The Human Respiratory System

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Introduction:

The respiratory system allows gas exchange between cells in the body and the atmosphere. The purpose for respiration is to obtain oxygen for cells and eliminate waste and carbon dioxide from the body. The major organ responsible for respiration is lung. There are two types of respiration that occur in the body; internal respiration and external respiration. Internal respiration is an intracellular metabolic process occurring within the mitochondria, using oxygen to produce carbon dioxide (Sherwood, 2007). External respiration is the exchange of O2 and CO2 between the atmosphere and the cells of the body. External respiration has four stages: 1) ventilation (the physical exchange of gases between the atmosphere and alveoli), 2) alveolar gas exchange (exchange of O2 and CO2 between the alveoli and the blood in the pulmonary capillaries, 3) gas transport (transport of O2 and CO2 from the lungs to the tissues of the body by the blood, 4) blood gas exchange (exchange of O2 and CO2 between the blood and the tissue (Sherwood, 2007). The Respiratory System is responsible for the first two stages. These steps involve the respiratory airways. The airways begin with the nasal passage, which open into the pharynx. The larynx is located at the entrance to the trachea, which divides into two main branches called bronchi. The bronchi further divide into bronchioles, at the ends of which are alveolar ducts, which are small, thin walled sacs where is the site of gas exchange in the lung (Sherwood, 2007). Air enters through the nose or mouth then passes through the nasopharynx/oropharynx, the glottis and larynx and then to the tracheobronchial tree. Atmospheric pressure, intra-alveolar pressure, and the intrapleural pressure are important pressures in ventilatio. In a resting lung the intra-alveolar pressure is the same as the atmospheric pressure, whereas the intrapleural pressure is always less than the intra-alveolar and atmospheric pressure. Transmural pressure gradient always exits and serves to stretch the lungs to fill the

thoracic cavity. The air pressure gradients between the alveoli and the atmosphere cause the flow of air into and out of the lungs. As the thoracic cavity expands and increase in volume the pressure inside the lungs decreases (Boyal's law PV=constant) allowing for the air flow from an area with high pressure (atmosphere) to an area with lower pressure (alveoli), this will result in inspiration. As the inspiratory muscles that contracted during inspiration relax and the elastic recoil of the lungs increases the alveolar pressure above that of atmospheric pressure so air moves out of the lungs, resulting in expiration (Sherwood, 2007). Expiration is passive during normal quiet breathing. 0.5L of air moves in and out of the lungs in each normal breathing, this is called tidal volume (TV). Additional volume of gas that can be inhaled above TV during a forced maximal inspiration is called Inspiratory reserve volume (IRV), which can reach up to 1.9L to 3.0L. The expiratory reserve volume (ERV) is the extra volume of air (0.7-1200L) that can be forced out of the lungs after normal TV expiration. 1.1L to 1.2L of air will always remain in the lungs to keep alveoli open and prevent collapsing of lung; this is the residual volume (RV). The maximum volume of air that can be exchanged is called Vital capacity (VC), which can be calculated by adding TV, IRV, and ERV (wikipedia).

The pons and the medulla regions of the brainstem generate respiration. The medulla oblongata controls breathing by responding to changes in PO2, PCO2, and pH levels. The medullary respiratory center is made up of the dorsal respiratory group and the ventral respiratory group.

The dorsal respiratory group (DRG) processes information from the chemoreceptors and lungs in order to modulate inspiration. The inspiratory neurons from the DRG has its fibers terminate on the motor neurons that innervate the inspiratory muscles. The ventral respiratory group (VRG) remains inactive during normal, quiet breathing. It takes information from the dorsal respiratory group and regulates inspiration and expiration after sensing changes in arterial gases,

which occurs during periods of increased ventilation demand. The VRG is especially important during active expiration. Pulmonary stretch receptors in the airways, when stretched, cause the inspiratory neurons to decrease firing which prevents over inflation of the lungs (Hering-Breur reflex).

The focus on this study will be on external respiration, the process of exchanging oxygen and carbon dioxide between the atmosphere and the cells of the body. In part one of this study the static lung volume will be measured. The tidal volume, inspiratory reserve volume, the expiratory reserve volume, and the vital capacity will be measured. In part two of this study the effects of inspired gas composition on respiration will be examined. The percent CO2 before and after breath-hold will be measured. The hypothesis is to see a higher percentage of CO2 before breath-hold in the Re-breathing, than in the normal breathing and hyperventilation. The percent of CO2 after breath-hold should be the same for all three since the breath is being held to the same level of discomfort. The time of breath-hold should be longest for Hyperventilation, since there is more oxygen in the lungs that can be exchanged with the blood, and shortest for re-breathing from a bag, since CO2 is being breathed back in and no new O2 is coming in. In part three of this study the effect of exercise on respiration will be examined. The hypothesis is to see an increase in ventilation just before the start of exercise and a gradual increase in the ventilation as workload is increases.

Materials and Methods:

Set up: nose clip, disposable cardboard tube mouthpiece, stopwatch, filter, spirometry station

(Bautista & Korber 2008).

The materials and methods of this experiment follow procedures listed in the NPB 101L Physiology Lab manual. In part one static lung volumes were measured. The subject's nose was

covered by a nose clipper and mouth piece was inserted into the subject's mouth. Subject was asked to breathe normally with complete inhale-exhale cycle. The exercise repeated for breath of 10-12 cycles for length of similar size breath, inhale deeply, 5 cycle of normal breath and exhale as deeply as possible followed by breathing normally for 5 cycles. Data regarding IRV, ERV, TV, and VC were recorded in table 1 of the lab manual. Minute ventilation and alveolar ventilation were calculated using $V_{E=}$TVxRR, V_{DS}=DS x RR, and $V_A=V_E-V_{DS.}$ (Bautista 2008).

In part two, effects of inhaled gas composition and lung volume on respiration were examined. The subject changed arterial P_{CO2} using a bag. Subject had a mouthpiece on while re-breathing into a bag. Length of breath-hold was measured and end-title CO_2 before and after breath-hold was recorded for normal, hyperventilation, and re-breathing exercises. In part 2 effects of lung volume on respiration and breath-hold duration were also examined. Breath-hold duration after normal inspiration, normal expiration, forced inhalation, and forced exhalation were measured following 2 minutes of normal breathing for each case. In the last part of the exercise the BIOPAC system, an exercise bicycle, reusable rubber mouthpiece, nose clip, and stopwatch were used. The subject was asked to sit on the bike and breathe normally for 2 minutes. After 2 minutes the subject started to bike and breath through the CO_2 analyzer. Exercise began in 2 minute intervals of 0, 0.5, 1, 1.5, and 2 kPa and then quickly dropped to 0 kPa. There were no significant changes from lab manual.

Result:

Measuring Static Lung Volumes:

Static lung volumes were measured in part one of this experiment. Results are recorded in table 1 below. BIOPAC software was used to record the peak of normal inspiration and the peak of maximum inspiration. These two data were used in order to measure IRV by getting their

differences. EVR was measured as the difference of trough of maximum exhalation to trough of last normal exhalation. TV was from peak to trough of normal breath.

Table 1 shows static lung volumes. Subject was a UCD female in her early 20s in all measurements. Static lung volume observed during normal breathing, deep inspiration and deep expiration. As expected VC is the largest value and TV is the smallest.

Table 1: static lung volumes

	Volume
IRV	0.921 L
ERV	1.025 L
TV	0.115 L
VC	2.926 L

Minute Ventilation: V_E=TV x RR= 0.115L/breath x 13 (Breath/min)=1.495L/min

Dead space calculation was made using the subject's weight 130lbs=130 ml=0.130L

Alveolar Ventilation (V_A): V_{DS}= DS x RR= 0.13 L x 13 breath/min =1.69 LxBreath/min

$V_A = V_E - V_{DS}$ = 1.495L/min-1.69L.Breath/min= [-0.195] Lx Breath/min

Table 2 shows the alveolar ventilation (calculated by subtracting dead space volume from alveolar ventilation). Dead space volume calculated using dead space (roughly the weight of the subject) times the respiration rate). Minute ventilation calculated by multiplying the TV with respiratory rate. Subject was a UCD female in her early 20's.

Table 2: Alveolar ventilation

Respiratory Rate	13Breath/min
Dead space	0.13L
Minute ventilation	1.495 L/min
Dead space volume	1.69L/min
Alveolar ventilation	0.195L/min

Effects of Inspired Gas Composition and Lung Volume On Respiration:

Table 3 below, shows changes of $C0_2$ percentage from before to after breath-hold and also duration of breath- hold. The duration of breath-hold is longest during the normal breathing trial (31 sec), then hyperventilation (29 sec), and finally re-breathing (4sec). This is as not as it was expected because in normal conditions a decrease in P_{CO2} in hyperventilation will decrease the inspiratory drive and causes the object to be able to hold his/her breath for a longer period of time. There is a decrease by 2 in duration of breath hold from normal breathing to hyperventilation. This can be due to experimental errors. Re-breathing decreased the duration

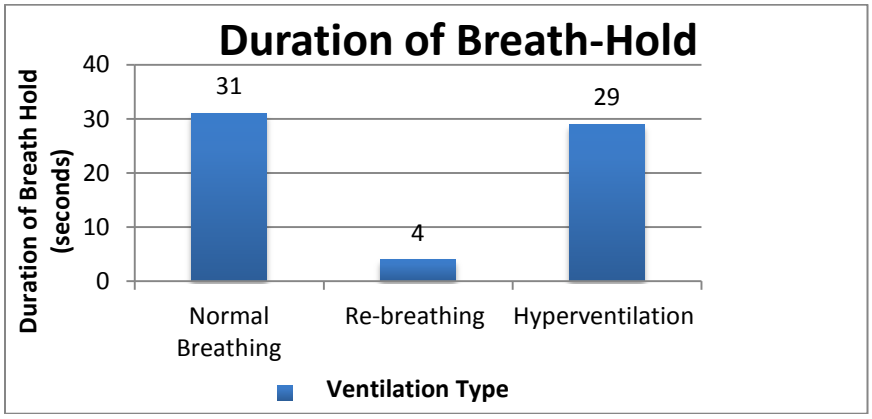
from normal breathing by 27 seconds. CO_2 level showed an increased in all three different breath trials after breath hold compare to before breathe hold. More details in table 3 below.

Table 3 shows results from Breath-holding in Response to normal breathing, re-breathing and hyperventilation. In each case the % of $C0_2$ in air before the breath-hold and after was measured as well as breath-hold duration. Data from time duration is not what it was expected. $C0_2$ levels are higher after breath-hold as expected.

Table 3: Breath-holding Data in Response to normal breathing, re-breathing and hyperventilation

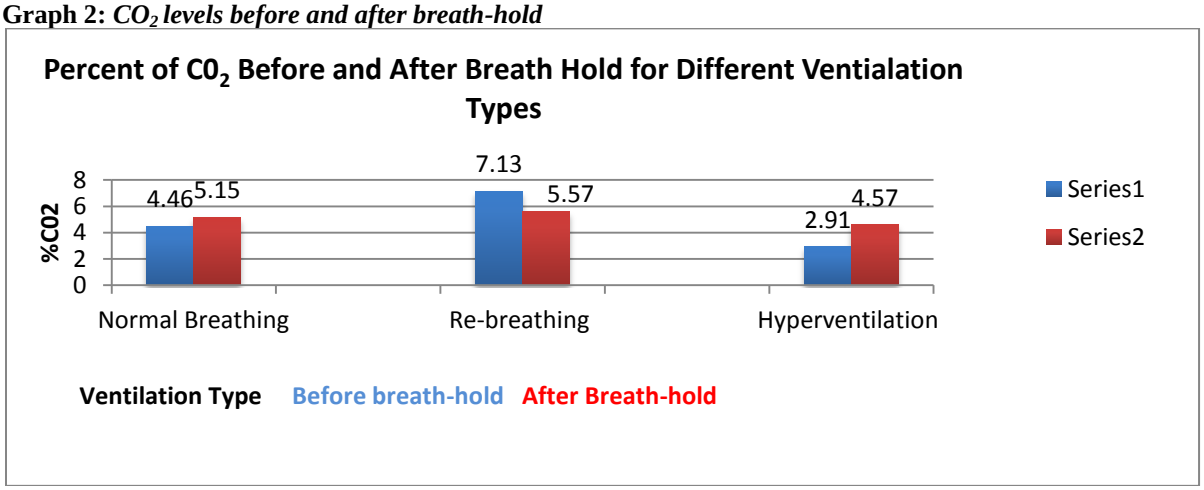
Condition	%$C0_2$ *Before Breath Hold*	%$C0_2$ After Breath-hold	Duration of Breath-Hold
Normal Breathing	4.46 % $C0_2$	5.15 % $C0_2$	31 sec
Re-Breathing	7.13 % $C0_2$	5.57 % $C0_2$	4 sec
Hyperventilation	2.91 % $C0_2$	4.57 % $C0_2$	29 sec

Graph 1: ***Duration for breath-hold for different types of ventilation***



Graph1: The graph above shows values from table 2. Re-breathing in the same air, decreased duration of breath hold. Hyperventilation has increased this the most.

Graph 2: ***CO_2 levels before and after breath-hold***



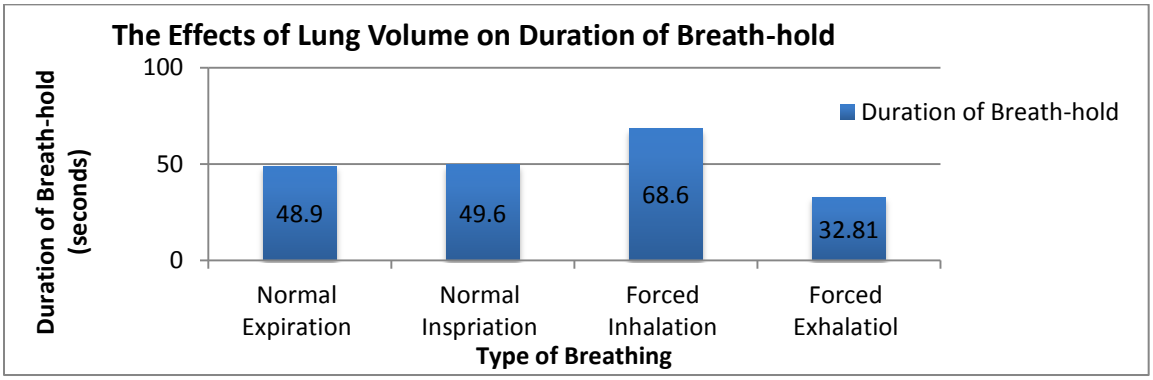
Graph 2: The data above shows values from table 3. Each case shows that CO_2 % increases after breath-hold. Normal breathing shows the greatest increase.

Breath-hold duration considering different volumes of air was also measured in table 4. Graph 3 below shows that forced inhalation leads to the longest duration of breath-hold (1 min& 08 sec) and forced exhalation leads to the shortest breath-hold (32.81 sec).

Table 4: The effects of Lung volume on duration of breath-hold

	Duration of Breath-Hold
Normal Breathing	48.90 Seconds
Normal Inspiration	49.60Seconds
Forced Inhalation	1 min and 08 seconds
Forced Exhalation	32.81Seconds

Graph 3: *The Effects of Lung Volume on Duration on Breath-hold*



Graph 3: The graph above shows the results of table 4. Normal inspiration and expiration duration time were close. Forced inhalation leads to the longest duration of breath-hold (1 min& 08 sec) and forced exhalation leads to the shortest breath-hold (32.81 sec).

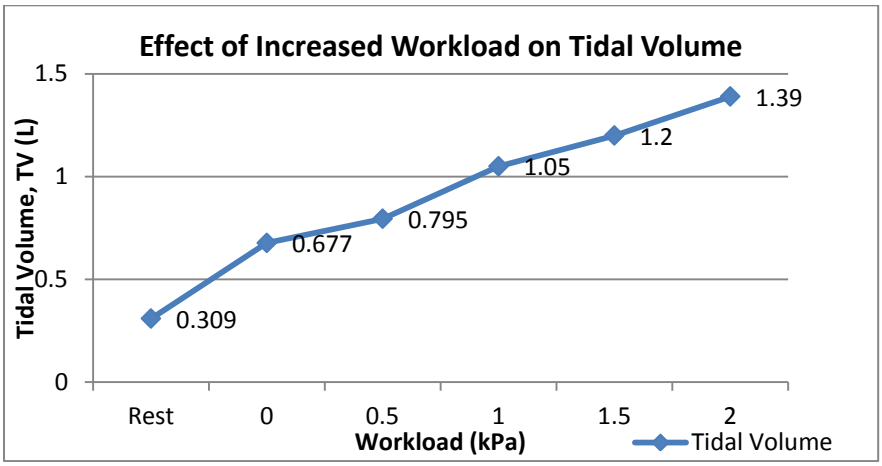
Exercise Hyperne:

Effects of moderate exercise on respiration were examined in this part of study. Workload was increase from 0 kPa up to 2.0 kPa and at the end it was quickly dropped back down to 0.0 kPa. TV, RR, V_E, $FE_{CO2,}$ Minute CO_2 were measured. All values were increased as the workload increased, and a noticeable decrease is seen as 2.0 kPa is reduced back to 0.0 kPa. There was a decrease in RR (from 16-12bpm) and V_E (from 10.83-9.54 L/min) from 0.00k Pa to 0.5kPa workload.

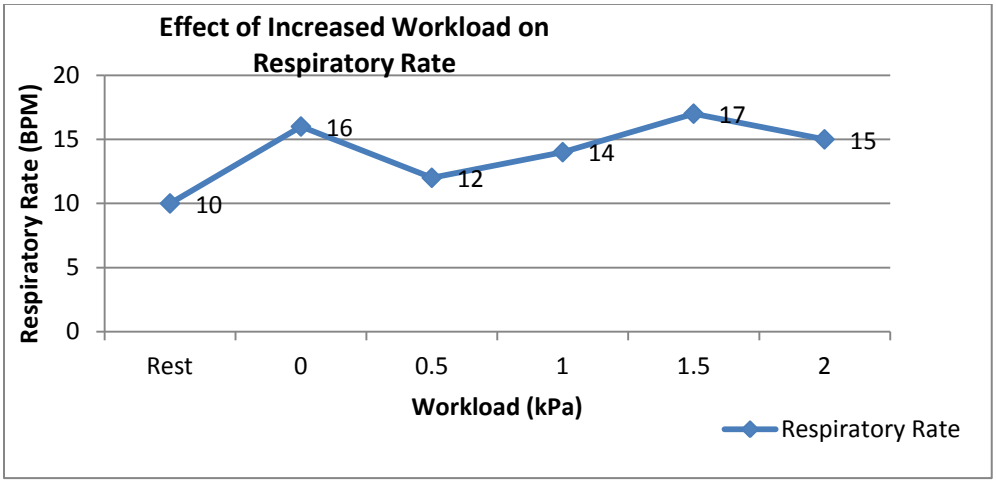
Table 5: table 5 shows values measured in part 3 of the study. Subject was a UCD male in his early 20's.The same subject exercised as workload was increased. The general correlation shows that increasing workload increased all 5 factors. TV, RR, FE_{C02} % were obtained from BIOPAC system.

Table 5 *Ventilatory Response*

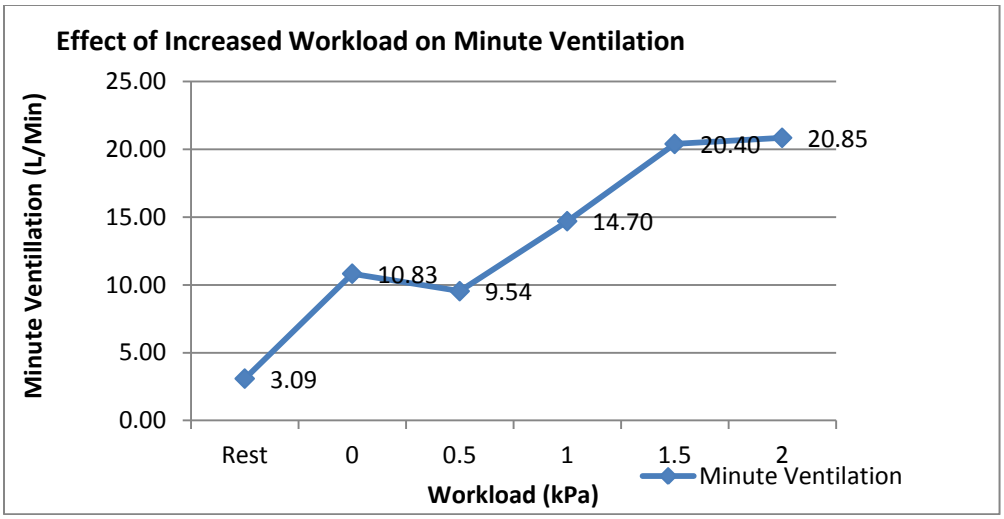
Workload (kPa)	TV (liters)	RR (bpm)	V_E (TVX RR) Lper min	FE_{C02} %	Minute CO_2 (VE X FE_{C02})
Rest	0.309L	10	3.09	4.476	13.830
0.0	0.677L	16	10.83	5.68	61.514
0.5	0.795L	12	9.54	5.76	54.950
1.0	1.05L	14	14.7	5.81	85.407
1.5	1.20L	17	20.4	6.47	131.988
2.0	1.39L	15	20.85	6.66	138.861
Back to 0.0	1.00L	12	12	5.28	63.36



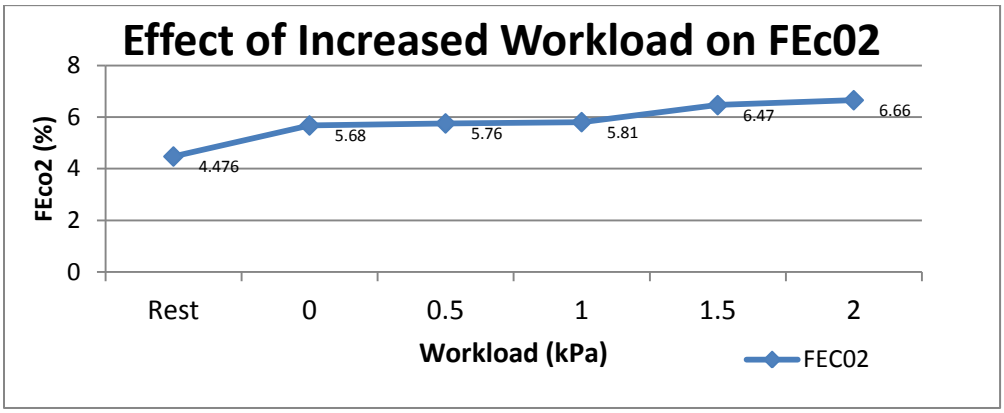
Graph 4 shows the relationship between workload and tidal volume. As workload increases, TV increases. This means higher volume of air is getting in and out of the lungs. BIOPAC system was on mean tool, Ch40 to measure TV which was the cyan color wave. Averages of 5 peaks in the last 40 seconds of each workload level were taken.



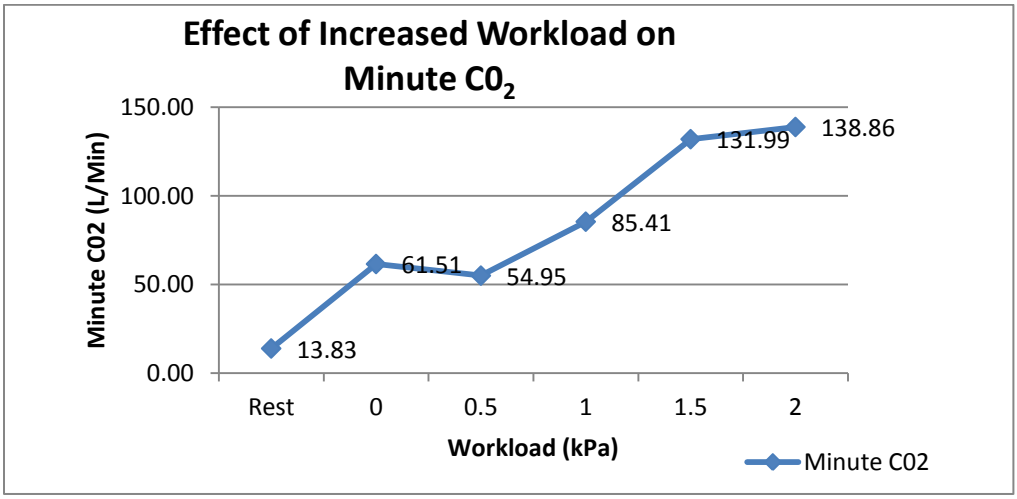
Graph 5 shows an increase in respiratory rate. RR measurements also used cyan color wave and ch40 on BIOPAC. Number of breaths in 15 seconds were collected and then multiplied by four to get BPM.



Graph 6: amount of air inhaled or exhaled per minute is called Minute ventilation (V_E). As workload increased, V_E increased as well. V_E is calculated by multiplying TV and RR.



Graph 7: FE_{C02} % represents the % of $C0_2$ in air at the end of exhalation. This graph shows that as workloads increases the FECO2 increases. For this graph BIOPAC was on Ch2 which shows $C0_2$ % expired, the max tool to find the peak value. Average of 5 peaks in the last 40 seconds of each interval were used.



Graph 8: V_E X FE_{C02} gives us minute $C0_2$. This will give us total percentage of $C0_2$ exhaled per minute. V_E and FE_{CO2} increase as workload increase thus minute CO_2 increases. There is a slight decrease from 0.0 kPa to 0.5 kPa.

Discussion:

The mechanics of inspiration and expiration are due to respiratory muscle and pressure gradients. After expiration right before inspiration, atmospheric pressure and pressure inside the alveoli become equal (Sherwood, 2010). Muscles of the rib cage are called intercostals. The external intercostals help inspiration, and the internal intercostals help expiration. Intercostals and the diaphragm contract enlarging the chest cavity. As the chest cavity enlarges, lungs enlarge causing pressure inside the alveoli to drop below atmosphere pressure (Sherwood, 2010). This pressure gradient, atmospheric pressure and alveolar pressure causes air flow get into the lungs until the gradient ceases. Increase in lung volume, during inspiration alveolar pressure is less than atmospheric pressure so air gets in the lungs until pressures are equal again. During expiration this mechanism is reversed. Inspiration is active because muscles have to be stimulated to initiate this process and at rest expiration consider being a passive process at rest because the respiratory muscles return to their normal positions. Expiration can be active during exercise and hyperventilation (Marieb 2014).When gasses are inside lungs, partial pressure of O_2 and CO_2 determine the diffusion of gases between alveoli and arterioles into the blood supply. Gasses will diffuse from areas of higher partial pressure to areas of lower partial pressure. Blood in the pulmonary circulation has a partial pressure gradient for oxygen to move into the blood and carbon dioxide to move out of the blood in order to have more oxygen to get into tissues and remove the waste from tissues. Normal range of gasses in venous blood is P_{O2} <40mmHg, P_{CO2}>46mmHg and in alveolar air is P_{O2} ~100mHg, P_{CO2}~40mmHg. These gas exchanges provide what body needs to remain in homeostasis condition.

Part 1: Measuring static lung volumes

The pursue for this part of the lab was to examine the tidal volume (TV), inspiratory reserve volume (IRV), the expiratory reserve volume (ERV) and the vital capacity (VC). The

hypothesis is to observe all the normal ranges for each section. The TV is the volume of air breathed in and out during a single, regular breath and usually averages about 0.5L (500 ml). Our TV was lower than expected at 0.115 L (115 ml), which might have been due to not breathing normally due to stress of the experiment. The IRV is the extra volume of air that can be inspired above the typical resting tidal volume and usually averages about 1500- 3000 ml respectively in female and male. Subject for this part of experiment was a UCD female in her early 20s. Our IRV was lower than expected volume at 0.92 L (920 ml). ERV is the extra volume of air that can be expired beyond the typical resting TV by maximal contraction of the expiratory muscles, abdominals and internal intercostals, and usually 700-1,100 ml respectively in female and male. Our ERV was higher than expected at 1.025L (1025 ml), which might have been due to some anxiety and hyperventilation. The VC is the maximum volume of air that can be breathed in and out during a single breath after a maximum expiration and usually averages about 3100-4,600 ml in female and male. Our VC was slightly lower than expected value at 2.926L (2,926 ml). The static lung volumes are influenced by many factors, including gender, weight, build, health and anxiety, athletes, which might play a role in our values being different from the expected values.

A study on athletes and nonathletic showed that "athletes may require additional of 10% (0.6/6 mL/kg) for males and 8.3% (0.5/6 mL/kg) for females during general anesthesia and critical care." (Baltopoulos 2013).Our object was a normal UCD student so she had a lower tidal volume. Our body and organs can change to a better shape as we use them more. Another study showed that men who remained active had higher forced expiratory volume in one second (FEV_1) and forced vital capacity (FVC) than the other groups (Y J Cheng 2003). Out of the 500ml that enter the lungs about 150ml remain in the dead space, which are parts of the respiratory tract that are not involved in gas exchange. Alveolar ventilation is the volume of air

that is exchanged between the atmosphere and alveoli per minute, and is calculated using the formula V_E= TV x RR and V_A= V_E – V_{DS}. For our results we used the weight of the subject (130lb) to estimate the dead space volume (130 ml), our tidal volume was 0.115L, and the respiratory rate was 13 breaths/min, giving us an alveolar ventilation of about 0.195 L/min. Decrease in ventilation leads to an increase in arterial P_{CO2}. Carbon dioxide will start to build up throughout the body causing a respiratory acidosis.

The rhythmic pattern of breathing is controlled by the respiratory control centers in the brain. The respiratory muscles are skeletal muscles. Muscles have to be stimulated by nerves to initiate contraction. There are involuntary control of rhythmic breathing and voluntary control to accomplish breath-holding, speaking, playing wind instruments, etc. (Sherwood, 2007). The medulla oblongata controls breathing by responding to changes in PO_2, PCO_2, and pH levels. The medullary respiratory center is made up of the dorsal respiratory group and the ventral respiratory group. The dorsal respiratory group processes information from the chemoreceptors and lungs in order to modulate inspiration. The inspiratory neurons from the DRG have its fibers terminate on the motor neurons that innervate the inspiratory muscles. The ventral respiratory group remains inactive during normal, quiet breathing. It takes information from the dorsal respiratory group and regulates inspiration and expiration after sensing changes in arterial gases, which occurs during periods of increased ventilation demand. The VRG is important in active expiration, which is needed in our exercise to obtain the expiratory reserve volume (ERV). Pulmonary stretch receptors in the airways, when stretched, cause the inspiratory neurons to decrease firing which prevents overinflation of the lungs (Hering-Breur reflex). A study showed that slowing and deepening of respirations, maybe caused by interrupting the Hering-Breuer reflex and not expansion and collapse of the lung or existence of a normal pulmonary circulation

in the vagotomized lung or Normal fluctuations in alveolar carbon dioxide tension (Moore 1927). Pre-Botzinger complex is the synaptic input that controls the rate of DRG inspiratory neurons (Sherwood, 2007). In addition to the medullary respiratory control center there are two other respiratory centers located in the pons, the apneustic center and the pneumotaxic center. These centers exert "fine-tuning" influences on the medullary respiratory centers. The apneustic center stimulates the inspiratory neurons in the DRG, thus promoting inspiration. The pneumotaxic center controls the duration of inspiration by sending impulses that shut off the inspiratory neurons in the DRG, thus stopping inspiration (Sherwood, 2007).

Respiration occurs when synaptic input from the pre-Botzinger complex causes the DRG inspiratory neurons to fire an action potential, which cause the inspiratory muscles (diaphragm and external intercostal muscles) to contract. The contraction of the muscles increases the thoracic cavity, which forces the lungs to expand; decreasing the intrapulmonary pressure within the lungs causes the volume to increases. The air, rich in O_2, flows from an area of high pressure (atmosphere) to an area of low pressure (lungs). The pneumotaxic center sends impulses that shut off the inspiratory neurons of the DRG and thus cause the inspiratory muscles to relax, which causes a decrease in the volume of the thoracic cavity and thus a corresponding increase in the intrapulmonary pressure. The air, now rich in CO_2 and low in O_2, flows from an area of high pressure (lungs) to an area of low pressure (atmosphere). Firing of the VRG inspiratory neurons and expiratory neurons is under voluntary control, and is responsible for us being able to inhale and exhale beyond the regular tidal volume limits (Sherwood, 2007). The purpose of this experiment was not achieved since our data either were higher or lower than normal ranges due to experimental errors such as not using the BIOPAC system properly or subject being tired from finishing another part of the experiment so she could be out of breath or not breathed normally.

Part 2: Effects of inspired gas composition and lung volume on Respiration.

In the first part of the second exercise we examined how long the subject can hold their breath after normal breathing, re-breathing and hyperventilation, and what the CO_2 content is before and after the breath-hold. We expected the breath-hold to be longest for hyperventilation, because the subject held their breath after taking a deeper breath than they would have taken during normal breathing. The physiology behind this is that in normal conditions a decrease in PCO_2 in hyperventilation will decrease the inspiratory drive and causes the object to be able to hold his/her breathe for a longer period of time. Increase in ventilation by an increase in respiratory rate and/or increasing tidal volume leading to a decrease in PCO_2 (hypocapnia). Rate of ventilation is higher than what is needed to remove carbon dioxide from blood. A decrease in PCO_2 will decrease the inspiratory drive so the subject can hold breaths for a longer period of time. Subject's CO_2 content before breath-hold was lowest at 2.91% for hyperventilation meaning that the lungs can hold more CO_2 before the maximum CO_2 capacity is reached and the person has to exhale. Normal breathing would be the second longest breast hold, because the breath is held after a normal inhalation and the CO_2 content before the breath hold is slightly higher at 4.46%, meaning that it will take less time for the maximum CO_2 capacity of the lungs to be reached. The shortest time of breath hold would be the re-breathing, in which the subject is breathing back the CO_2 the exhale into the bag. The CO_2 content before breath hold is at a high value of 7.13%, meaning it will take them less time to reach the maximum CO_2 capacity of the lungs then during normal breathing and during hyperventilation. Our results for normal breathing and hyperventilation did not match the expected results. In our results the time of breath hold was longest for normal breathing at 31 seconds, the second longest for hyperventilation at 29 seconds. These differences might have been caused by the subject not holding their breath to the

same level of discomfort, or not having enough time to recover in between the different steps of the exercise. The results for re-breathing matched what was expected, with the time of breath-hold being the shortest at 4 seconds. The goal of ventilation is to provide oxygen to the blood and to constantly remove carbon dioxide from the blood. The blood entering the lungs is low in partial pressure of O_2 and high in partial pressure of CO_2. After an inspiration the alveoli have a higher PO_2 and a lower PCO_2, which causes O_2 and CO_2 to diffuse down their partial pressure gradients. O_2 goes from the alveoli to the blood, and CO2 goes from the blood to the alveoli to be exhaled out (Sherwood, 2007).This is the CO_2 content that we measure before and after breath-hold. In part one of our exercise we measured the content of the CO_2 in the exhaled breath before and after breath-hold.

Based on what it was said above, we expect a person to hold their breath the longest when the subject starts with a high alveolar PO_2 and a low PCO_2 because there is more O_2 available for exchange with blood and more CO_2 can be transferred to the alveoli. So we would expect the time of breath-hold to be longest for a forced inhalation, where the CO_2 has been eliminated during expiration and there is a larger supply of O_2 being taken in. The breath-hold should be the shortest for forced exhalation, where, even though more CO_2 has been eliminated during the active expiration, there is no fresh O_2 to get to the tissues, causing to the desire to inhale. The time of breath-hold after a normal inspiration and a normal expiration should be between the times for a forced inhalation and a forced exhalation. The results gotten in part 2 of this exercise matched the expected values, with the time of breath-hold being the longest for forced inhalation at 1 min and 08 seconds, second longest for normal inspiration at 49.60 seconds, only slightly longer than normal expiration at 48.90 seconds, and the shortest for forced exhalation at 32.81 seconds.

Part 3: Exercise Hyperpnea

In the final part of this experiment, the effects of exercise workload on ventilation were examined and then displayed in table 5 and graphs 4-8. Graphs 4-8 showed an increase in their ventilatory responses as exercise workload increased. There were only slightly decrease in RR and V_E from 0.0 kPa to 0.5kPa. Subject may slowed down for a second as the workload changed and that resulted in the values we observed. Graph 4 displays that TV increased as the workload increased to provide body demands that required an increase in oxygen uptake. Figure 5-8 shows an increase in RR, $V_{E,}$ FE_{CO2}% and minute $CO_{2.}$ In all of them there was a decrease after workload was quickly dropped back to 0.0 kPa. During heavy exercise the metabolic rate of muscle increases, resulting in increase demand of O_2 and a buildup of CO_2 and H+ as well as the alveolar ventilation (Sherwood, 2007).The rate of ventilation changes in the response of PO_2, PCO_2, and the acidity of the blood. Changes in these three chemical factors are detected by the peripheral chemoreceptors, and central chemoreceptors, which send the information to the medullary respiratory center. The medullary respiratory center responds to the changes in PO_2, PCO_2 and H+ in the blood, by sending the appropriate stimuli to the motor neurons to adjust the respiratory rate (RR), tidal volume (TV) and rate of ventilation (Sherwood, 2007). Our results showed such a pattern. As workload increased TV and RR increased, and the purpose of this part was observed. TV increased from 0.309L to 1.39L from workload of 0.0 kPa to 2.0kPa. RR increased from 10 bpm to 15 bpm. The only difference in our results was that there was a decrease in RR from 16L to 12 L from workload 0.0 kPa to 0.5 kPa. This decrease obviously makes a change in V_E as well since V_E=TV*RR when RR decreases $V_{E.}$ The decrease in V_E was from 10.83L to 9.54L from workload 0.0 kPa to 0.5 kPa. The increase in TV and RR results in an increase in the minute ventilation. V_E increased from 3.09 L/min to 20.85 L/min from workload of 0.0 kPa to 2.0kPa.We would also expect an increase in the end-tidal CO_2% (FE CO_2%).

Increase in exercise, increases the metabolism of the muscle cells, resulting in using more O_2 and producing more CO2 in the muscle and the CO_2 and waste has to eliminate from body. The results for FE CO_2% proved what is expected and hypothesis was achieved in this part. FE CO_2%increased from 4.476% to 6.66% from workload of 0.0 kPa to 2.0kPa. The increase in FE CO_2% will results in an increase in the minute CO2, which is calculated by V_E * FE CO_2%.