak
	SCM	0.20	Very weak

*Correlation is significant at the 0.05 level
VT – ventilatory threshold; SCM – sternocleidomastoid; EX - exercise

CHAPTER 5 – DISCUSSION

The primary aim of this study was to determine whether accessory respiratory muscle activation increases with increasing exercise intensity. Surface EMG was used to capture muscle activation amplitude for the scalene and SCM muscles during a GXT in healthy participants. The EMG muscle activation was normalized and presented as the %MVIC for each muscle. The results of this study demonstrated that scalene and SCM muscle activation increased in a curvilinear fashion during the test. In contrast to the SCM, the scalenes were active during quiet breathing. The activation amplitude in the scalenes always exceeded that of the SCM. The increased muscle activation was also associated with a decrease in the participants' IRV. This is the first study to investigate muscle activation of both the scalenes and SCM during exercise in a healthy population. It is also the first study to normalize the activation to a percent of the muscle's maximal contraction, which allows for direct comparison between muscles and individuals. The results of this study are important because they lay the groundwork for future studies to understand changes in muscle activation in people with COPD, including assessing the effects of interventions to improve respiratory muscle function.

5.1   Scalene and SCM Pattern of Activation during Exercise

The healthy participants in this study demonstrated a progressive increase in scalene and SCM activation, in response to the increased ventilatory demand during the GXT. The pattern of activation can be described in terms of, amplitude and timing. The scalenes reached a peak mean muscle activation of 29.5% of the MVIC and the SCM

reached 12.7% of its MVIC. At end EX the scalenes and SCM were 2.97 and 25.4 times more active, respectively, compared to at rest. The pattern of muscle activation was curvilinear demonstrating an inflection around the VT. Poulsen et al.[94] and Johnson et al.[95] demonstrated that in healthy individuals, during cycle GXTs, diaphragm muscle activation amplitude plateaued around 70% of workload. Poulsen et al. found that despite the plateau in diaphragm EMG activity; VO_2, V_E, V_T and RR continued to increase.[94] These researchers hypothesized that, accessory respiratory muscle activity increased in order to fulfill the need for increased ventilation. Our work supports that hypothesis. Although we did not monitor diaphragm activation, we saw the curvilinear increase in scalene and SCM muscle activation become more dramatic around VT, which, for our participants, was between 60-80% of their maximum workload.

Although activity in both accessory respiratory muscles increased during the exercise test, the pattern of activation was different for the two muscles. The scalenes were active during quiet breathing and then progressively increased their activation throughout the exercise test. Alternatively, the SCM were not active during rest and demonstrated progressive recruitment throughout the test. Initiation of activity differed among participants. Activation was noted in five, fifteen and nineteen participants at 20%, 40%, and 60% of workload maximum, respectively. It was not until 80% of workload maximum that all participants demonstrated SCM activation. One explanation for the difference in the muscle activation patterns is that higher workloads demand more power than endurance to meet the increased ventilatory demand. During high intensity exercise RR increases and the time for inspiration and expiration decreases. Airflow must accelerate, in order to increase V_T in response to the exercise challenge. Generating

higher flow rates is a power activity[13] typically generated by the type II, fast twitch fibers. In fact, researchers have assessed the fiber type composition of the scalenes and SCM in animal models such as hamsters[96] and rhesus monkeys.[97] Mattson and colleagues measured the relative fiber type composition in a number of respiratory muscles in hamsters. They found that the scalenes had 53% ± 3% type IIa fibers. This was similar to the results in the rhesus monkey.[97] In this study, Richmond et al. took 1-2cm blocks of the scalene and SCM from the lateral edge to the middle of the muscle and assessed the fiber type composition in 16 sequential slices from each block. They reported that the scalenes consisted of 35-50% type IIa fibers, 15-40% type IIb fibers and only 20-35% type I fibers. These investigators determined that the SCM was composed of 90% fast twitch fiber subtypes but did not report the type II subgroups. Richmond and colleagues stated that they believed their results were representative of muscle groups found in humans. No studies have assessed the fiber type composition of the accessory muscles in humans. However, if Richmond and colleagues are correct and monkey and human muscles are similar then the predominance of type II fibers in the accessory respiratory muscles we studied would support our hypothesis about the origin of the activation patterns we observed.

Although an increase was observed in muscle activation for both muscles throughout the GXT, neither muscle was close to 100% activation at the highest workload. In other peripheral muscles in the leg; during a gait cycle most muscles only reach 20-30% MVIC in asymptomatic individuals. Considering this, we can accept that 29% muscle activation for the scalenes is quite high and that the muscle activation may not achieve a higher amplitude. The smaller amplitude increases we observed in the

scalene and SCM muscles may have occurred for two reasons. First, the participants terminated their exercise due to leg discomfort rather than breathing exertion and therefore the ventilatory demand was not high enough to require maximal activation of the accessory respiratory muscles. Female participants in this study had an average VO_{2PEAK} of 20.8 ± 3.7 ml/min/kg and our males had an average VO_{2PEAK} of 28.5 ± 7.9ml/min/kg. Participants in this study did not meet the criteria that define a VO_{2MAX}.[98] However compared to the normative values set by the American College of Sports Medicine (ACSM), these VO_{2PEAK} values would correspond to 68% and 71% of VO_{2MAX} for our female and male participants, respectively[98] Even when taking into account that the VO_{2MAX} on a bike is less than that obtained during treadmill testing, these values indicated that our participants had only fair exercise capacity.[98] This suggests that the ventilatory demand during the test was not sufficiently high enough to limit V_T expansion. This may seem surprising as common knowledge suggests that the first VT occurs at approximately 40-60% of $VO_{2MAX.}$ Our participants exceeded this intensity, where V_E and thus respiratory muscle activity should increase dramatically. This was not the case in our healthy participants. However, Carey et al.,[99] and others[100] have reported the presence of a second VT at approximately 80-95% $VO_{2MAX.}$ Our participants did not reach the second VT because at end EX our female and male participants had a VO_{2PEAK} of 68% and 71%, respectively, relative to normative VO_{2MAX} values for their age group. Therefore we believe that our participants' ventilation did not require significant accessory muscle activation because ventilation was still being generated by the diaphragm as Poulsen and colleagues suggest its activation does not plateau until 70% of the $VO_{2MAX.}$

The second reason why the accessory respiratory muscle activation was not close to their maximum may be the technique we used to assess maximum activation. The accessory respiratory muscles have functions other than respiration and we normalized the muscle activation to these non-ventilatory maneuvers that did not reflect the muscle action during the GXT. For instance, the SCM, whose primary function is to rotate the head, only achieved 12.7% of its MVIC at end EX. It is possible that these muscles may never achieve 100% muscle activation during respiratory activity. In other research, muscles were normalized using an MVIC maneuver that is involved in the movement or activity being investigated.[24,82] An alternative maneuver to normalize the scalene and SCM muscle activation could be to use a MIP because it is a maximal respiratory maneuver for which normative values are available for all age ranges.[57,58] Research conducted by Sinderby et al.[101] and Jolley et al.[102] both used the MIP, to normalize diaphragm muscle activation and found that the MIP produced a large MVIC for the diaphragm.[102] One difficulty with the MIP maneuver is that it measures the maximal pressure generated by all the inspiratory muscles, which is undoubtedly dominated by the diaphragm and it would not be possible to distinguish the contribution of specific accessory respiratory muscles. Therefore, it seems desirable to normalize using non-respiratory actions, which allows accurate measure of the increase in activation for the specific muscles of interest.

5.2   The Relationship between Muscle Activation and IC

We were interested in examining the relationship between accessory respiratory muscle activation and the IC because, in people with COPD, hyperinflation during

exercise is associated with a decrease in IC, which leads to an increase in dyspnea and ultimately premature exercise termination. This, in combination with the decrease in compliance associated with hyperinflation, increases the work of the respiratory muscles.[4] In that situation, activation of the scalenes and SCM could facilitate an increase in ventilation. While healthy individuals do not demonstrate a decrease in the IC during exercise[4] data from this study will form the basis for comparison between respiratory muscle function between healthy people and those with COPD. Therefore, it was important to document the relationship in people without disease. As expected, we did not see a decrease in IC, during exercise testing. Other changes in lung volumes that we observed in our healthy participants were similar in direction to those typically observed during incremental exercise in people with COPD. For instance, tidal volume increases during an incremental exercise test and is associated with a progressive reduction in IRV. We noted a moderate association between IRV and accessory respiratory muscle activity suggesting that these muscles assisted in increasing the V_T. Until diaphragm muscle activation beings to plateau at 70% of $VO_{2MAX,}$[94] changes in V_T would be generated by all the inspiratory muscles including the accessory muscles, explaining the limited increase in accessory respiratory muscle activation and therefore only a moderate association with IRV.

Research by O'Donnell and colleagues demonstrated that, in both healthy individuals and people with COPD, a decrease in IRV was associated with an increase in breathing exertion/dyspnea. In fact they showed that in both groups, termination of exercise was associated with a reduction in IRV that limited further expansion of V_T.[103] We found a moderate correlation between IRV and breathing exertion (r=0.49) and

expected similar correlation strength between accessory respiratory muscle activation and dyspnea as these muscles play an important role in ventilation at the end of exercise when lung compliance is low. Therefore, we were surprised by the weak correlation our analysis revealed. Scalene and SCM muscle activation at the end of exercise was r=0.24 and r=0.30, respectively. This supports our belief that our participants' diaphragm activation did not plateau; therefore accessory respiratory muscle activation was minimal.

5.3   Accessory Respiratory Muscle EMG

5.3.1   EMG Signal Assessment

To our knowledge this is the first study to assess respiratory muscle activity over a series of consecutive breaths, specifically looking at the muscle activation during inspiration. We assessed the muscle activation over a 20 second period during each exercise stage. Other studies have assessed the pattern of activation at different static lung pressures. Breslin et al. and Chiti et al., assessed scalenes and SCM muscle activation, respectively, while having their participants perform MIP maneuvers at different lung volumes.[66,67] Alternatively, research conducted by De Troyer and colleagues[41] assessed scalene activation during quiet breathing during a single breath and used the peak activation value from 3 such breaths to represent the peak muscle activation. They assessed activation by using an EMG recording that was initiated in response to the inspiratory and expiratory flow observed on the screen of a metabolic unit. In preliminary work for this thesis, we found this approach created difficulty at higher respiratory rates where time for inspiration and expiration becomes very short. In those circumstances we were unable to reliability trigger the EMG recording at the start of the respiratory cycle.

In contrast to De Troyer we feel that it is very important to capture a series of breaths during exercise to ensure clear identification of the peak muscle activation.

5.3.2   EMG Signal Normalization

Another unique aspect of this study was the MVIC normalization technique that was used. Duiverman and colleagues are the only other research group to use a normalization technique during assessment of accessory respiratory muscle activation.[19] These researchers normalized activation during a GXT using a logarithmic transformation of the ratio of the peak EMG activity during exercise and the peak activation at rest.[19] This is problematic because the technique does not quantify the activation with respect to a known maximum, making it impossible to describe how much the muscle is activating to achieve the given ventilatory load. This is important because it prevents researchers from comparing activation levels among individuals. It also makes it difficult to assess the effects of interventions such as pulmonary rehabilitation or medication on accessory muscle activation.

The individuals who participated in this study performed three movements to determine the MVIC, because no previous research has performed normalization using the MVIC technique. Our procedures were based on previous research, which described the need to use multiple maneuvers to elicit a maximal contraction in other peripheral muscles.[80,83] The current study provides strong evidence to support using just one maneuver, forward flexion, to produce maximum activation for both the scalenes and SCM.

5.4   Implications with COPD

The rationale for this study was to establish a technique that would allow us to assess the pattern of accessory respiratory muscle activation in healthy individuals during a GXT. Now that we understand the accessory muscle activation response to exercise in healthy individuals we can use the procedures and associated data for comparison with studies involving individuals with COPD. The pathophysiology of the disease affects the force generating capacity of the diaphragm, which may result in increased accessory muscle activity both from a timing and amplitude perspective compared to that seen in healthy individuals. Specifically future research should compare the accessory muscle activation in individuals with COPD who do and do not experience hyperinflation during a GXT, helping to clarify the role of the accessory muscles during dynamic hyperinflation. Such knowledge would form the groundwork for understanding how interventions such as medications and exercise affect accessory muscle activity, helping to develop and optimize therapeutic interventions.

5.5   Limitations

Collectively, the findings in this thesis provide objective evidence that healthy older adults activate their accessory respiratory muscles to assist breathing during times of increased ventilatory demand. While considerable care was taken, some study limitations exist.

1. It was difficult and sometimes impossible to accurately measure the IC due to problems identifying the end of expiration. Both the portable metabolic unit and the recumbent bike contributed to this problem. The Oxycon Mobile™

has a time delay in the presentation of the breathing cycle on the computer screen and therefore we could not use this method to start the IC measurement. During the resting stages we were able to watch the participant's chest movements to initiate the IC maneuver; however, this was not possible during the cycling stages. The recumbent position introduced excess movement of the upper body, making it impossible to watch their chest movement for end expiration. One solution to this problem would be to use a stationary metabolic unit that has minimal screen time delay. Alternatively verbally cueing the participant with "on your next breath take a big breath in," could also be used to obtain an IC.[9]

2. The EMG signal was corrected for subject bias while the person was in supine lying during quiet breathing. The literature shows that the scalenes are always active during quiet breathing[43], which was also true for all of the participants in the current study. Additionally, we found in some of our participants that the SCM was also active during quiet breathing. Typically, a subject bias trial measures resting muscle activity, meaning that there should be very minimal muscle activation. In this study, the resting subject bias was taken during a period of quiet breathing and thus the scalenes and SCM could have been activated during this trial. The processed data was taken from each exercise stage minus the resting bias, which would ultimately present lower EMG amplitude. Therefore, taking a resting bias during quiet breathing, as was done in this study, may not give a true resting bias of the muscle, which may result in lower EMG muscle amplitudes throughout the exercise test. We do

not expect this to have a significant effect on the EMG amplitude values in healthy individuals due to the low levels of activation of the scalenes and SCM expected during quiet breathing in healthy individuals. However, this may pose a greater issue in individuals with COPD who use these muscles to a greater extent during quiet breathing. Future research should investigate if the scalenes and SCM are activated to a lower amount while an individual is holding their breath than during quiet breathing. If this is true, future research should consider using this breath-hold method for the subject bias trial rather than a quiet breathing trial.

5.6   Conclusion

A technique was established that allows researchers to assess accessory respiratory muscle activation during a GXT. Overall both the scalenes and SCM activation increased in a curvilinear fashion with an increase in exercise intensity, demonstrating a significant difference between the two muscles, at each of the time points (rest, VT and end EX). No relationship was found between the muscle activation and IC, which was expected since we had healthy participants. In addition we investigate the relationship between IRV and muscle activation, which has been noted to have a stronger relationship in both a healthy population and individuals with COPD. We confirmed these results, demonstrating a moderate correlation between IRV and muscle activation. Despite the moderate correlation between IRV and muscle activation, a weak correlation was found between muscle activation and breathing exertion.

Although there were concerns about using a non-respiratory maneuver to obtain the MVIC for normalization, it is still the best technique for isolating the scalene and SCM muscle activity. The results of this study suggest that healthy individuals who fail to achieve a VO_{2MAX} use minimal accessory muscle activation to increase ventilation. Despite this, accessory muscle activation demonstrated a moderately strong association with the decrease in the participants' IRV. There was only a weak association between activation and breathing exertion, as limitation of tidal volume expansion was not observed in this study.

This was the first study to measure and quantify accessory muscle activation using an MVIC normalization technique. This study has established the pattern of activation for the scalenes and SCM in response to an increase in exercise intensity. These findings lay the groundwork to investigate accessory muscle activation in individuals with COPD and evaluate the effects of interventions on the disease.

REFERENCES

1. O'Donnell DE, Hernandez P, Kaplan A, et al. Canadian thoracic society recommendations for management of chronic obstructive pulmonary disease - 2008 update - highlights for primary care. *Can Respir J*. 2008;15 Suppl A:1A-8A.
2. Global Initiative for Chronic Obstructive Lung Disease, Inc. Global strategy for the diagnosis, management, and prevention of chronic obstructive pulmonary disease.
3. Stockley RA, Rennard SI, Rabe K. Chronic obstructive pulmonary disease a practical guide to management. Updated 2008.
4. O'Donnell DE, Laveneziana P. Physiology and consequences of lung hyperinflation in COPD. *European Respiratory Review*. 2006;15(100):61-67.
5. RodrÃguez-Roisin R. The airway pathophysiology of COPD: Implications for treatment. *COPD*. 2005;2(2):253-262.
6. Leff AR, Schumacker PT. *Respiratory physiology : Basics and applications*. Philadelphia: W.B. Saunders Co.; 1993.
7. Ferguson GT. Why does the lung hyperinflate? *Proc Am Thorac Soc*. 2006;3(2):176-179.
8. Pepin V, Saey D, Laviolette L, Maltais F. Exercise capacity in chronic obstructive pulmonary disease: Mechanisms of limitation. *COPD*. 2007;4(3):195-204.
9. Guenette JA, Chin RC, Cory JM, Webb KA, O'Donnell DE. Inspiratory capacity during exercise: Measurement, analysis, and interpretation. *Pulm Med*. 2013;2013:956081.
10. O'Donnell DE, Revill SM, Webb KA. Dynamic hyperinflation and exercise intolerance in chronic obstructive pulmonary disease. *Am J Respir Crit Care Med*. 2001;164(5):770-777.
11. Banner MJ. Respiratory muscle loading and the work of breathing. *J Cardiothorac Vasc Anesth*. 1995;9(2):192-204.
12. Celli B. The diaphragm and respiratory muscles. *Chest Surg Clin N Am*. 1998;8(2):207-224.
13. Kift J, Williams EM. The respiratory time and flow profile at volitional exercise termination. *J Sports Sci*. 2007;25(14):1599-1606.
14. O'Donnell DE, Laveneziana P. Dyspnea and activity limitation in COPD: Mechanical factors. *COPD*. 2007;4(3):225-236.
15. Cooper CB. Determining the role of exercise in patients with chronic pulmonary disease. *Med Sci Sports Exerc*. 1995;27(2):147-157.
16. Casaburi R, Patessio A, Ioli F, Zanaboni S, Donner CF, Wasserman K. Reductions in exercise lactic acidosis and ventilation as a result of exercise training in patients with obstructive lung disease. *Am Rev Respir Dis*. 1991;143(1):9-18.
17. Maltais F, LeBlanc P, Jobin J, et al. Intensity of training and physiologic adaptation in patients with chronic obstructive pulmonary disease. *Am J Respir Crit Care Med*. 1997;155(2):555-561.
18. O'Donnell DE. Hyperinflation, dyspnea, and exercise intolerance in chronic obstructive pulmonary disease. *Proc Am Thorac Soc*. 2006;3(2):180-184.
19. Duiverman ML, de Boer EW, van Eykern LA, et al. Respiratory muscle activity and dyspnea during exercise in chronic obstructive pulmonary disease. *Respir Physiol Neurobiol*. 2009;167(2):195-200.
20. Burden A. How should we normalize electromyograms obtained from healthy participants? what we have learned from over 25years of research. *J Electromyogr Kinesiol*. 2010;20(6):1023-1035.
21. Konrad P. The ABC of EMGA practical introduction to kinesiological electromyography. . 2005.
22. Yang JF, Winter DA. Electromyographic amplitude normalization methods: Improving their sensitivity as diagnostic tools in gait analysis. *Arch Phys Med Rehabil*. 1984;65(9):517-521.

23. Rutherford DJ, Hubley-Kozey CL, Stanish WD. Knee effusion affects knee mechanics and muscle activity during gait in individuals with knee osteoarthritis. *Osteoarthritis Cartilage*. 2012;20(9):974-981.

24. Hubley-Kozey CL, Robbins SM, Rutherford DJ, Stanish WD. Reliability of surface electromyographic recordings during walking in individuals with knee osteoarthritis. *J Electromyogr Kinesiol*. 2013;23(2):334-341.

25. Barnes PJ, Celli BR. Systemic manifestations and comorbidities of COPD. *Eur Respir J*. 2009;33(5):1165-1185.

26. West JB. *Respiratory physiology: The essentials*. Wolters Kluwer Health/Lippincott Williams \& Wilkins; 2008.

27. Murray JF. *The normal lung: The basis for diagnosis and treatment of pulmonary disease*. W.B. Saunders Company; 1976.

28. Spieth PM, Guldner A, de Abreu MG. Chronic obstructive pulmonary disease. *Curr Opin Anaesthesiol*. 2012;25(1):24-29.

29. Nieman G. Acute effects of "standard" smoke inury to the lung parenchyma. *Respiratory Care*. 1991;36(11):1211.

30. Standards for the diagnosis and care of patients with chronic obstructive pulmonary disease. american thoracic society. *Am J Respir Crit Care Med*. 1995;152(5 Pt 2):S77-121.

31. Vandivier RW, Voelkel NF. The challenges of chronic obstructive pulmonary diseases (COPD)--a perspective. *COPD*. 2005;2(1):177-184.

32. Loring SH, Garcia-Jacques M, Malhotra A. Pulmonary characteristics in COPD and mechanisms of increased work of breathing. *J Appl Physiol*. 2009;107(1):309-314.

33. Achenbach T, Weinheimer O, Biedermann A, et al. MDCT assessment of airway wall thickness in COPD patients using a new method: Correlations with pulmonary function tests. *Eur Radiol*. 2008;18(12):2731-2738.

34. Bosken CH, Wiggs BR, Pare PD, Hogg JC. Small airway dimensions in smokers with obstruction to airflow. *Am Rev Respir Dis*. 1990;142(3):563-570.

35. Deveci F, Murat A, Turgut T, Altuntas E, Muz MH. Airway wall thickness in patients with COPD and healthy current smokers and healthy non-smokers: Assessment with high resolution computed tomographic scanning. *Respiration*. 2004;71(6):602-610.

36. Garcia-Aymerich J, Lange P, Benet M, Schnohr P, Anto JM. Regular physical activity reduces hospital admission and mortality in chronic obstructive pulmonary disease: A population based cohort study. *Thorax*. 2006;61(9):772-778.

37. Moy ML, Matthess K, Stolzmann K, Reilly J, Garshick E. Free-living physical activity in COPD: Assessment with accelerometer and activity checklist. *J Rehabil Res Dev*. 2009;46(2):277-286.

38. O'Donnell DE, Laveneziana P. The clinical importance of dynamic lung hyperinflation in COPD. *COPD*. 2006;3(4):219-232.

39. Tortora GJ, Derrickson BH. *Principles of anatomy and physiology*. Wiley; 2011.

40. Luce JM, Culver BH. Respiratory muscle function in health and disease. *Chest*. 1982;81(1):82-90.

41. De Troyer A, Estenne M. Coordination between rib cage muscles and diaphragm during quiet breathing in humans. *J Appl Physiol Respir Environ Exerc Physiol*. 1984;57(3):899-906.

42. Legrand A, Schneider E, Gevenois P, Troyer AD. Respiratory effects of the scalene and sternomastoid muscles in humans. *J Appl Physiol*. 2003;94(4):1467-1472.

43. De Troyer A, Estenne M. Functional anatomy of the respiratory muscles. *Clin Chest Med*. 1988;9(2):175-193.

44. Han JN, Gayan-Ramirez G, Dekhuijzen R, Decramer M. Respiratory function of the rib cage muscles. *Eur Respir J*. 1993;6(5):722-728.

45. De Troyer A. Effect of hyperinflation on the diaphragm. *Eur Respir J*. 1997;10(3):708-713.

46. Fitting JW. Respiratory muscles in chronic obstructive pulmonary disease. *Swiss Med Wkly*. 2001;131(33-34):483-486.

47. Hamid Q, Shannon J, Martin J. *Physiologic basis of respiratory disease.* BC Decker, Incorporated; 2005.

48. Hudson AL, Gandevia SC, Butler JE. The effect of lung volume on the co-ordinated recruitment of scalene and sternomastoid muscles in humans. *J Physiol*. 2007;584(Pt 1):261-270.

49. De Troyer A, Kelly S. Action of neck accessory muscles on rib cage in dogs. *J Appl Physiol Respir Environ Exerc Physiol*. 1984;56(2):326-332.

50. Estenne M, De Troyer A. Relationship between respiratory muscle electromyogram and rib cage motion in tetraplegia. *Am Rev Respir Dis*. 1985;132(1):53-59.

51. Gandevia SC, Leeper JB, McKenzie DK, De Troyer A. Discharge frequencies of parasternal intercostal and scalene motor units during breathing in normal and COPD subjects. *Am J Respir Crit Care Med*. 1996;153(2):622-628.

52. Winslow C, Rozovsky J. Effect of spinal cord injury on the respiratory system. *Am J Phys Med Rehabil*. 2003;82(10):803-814.

53. Sharp JT, Druz W, Danon J, Kim MJ. Respiratory muscle function and the use of respiratory muscle electromyography in the evaluation of respiratory regulation. *Chest*. 1976;70(1 Suppl):150-154.

54. De Troyer A, Estenne M, Vincken W. Rib cage motion and muscle use in high tetraplegics. *Am Rev Respir Dis*. 1986;133(6):1115-1119.

55. Tamplin J, Brazzale DJ, Pretto JJ, et al. Assessment of breathing patterns and respiratory muscle recruitment during singing and speech in quadriplegia. *Arch Phys Med Rehabil*. 2011;92(2):250-256.

56. Ruppel G. *Manual of pulmonary function testing.* Mosby, Incorporated;

57. Miller MR, Crapo R, Hankinson J, et al. General considerations for lung function testing. *Eur Respir J*. 2005;26(1):153-161.

58. Gosselink R, Stam H. *European respiratory monograph 31: Lung function testing:* European Respiratory Society Journals; 2005.

59. Soderberg GL, Knutson LM. A guide for use and interpretation of kinesiologic electromyographic data. *Phys Ther*. 2000;80(5):485-498.

60. Yokoba M, Abe T, Katagiri M, Tomita T, Easton PA. Respiratory muscle electromyogram and mouth pressure during isometric contraction. *Respir Physiol Neurobiol*. 2003;137(1):51-60.

61. Lawrence JH, De Luca CJ. Myoelectric signal versus force relationship in different human muscles. *J Appl Physiol*. 1983;54(6):1653-1659.

62. Woods JJ, Bigland-Ritchie B. Linear and non-linear surface EMG/force relationships in human muscles. an anatomical/functional argument for the existence of both. *Am J Phys Med*. 1983;62(6):287-299.

63. Alkner BA, Tesch PA, Berg HE. Quadriceps EMG/force relationship in knee extension and leg press. *Med Sci Sports Exerc*. 2000;32(2):459-463.

64. Krishnan C, Williams GN. Sex differences in quadriceps and hamstrings EMG-moment relationships. *Med Sci Sports Exerc*. 2009;41(8):1652-1660.

65. Nici L, Donner C, Wouters E, et al. American thoracic society/european respiratory society statement on pulmonary rehabilitation. *Am J Respir Crit Care Med*. 2006;173(12):1390-1413.

66. Chiti L, Biondi G, Morelot-Panzini C, Raux M, Similowski T, Hug F. Scalene muscle activity during progressive inspiratory loading under pressure support ventilation in normal humans. *Respir Physiol Neurobiol*. 2008;164(3):441-448.

67. Breslin EH, Garoutte BC, Kohlman-Carrieri V, Celli BR. Correlations between dyspnea, diaphragm and sternomastoid recruitment during inspiratory resistance breathing in normal subjects. *Chest*. 1990;98(2):298-302.

68. Lehman GJ, McGill SM. The importance of normalization in the interpretation of surface electromyography: A proof of principle. *J Manipulative Physiol Ther*. 1999;22(7):444-446.

69. Mathiassen SE, Winkel J, Hagg GM. Normalization of surface EMG amplitude from the upper trapezius muscle in ergonomic studies - A review. *J Electromyogr Kinesiol*. 1995;5(4):197-226.

70. Knutson LM, Soderberg GL, Ballantyne BT, Clarke WR. A study of various normalization procedures for within day electromyographic data. *J Electromyogr Kinesiol*. 1994;4(1):47-59.

71. Mirka GA. The quantification of EMG normalization error. *Ergonomics*. 1991;34(3):343-352.

72. Oddsson LI, Giphart JE, Buijs RJ, Roy SH, Taylor HP, De Luca CJ. Development of new protocols and analysis procedures for the assessment of LBP by surface EMG techniques. *J Rehabil Res Dev*. 1997;34(4):415-426.

73. Soderberg GL, ed. ***Selected topics in surface electromyography for use in the occupational setting: Expert perspectives***. 1992nd ed. Rockville: U.S Department of HEalth and Human Services; 1992.

74. Burden A, Bartlett R. Normalization of EMG amplitude: An evaluation and comparison of old and new methods. *Med Eng Phys*. 1999;21(4):247-257.

75. Kasman GS, Cram JR, Wolf SL. *Clinical applications in surface electromyography: Chronic musculoskeletal pain.* Aspen; 1998.

76. Farina D, Holobar A, Merletti R, Enoka RM. Decoding the neural drive to muscles from the surface electromyogram. *Clin Neurophysiol*. 2010;121(10):1616-1623.

77. Hanada EY, Hubley-Kozey CL, McKeon MD, Gordon SA. The feasibility of measuring the activation of the trunk muscles in healthy older adults during trunk stability exercises. *BMC Geriatr*. 2008;8:33-2318-8-33.

78. Hubley-Kozey CL, Hanada EY, Gordon S, Kozey J, McKeon M. Differences in abdominal muscle activation patterns of younger and older adults performing an asymmetric leg-loading task. *PM R*. 2009;1(11):1004-1013.

79. Kendall FP. *Muscles, testing and functions: With posture and pain.* Lippincott Williams \& Wilkins; 2005.

80. Vera-Garcia FJ, Moreside JM, McGill SM. MVC techniques to normalize trunk muscle EMG in healthy women. *J Electromyogr Kinesiol*. 2010;20(1):10-16.

81. Falla D, Dall'Alba P, Rainoldi A, Merletti R, Jull G. Repeatability of surface EMG variables in the sternocleidomastoid and anterior scalene muscles. *Eur J Appl Physiol*. 2002;87(6):542-549.

82. McGill SM. Electromyographic activity of the abdominal and low back musculature during the generation of isometric and dynamic axial trunk torque: Implications for lumbar mechanics. *J Orthop Res*. 1991;9(1):91-103.

83. Rutherford DJ, Hubley-Kozey CL, Stanish WD. Maximal voluntary isometric contraction exercises: A methodological investigation in moderate knee osteoarthritis. *J Electromyogr Kinesiol*. 2011;21(1):154-160.

84. Hubley-Kozey CL, Deluzio KJ, Landry SC, McNutt JS, Stanish WD. Neuromuscular alterations during walking in persons with moderate knee osteoarthritis. *J Electromyogr Kinesiol*. 2006;16(4):365-378.

85. Hermens HJ, Merletti R, Freriks B. *SENIAM*. Enschede, The Netherlands: Roessingh Research and Development; 1999.

86. Angrisani L, Moriello RSL, D'Arco M, Greenhall C. Optimal bandpass sampling strategies for enhancing the performance of a phase noise meter. *Meas Sci Technol*. 2008;19(10):1-11.

87. Duiverman ML, van Eykern LA, Vennik PW, Koeter GH, Maarsingh EJ, Wijkstra PJ. Reproducibility and responsiveness of a noninvasive EMG technique of the respiratory muscles in COPD patients and in healthy subjects. *J Appl Physiol (1985)*. 2004;96(5):1723-1729.

88. Ng JK, Parnianpour M, Kippers V, Richardson CA. Reliability of electromyographic and torque measures during isometric axial rotation exertions of the trunk. *Clin Neurophysiol*. 2003;114(12):2355-2361.

89. Winter DA. *Biomechanics and motor control of human movement.* Wiley; 2009.

90. Beaver WL, Wasserman K, Whipp BJ. A new method for detecting anaerobic threshold by gas exchange. *J Appl Physiol (1985)*. 1986;60(6):2020-2027.

91. TAYLOR HL, BUSKIRK E, HENSCHEL A. Maximal oxygen intake as an objective measure of cardio-respiratory performance. *J Appl Physiol*. 1955;8(1):73-80.

92. American College of Sports Medicine, Whaley MH, Brubaker PH, Otto RM, Armstrong LE. *ACSM's guidelines for exercise testing and prescription.* Lippincott Williams \& Wilkins; 2006.

93. Evans JD. *Straightforward statistics for the behavioral sciences.* Brooks/Cole Publishing Company; 1996.

94. Poulsen MK, Thomsen LP, Mifsud NL, et al. Electrical activity of the diaphragm during progressive cycling exercise in endurance-trained men. *Respir Physiol Neurobiol*. 2015;205:77-83.

95. Johnson BD, Babcock MA, Suman OE, Dempsey JA. Exercise-induced diaphragmatic fatigue in healthy humans. *J Physiol*. 1993;460:385-405.

96. Mattson JP, Miller TA, Poole DC, Delp MD. Fiber composition and oxidative capacity of hamster skeletal muscle. *J Histochem Cytochem*. 2002;50(12):1685-1692.

97. Richmond FJ, Singh K, Corneil BD. Neck muscles in the rhesus monkey. I. muscle morphometry and histochemistry. *J Neurophysiol*. 2001;86(4):1717-1728.

98. Thompson WR, American College of Sports Medicine, Gordon NF, Pescatello LS. *ACSM's guidelines for exercise testing and prescription.* Lippincott Williams \& Wilkins; 2010.

99. Carey DG, Pliego GJ, Rohwer JL. The ventilatory response to incremental exercise: Is it one or two breakpoints? *J Strength Cond Res*. 2010;24(10):2840-2845.

100. Jacobs I. Blood lactate. implications for training and sports performance. *Sports Med*. 1986;3(1):10-25.

101. Sinderby C, Spahija J, Beck J, et al. Diaphragm activation during exercise in chronic obstructive pulmonary disease. *Am J Respir Crit Care Med*. 2001;163(7):1637-1641.

102. Jolley CJ, Luo YM, Steier J, et al. Neural respiratory drive in healthy subjects and in COPD. *Eur Respir J*. 2009;33(2):289-297.

103. O'Donnell DE, Webb KA. Exertional breathlessness in patients with chronic airflow limitation. the role of lung hyperinflation. *Am Rev Respir Dis*. 1993;148(5):1351-1357.

104. Kim KS, Byun MK, Lee WH, Cynn HS, Kwon OY, Yi CH. Effects of breathing maneuver and sitting posture on muscle activity in inspiratory accessory muscles in patients with chronic obstructive pulmonary disease. *Multidiscip Respir Med*. 2012;7(1):9-6958-7-9.

Appendix A:

Screening Document

Date:_____

Participant #:_____
Age:_____
Sex:_____

Healthy

	Yes	No
Have you been hospitalized in the past month?		
Do you have any heart issues? History of a heart attack Angina Uncontrolled arrhythmias		
Do you have any lung diseases?		
Do you use supplemental oxygen?		
Do you have any joint or muscle issues that would make it difficult for you to ride a bike?		

Comments:_____
_____
_____
_____

Did the individual agree to participate in the study? _____

Date of Testing Visit:

_____

Appendix B:

Questions: "Please rate your breathing exertion." &   "Please rate your leg discomfort"

10-Point Modified Borg Scale

rating	description
0	NOTHING AT ALL
0.5	VERY, VERY LIGHT
1	VERY LIGHT
2	FAIRLY LIGHT
3	MODERATE
4	SOMEWHAT HARD
5	HARD
6	
7	VERY HARD
8	
9	
10	VERY VERY HARD (MAXIMAL)

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Borg G.A. Psychophysical bases of perceived exertion. *Medicine and Science in Sports and Exercise*. 1982; 14:377-381.

The American Thoracic Society (ATS) defines dyspnea as subjective experience of breathing discomfort that consists of specific sensations that varies in intensity. Dyspnea is composed of a sensory dimension and an affective dimension, both align with the definition of dyspnea but they are not mutually exclusive. The sensory dimension describes the intensity of breathing discomfort and the affective dimension refers to the experience of unpleasantness. When choosing how to assess dyspnea, it is important to know what dimension you want to measure. Specifically for this study we were interested in assessing the sensory dimension of dyspnea. We used the Borg scale and asked our participants to rate their breathing exertion.

Appendix C:

Graded Exercise Test

Date:_____   Participant:_____

Age:_____   Height: _____

Sex: _____   Weight:_____

	Resting Stage	Unloaded Pedaling	Stage 1	Stage 2	Stage 3	Stage 4	Stage 5
Breathing Exertion							
Leg Discomfort							

	Stage 6	Stage 7	Stage 8	Stage 9	Stage 10	Stage 11	Stage 12
Breathing Exertion							
Leg Discomfort							

	Stage 13	Stage 14	Stage 15	Stage 16
Breathing Exertion				
Leg Discomfort				

Reason for ending the test: Leg discomfort

Breathing discomfort

Both breathing and leg discomfort

Unable to continue safely

Unable to maintain rpm

Other reason

Appendix D:

Exercise Stages	RMPs	Watts
1	60	26
2	60	28
3	60	41
4	60	55
5	60	70
6	60	85
7	60	100
8	60	113
9	60	126
10	60	136
11	60	150
12	60	165
13	60	180
14	60	199
15	60	219
16	60	238

Appendix E:

Preliminary Data

1.1   Project Data

In PHYT 5300.03: Skeletal Muscle Function through Surface Electromyography I conducted a study to determine the feasibility of assessing accessory respiratory muscle activation using surface EMG, during a graded cycle ergometer test. Activation would be assessed over a single breathing cycle at each stage of the test.

Specific objectives were:

   a. To determine whether it was possible to identify and subsequently record EMG from the inspiratory and expiratory phase of the breathing cycle.

   b. To determine if there were differences in the %MVIC muscle activation of the SCM and the scalene throughout the GXT.

   c. To determine the feasibility of using an upright bike, given the potential need for shoulder girdle bracing and associated contamination of the EMG signals at higher exercise levels.

1.1.1   Methods:

One male (35 years, 178 cm, 69.7 kg) subject with no previous respiratory diseases performed a GXT on an upright cycle ergometer. Surface electrodes (Ag/AgCl 10mm diameter, Red Dot) were placed over two respiratory muscle sites on the right side, based on placements suggested in the research.[66,73,81] The muscles investigated were the SCM and scalene. The electrode placement for the scalene was the posterior triangle of the neck at the level of the cricoid cartilage and the SCM the placement was 1/3 of the

distance from the sternal notch to the mastoid process. Minor location adjustments were made based on individual anthropometrics. MVIC trials were performed based on one maximum contraction suggested by Kendall's Muscles: Testing and Function book, anterolateral neck flexion.[79]

The participant performed a graded bike test on an Ergoline Bike mechanically braked bike. The test started at 40watts and increased by 20 watts every 2 minutes. The participant wore an Oxycon™ Mobile (VIASYS Healthcare, GmBH, Germany), portable metabolic unit throughout the exercise test. The unit collects breath-by-breath data, while the participant breathes through a mask. Ventilatory parameters: RR, V_T, V_E, oxygen consumption (V_{O2}), and carbon dioxide production (V_{CO2}), were collected throughout the exercise test.

During the exercise test, the EMG data was collected over one breath in the last 10 seconds of the exercise stage. The research assistant followed the breathing cycle, visible on the computer screen. The research assistant pressed the footswitch at the beginning of inspiratory, the beginning of expiration and at the beginning of the subsequent inspiration to trigger EMG collection. The raw EMG signal was pre-amplified 200-times and additional amplification (bandpass 10-1000Hz, CMRR=115db at 60Hz, input impedance approximately $10G\Omega$, ± 4V) was performed using an eight channel surface EMG system (AMT-8 EMG, Bortec Inc., Calgary, Alberta). Using an A/D converter (National Instruments BNC-2090 16-bit ± 10V) the raw EMG signal was digitized at 2000Hz. The EMG data was processed using a custom Matlab™ (MathWorks

Inc., Natick, MA, USA) code[2] that used a 2nd order, recursive filter. The data was full wave rectified, corrected for the subject and system bias. A lowpass 2nd order Butterworth filter with a cutoff frequency of 6Hz was used on the rectified data to create a linear envelope providing a profile of the myoelectrical activity of the muscles over one breathing cycle. The MVIC values were processed using a 0.1ms moving window to find the maximum EMG activation for amplitude normalization.

1.1.2 Results:

The participant completed 10 stages of the exercise test, up to 220 watts. The exercise test was terminated due to leg discomfort. A linear increase in muscle activation was seen as workload increased (Table 11) throughout the exercise test because the high R^2 value indicated a good fit of the linear model (Figure 12). The scalene and SCM displayed significant increases in activation during the GXT. Both the scalene and SCM displayed an activation pattern showing that they were active during inspiration (0-50% of the breathing cycle) as seen in Figure 13. The scalene muscle activation was higher than that of the SCM, reaching a muscle activation of 14% MVIC and 8.5% MVIC respectively during the last bike stage (220watts).

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[2] Custom Matlab Code written by Colleen Dewis (PhD Candidate), Department of Industrial Engineering

Table 11. Activation for the scalenes and SCM during the GXT

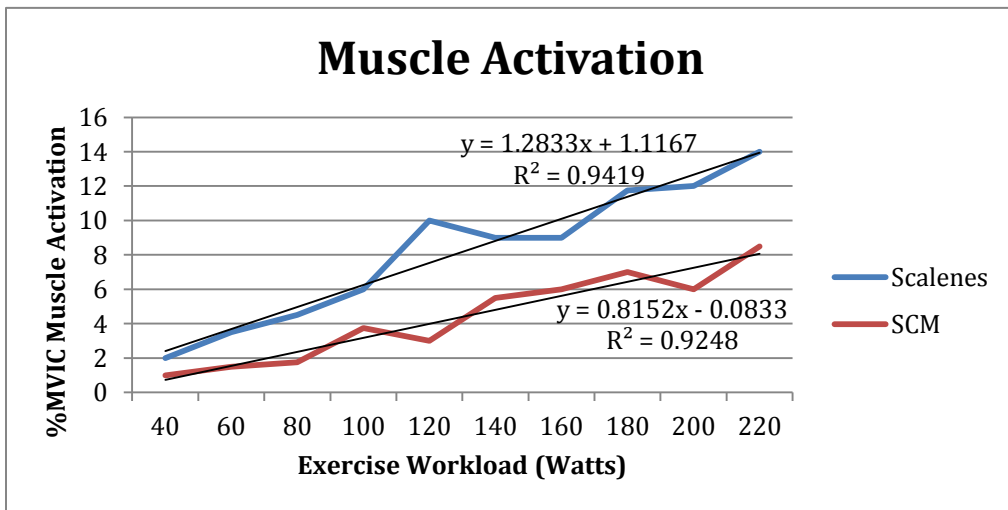| Exercise Stage (Watts)	% MVIC Activation: Scalenes	% MVIC Activation: SCM
40	2.0	1.0
60	3.5	1.5
80	4.5	1.75
100	6.0	3.75
120	10.0	3.0
140	9.0	5.5
160	9.0	6.0
180	11.75	7.0
200	12.0	6.0
220	14.0	8.5

Data is expressed as a % of the MVIC

Figure 12. Normalized muscle activation response to increased exercise intensity.
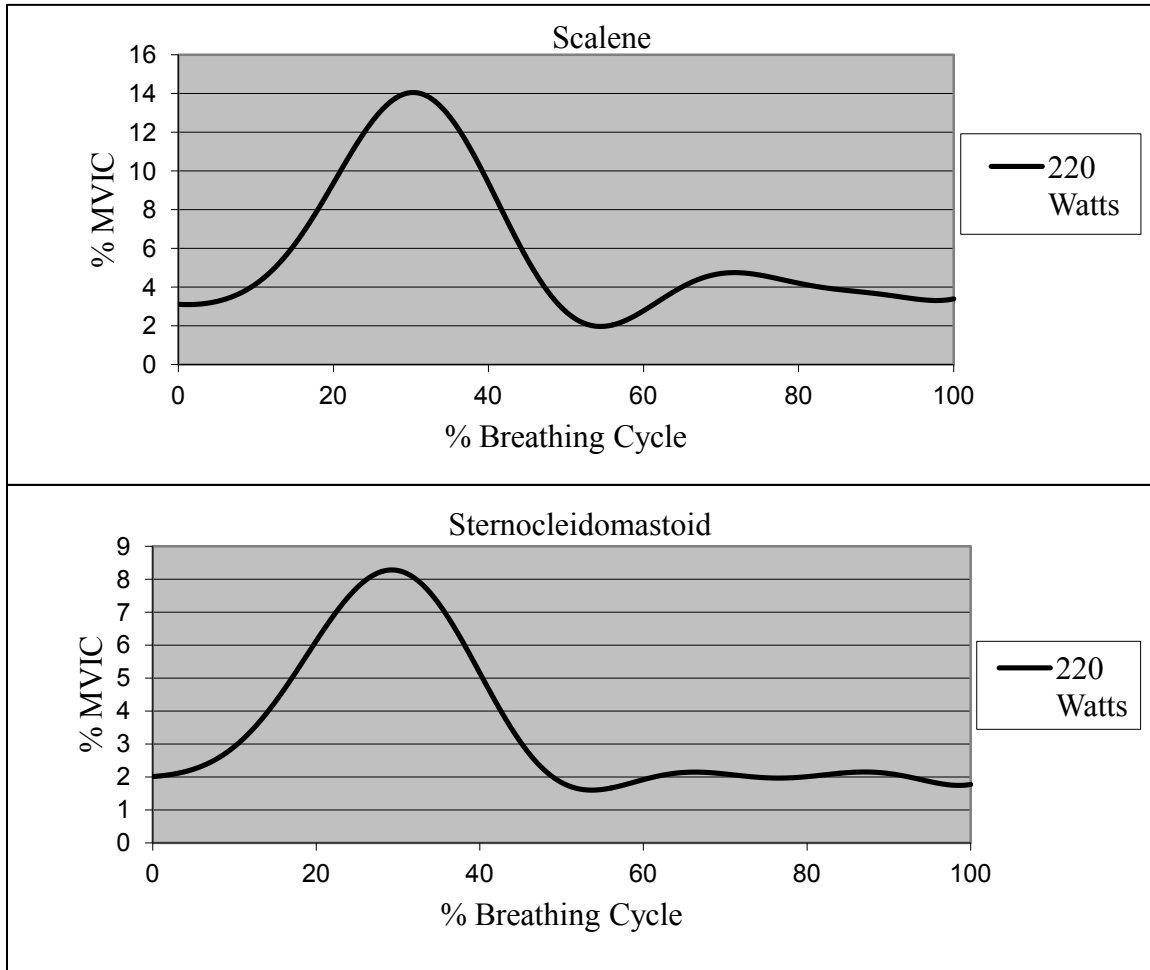
Figure 13. MVIC normalized EMG data for the scalene and SCM over one breathing cycle**.** [inspiration (0-50%); expiration (50-100%)] during the last stage of the graded exercise test (220 watts).

1.1.3   Discussion:

The participant demonstrated an increase in accessory respiratory muscle activation during the GXT. It was possible to identify the breathing cycle from the EMG signal, with both muscles displaying significant muscle activation during inspiration. The study results confirmed existing literature stating that the scalenes are active during quiet breathing and therefore are considered primary respiratory muscles.[49] I identified some important limitations in the study methodology:

a. Human error using the footswitch to identify the phases of the breathing cycle and trigger EMG collection. There was a slight delay between the individual breathing and the breathing cycle that appeared on the computer monitor. As the participant's RR increased it became even more difficult to press the footswitch at the appropriate points during the breathing cycle. Future research looking to evaluate EMG activation over a breathing cycle should record a series of breaths and pick out the breathing cycles.

b. Electrode size and potential crosstalk – when using surface electromyography one must be aware of signal crosstalk especially when assessing small muscles, particularly when they are close together and have similar activities.[21] There are ways to minimize the crosstalk; one of the most effective is to use smaller electrodes with a smaller detection size. It is possible that crosstalk was an issue in this data collection; therefore I will use 5mm diameter electrodes in my subsequent research.

c. Use of an upright cycle ergometer – A typical position for an individual with COPD is to lean forward and brace themselves to place the respiratory muscles in a more optimal position to fulfill the ventilatory needs. Previous research has also shown that this position increases scalene and SCM activation.[104] Therefore we wanted to avoid any form of bracing during the exercise test such as leaning forward and holding onto the handlebars of the bike. In this study we had advised our participant to avoid holding onto the handlebars during the exercise test; however he was not able to do this during the last two stages of the test. It is possible that this may have increased the muscle activation of the scalenes and

SCM and created erroneous results. Therefore, in subsequent research I will use a semi-recumbent bike, as Duiverman and colleagues did.

1.2   Preliminary Data:

To address the study limitations reported above and to explore the best approach to EMG processing, I collected data on two subjects who would be similar to my participant population. The aim of this work was:

a. To determine the appropriate cut-off frequency for the linear envelope processing.

b. To determine how many breathing cycles are required to get a true representation of the maximum %MVIC of the scalenes and SCM during the exercise test.

c. To determine what MVIC exercise elicited a maximal contraction.

d. To determine the feasibility of using a recumbent bike.

1.2.1   Methods:

One male and one female over the age of 40 with no respiratory diseases performed a GXT on a recumbent cycle ergometer (Vision Fitness). Surface electrodes (Ag/AgCl 5mm diameter, Duo-Trode) were placed over the SCM and the scalene on the right side.[66,73,81] The electrode placement for the scalene was the posterior triangle of the neck at the level of the cricoid cartilage and the SCM the placement was 1/3 of the distance from the sternal notch to the mastoid process. Minor location adjustments were made based on individual anthropometrics. Three maneuvers were chosen for the MVIC procedure based on previous work[81] and muscle function testing procedures.[79] The MVIC

maneuvers for the scalene and SCM muscle tests include anterolateral neck flexion, forward neck flexion and lateral neck flexion.

The participants performed a graded bike test on the bike. The test started at 26watts and increased resistance every minute. The participants wore an Oxycon[TM] Mobile (VIASYS Healthcare, GmBH, Germany), as in the previous data collection described above. During the exercise test, the EMG data was collected 20 seconds into the exercise stage for 20-second duration, to capture a series of breathing cycles and perceived breathing and leg exertion was recorded at every exercise stage. Inspiratory capacity maneuvers were performed during even stages.

1.2.2 EMG Data Processing:

The processing for the raw EMG data was determined based on the breathing cycle. The frequency content of the breathing cycle is determined by the respiratory rate and measured in hertz (Hz), representing cycles per second. For the purpose of this study I considered using a resting respiratory rate of 15 breaths per minute and a respiratory rate of 47 breaths per minute at maximal exercise. This corresponds to 0.25Hz and 0.75 Hz respectively.

A sampling rate refers to the frequency with which the signal is collected and it is expressed in Hz. The sampling rate needs to be sufficient so as to provide a signal without any attenuation or aliasing.[86] To achieve this, the Nyquist Theorem was followed, which states that a signal must be sampled at a frequency that is greater than twice the highest frequency of the signal.[86] For EMG, the highest frequency is 500Hz, therefore based on the Nyquist Theorem; a sampling rate of at least 1000Hz is required to

avoid signal loss.[21,73] Previous research investigating respiratory muscle activity with surface EMG found that sampling at 2000Hz, provided a good representation of the signal in both healthy individuals and individuals with COPD during conditions of increased ventilatory demand.[19,87]

The raw EMG signal was pre-amplified 200-times and additional amplification (bandpass 10-1000Hz, CMRR=115db at 60Hz, input impedance approximately $10G\Omega$, $\pm$ 4V) was performed using an eight channel surface EMG system (AMT-8 EMG, Bortec Inc., Calgary, Alberta). Using an A/D converter (National Instruments BNC-2090 16-bit $\pm$ 10V) the raw EMG signal was digitized at 2000Hz. The EMG data was processed using a custom Excel template[3] that used a 2nd order, non-recursive filter. The Butterworth filter cut off coefficients were calculated in excel using standard equations.[89] The data was corrected for the subject bias and gain and converted to μV. The data was then full wave rectified. A lowpass 2nd order Butterworth filter was used with three different cutoff frequencies (2Hz, 6Hz and 100Hz) to create a linear envelope, to determine which one was suitable for future research. Since the frequency content for the breathing cycle is between 0.25-0.75Hz, 2 Hz is the lowest cutoff frequency to ensure that we are not eliminating any of the signal.

1.2.3 Results:

Each participant was able to complete the GXT on the recumbent cycle ergometer. Figure 14 shows progressive activation of the SCM and scalene muscles as

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[3] Custom Excel template written by Dr. John Kozey, School of Biomedical Engineering, School of Health and Human Performance & Department of Industrial Engineering

exercise intensity increases in one of the subjects. A similar pattern was seen in the second participant.
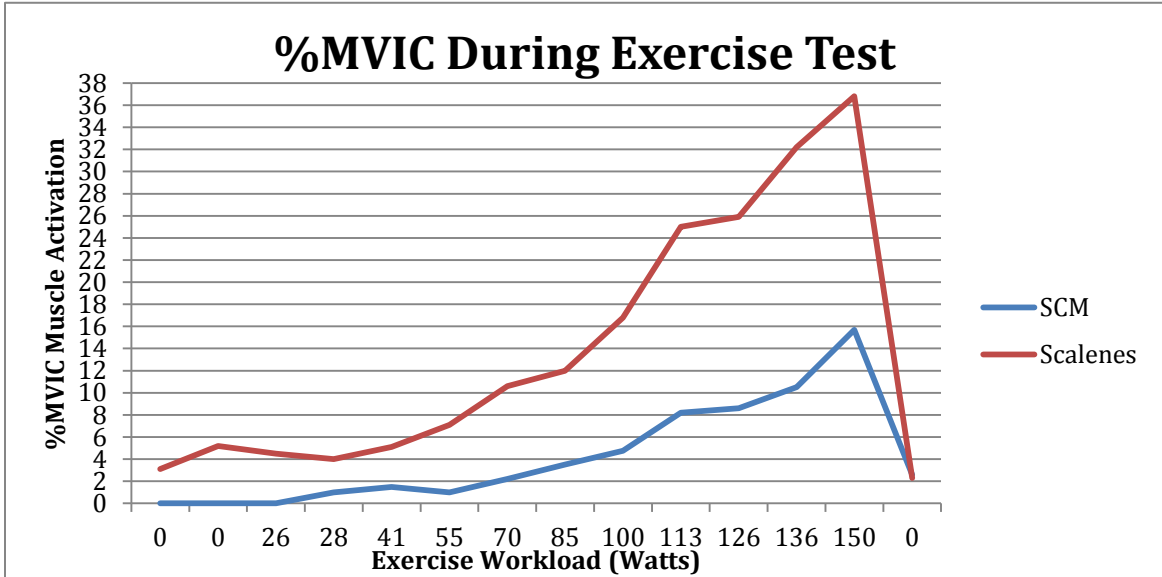

Figure 14. EMG data during the GXT. The two 0s at the beginning of the workload correspond to the rest and reference stage and the 0 at the end represents recovery.

Figure 15 shows the EMG signal processed at the three cutoff frequencies. A frequency cutoff of 2Hz produces data that shows the peak activation most clearly and does not introduce additional noise.
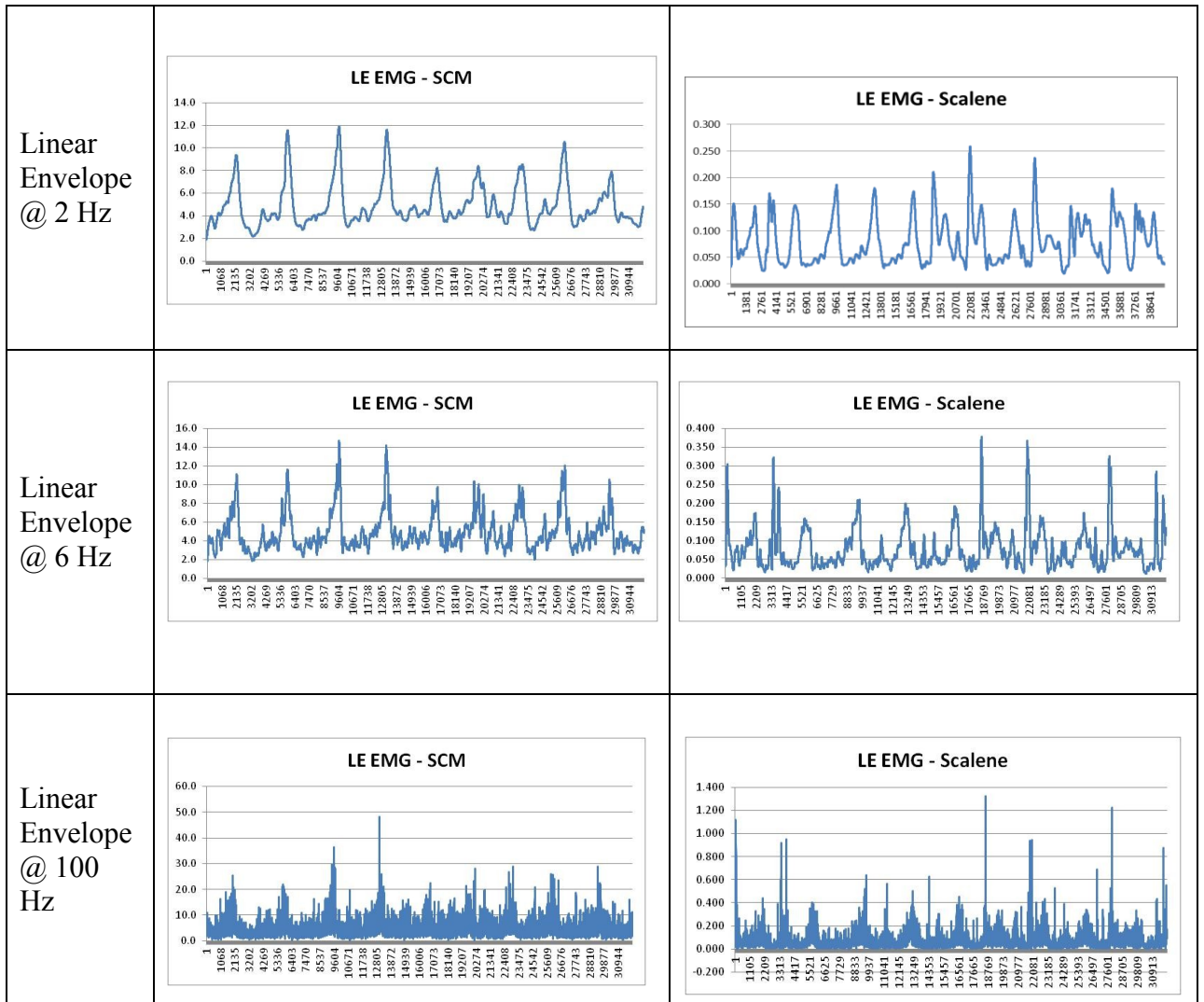
Linear Envelope @ 2 Hz	LE EMG - SCM	LE EMG - Scalene
Linear Envelope @ 6 Hz	LE EMG - SCM	LE EMG - Scalene
Linear Envelope @ 100 Hz	LE EMG - SCM	LE EMG - Scalene

Figure 15. Processed EMG signal with various cutoff frequencies. (LE – Linear envelope) x-axis: time y-axis: micro volts

The results from the project data demonstrated that it would not be possible to use a single breathing cycle for data processing. Therefore when collecting data from these two subjects I chose to acquire 20 seconds of data at the end of each exercise stage. Once the 20 seconds of EMG data was processed I identified the peaks from each breathing cycle, calculated the mean of the peaks and then calculated ±10% of the peak activation to see which peaks fell within that window. The peaks that were within the 10% window were then used to recalculated the peak muscle activation of both muscles.
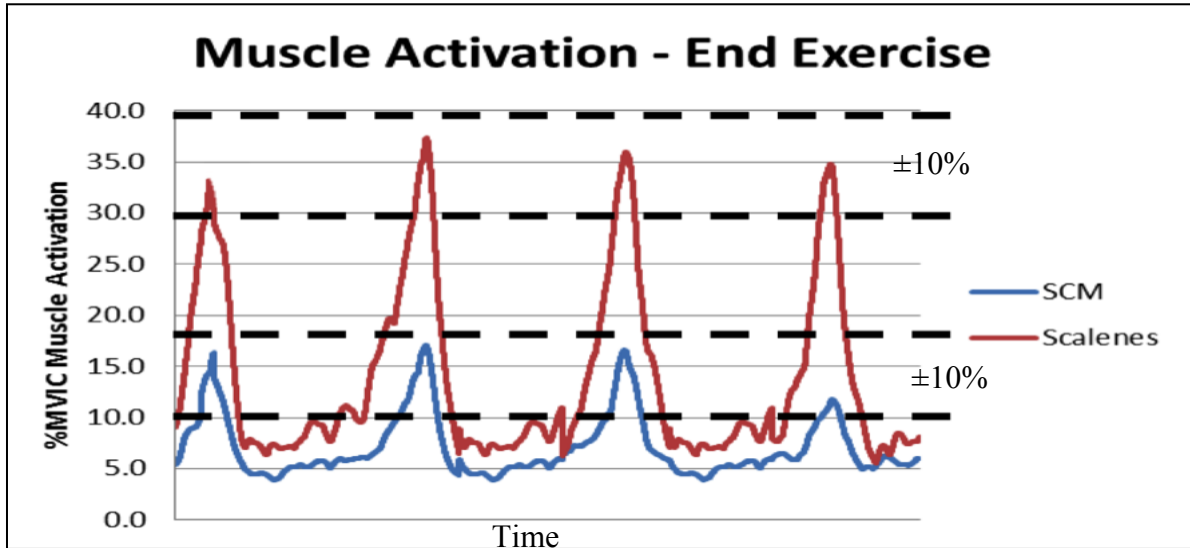
Figure 16. Determining the peak muscle activation

1.2.4 Discussion:

Two healthy participants, similar to our participant population were able to successfully complete the study visit based on the methodology adaptations that were made as a result of the previous project study limitations. Both participants demonstrated an increase in accessory respiratory muscle activation during the GXT. I was able to resolve the issues involving the cutoff frequency and the number of breathing cycles that are required to determine the peak %MVIC for each exercise stage, since there is no previous literature in the respiratory field. Based on the breathing cycle and testing different cutoff frequencies, 2Hz is the cutoff frequency that should be used because it does not introduce any unnecessary noise. A 10% window should be used to determine the peak muscle activation for each exercise stage.

The participants were able to perform the exercise test on the recumbent bike without having to grip the handlebars; therefore I am confident that the muscle activation

is coming from the participant's breathing. The final objective of the study was to determine if one of the three MVIC exercises elicited a maximal contraction in both participants. None of the MVIC exercises were superior, in fact the maximal contraction for each participant came from a different exercise, and therefore I would recommend that all three MVIC maneuvers be used when investigating these muscles.

My thesis will follow the methodology from my preliminary data, as I have been able to resolve the data collection and processing issues that were present after my initial feasibility study. I am confident that moving forward to my thesis participant population, I will be able to collect and process the EMG activation from the scalene and SCM to investigate my proposed objectives.