Human Respiratory System Experiment

Name: Zijun Liu

Group members: Xiaodong Shi, Conner Tiffany, Allen G.

Section  03

TA: Ken Eum

Nov 18, 2013
**Introduction:**

The main purpose of breathing is to have gas exchange by supplying fresh oxygen (O$_2$) for blood and constantly removing carbon dioxide (CO$_2$) unloaded from the blood. The simple passive diffusion of O$_2$ and CO$_2$ down partial pressure gradients drives the gas exchange between alveoli and pulmonary capillary blood. As respiration occurs, most of O$_2$ is carried by hemoglobin protein entering blood and CO$_2$ leaves the blood in the lungs passively down partial pressure gradients that indicates a difference in partial pressure between capillary blood and surrounding structures. The diffusion ends when the partial pressure becomes equal across the membrane (Sherwood 2010, Pg. 486).

When air enters body, it firstly travels through the conducting zone of the lung, which includes trachea, bronchi, bronchioles, and terminal bronchioles. Then it travels to the transitional respiratory zone which consists of respiratory bronchiole, alveolar ducts, and alveolar sacs. The volume of air that moves in and out of the lung per minute is the Minute Ventilation (V$_E$). The exchange of oxygen (O$_2$) and carbon dioxide (CO$_2$) only take place in the alveolar sacs where capillaries are found. The amount of air that leaves the alveolar per minute is called the Alveolar Ventilation (V$_A$). Since there is no alveolus in the conducting zone, no gas exchange occurs. Because the conducting zone is also called the dead space, the volume of air in the dead space is labeled as V$_{DS}$ (Dead Space Ventilation). Air flows into the lung due to pressure gradient. When pressure of atmosphere is greater than the pressure inside the lung, air goes from atmosphere into the lung knowing as inspiration, and air goes an opposite direction due to the greater pressure in the lungs knowing as expiration. When subject takes a normal breath, the volume of air inhaled and exhaled is the resting tidal volume (TV). (Sherwood 2010, pg. 479) For males, TV is about 500 ml. When a person
forces an inhalation, extra air let into the lung is the inspiratory reserve volume (IRV). The maximum amount of air inhaled is the inspiratory capacity (IC). For forced exhalation, the extra volume of air expelled is the expiratory reserve volume (ERV). The total amount of air one can inhale and exhale is the vital capacity (VC). Body reserves some air to prevent the lung from collapsing. The reserved air is the residual volume (RV). With each breath, CO₂ is produced as a result of cellular metabolism. The percent of exhaled air that is CO₂ is represented by fraction of expired CO₂ (EF CO₂).

During inspiration, air moves from the environment into the alveoli through airways, and the air is moved from alveoli into external environment during expiration. For the airflow, it is like blood flow, the air moves by bulk flow—from a region of higher pressure to one of lower pressure. And the relevant pressures are alveolar pressure (P_{alv}) which is inside the lung and atmospheric pressure (P_{atm}). During inspiration, the diaphragm and external intercostals muscles are used. Diaphragm is the primary muscle for inspiration and it is innervated by phrenic nerve. As the inspiration occurs, the diaphragm moves downward. When external intercostals muscle is innervated, external intercostals muscles contract and expand rib cage up and outward. As a result, the intrapleural pressure (P_{ip}) that is the pressure between the interface of lung and chest wall decreases, and the transpulmonary pressure (P_{tp}) that is the result of P_{alv} – P_{ip} increases. As the P_{tp} is larger than the elastic recoil exerted by the lungs, lungs or alveoli are forced to expand. Then the P_{alv} decreases and is below P_{atm} causing the inhalation of the air from atmosphere to alveoli. At the end of inspiration, the P_{alv} increases and exceeds the P_{atm} leading to the air flows out of the lung, and the expiration occurs. It occurs passively during normal breath with the decreased firing of phrenic nerves causing the
diaphragm and intercostals muscles to relax. Thus, the chest wall starts to recoil to move inward causing the $P_{ip}$ increases and $P_{tp}$ decreases. Ventilation is the result of changes in pressure that are caused by the change of lung dimensions.

Control of ventilation is manipulated by one stretch receptor and two chemoreceptors: central chemoreceptor and peripheral chemoreceptor. Peripheral chemoreceptor is located in the aortic arch and carotid sinus. It senses the decreased partial pressure of O2 and increased proton concentration. Central chemoreceptor as the main regulator of respiratory drive locating in the medulla senses increased partial pressure of $CO_2$ by detecting the increased proton concentration. (Sherwood 2010, pg. 503) $CO_2$ diffuses across capillary blood and react with water to form bicarbonate and hydrogen ion, which decrease the PH value in blood.

In this lab, the purpose is to test human static lung volumes, CO2 percentage before and after breath-hold on normal breathing, re-breathing, hyperventilation, the duration of breath hold under these three conditions, and the effects of lung volume on duration of breath-hold. These tests were completed by a healthy 24 year old female subject. And also, the effects of exercise on ventilation were tested by a healthy 23 year old male subject.

The hypotheses for static lung volume is that the sum of IRV, ERV, and TV is VC, also $V_E$ is the sum of $V_{DS}$ and $V_A$. For the examination of the effect of gas composition and breath-hold, before breath hold, the CO2 percentage in hyperventilation should be the smallest one, and the re-breathing should be the largest one. After breath-hold, CO2 percentage in all of these three breath types should be the same level, and hyperventilation should have the longer breath-hold duration; and re-breathing should have the shorter
duration. And the forced inspiration should have the largest breath-hold duration, and forced expiration should have the smallest one. The hypotheses for exercise experiment should be that the ventilation increases as the workload increases.

**Method:**

Subject of this experiment were a healthy 24 year old female and a healthy 23 year old male. The lab details about methods and materials can be found in Experiment 6 on page 57-62 in lab manual NPB 101 Systemic Physiological Lab Manual (Bautista and Korber 2008, Pg 57-62). Interpretations and analysis of data were done by using excel and Biopac System’s I bar, BPM, Max, and delta function.

**Results:**

As subject breathed normally, the data are observed that the tidal volume was 0.46 L (TV). The inspiratory reserve volume is 1.10 L (IRV). The expiratory reserve volume is 1.91 L (ERV). ERV is 0.81 L which is 73.6% higher than IRV. When subject inhaled and exhaled maximally, both IRV and ERV are observed more than twice bigger than tidal volume that is under normal breath. Vital capacity (VC) is 3.43 L, which is roughly the sum of IRV, ERV, and TV. The sum volume of these three conditions is observed as 3.47 L that is only 0.04 L or 1.17% different with measured VC. Minute ventilation (V_E) is the product of TV and respiratory rate (RR), 7.36 L/Min. It is 2.08 L/Min bigger than alveolar ventilation (V_A) that is 5.28 L/Min. And the ventilation difference of 2.08 L/min between V_A and V_E indicates the dead pace ventilation (V_DS). (Table 1)
Before breath-hold, the exhaled percentage of CO₂ is observed that it is dropped from 6.57% of re-breathing to 3.44% of hyperventilation. Under re-breathing, the CO₂ percentage of 6.57% is almost 1% larger than control group which is normal breathing condition, and almost twice of that under hyperventilation condition which is 3.44%.

After breath-hold, there is an observed dropping from re-breathing to hyperventilation too, which is from 7.1% to 6.11%. But there are only 0.4-0.6% different compared to control group. To compare individually, after breath-hold, the re-breathing condition CO₂ exhaled is increased to 7.1% from 6.57% which is before breath hold. It is about 0.5% difference. And hyperventilation after breath-hold is increased to 6.11% from 3.44% that is before breath-hold. It is almost one time difference. Generally, all of the exhaled CO₂ contents under three breathing types after breath hold are higher than that of before breath hold. (Figure 1)

<table>
<thead>
<tr>
<th></th>
<th>Volume</th>
</tr>
</thead>
<tbody>
<tr>
<td>IRV</td>
<td>1.10 L</td>
</tr>
<tr>
<td>ERV</td>
<td>1.91 L</td>
</tr>
<tr>
<td>TV</td>
<td>0.46 L +/-0.05</td>
</tr>
<tr>
<td>VC</td>
<td>3.43 L</td>
</tr>
<tr>
<td>VE</td>
<td>7.36 L/Min</td>
</tr>
<tr>
<td>VDS</td>
<td>2.08 L/Min</td>
</tr>
<tr>
<td>VA</td>
<td>5.28 L/Min</td>
</tr>
</tbody>
</table>

Table 1. Static lung volumes of human subject collected under twelve normal inhale-exhale cycles, one deep inhale, and one exhale. Subject breathe normally for one minute before starting the experiment.
Figure 1. Comparison of percent of CO₂ in healthy human subject before and after breath-hold under conditions: re-breathing, normal breathing, and hyperventilation. Subject’s nose was clipped and breathed through mouth. Only the last 20-50% of exhalant volume was collected and examined.

Re-breathing hold duration is observed as the shortest one which is 33.2 second. It is almost a half of control group which is normal breathing hold duration, 60.54 seconds. Hyperventilation duration which is 56.75 seconds is similar with control group, but it is still have 4 seconds shorter than the normal breathing. (Figure 2)
Figure 2. Comparison of a healthy human subject’s breath-hold duration under three ventilation type: normal, re-breathing, and hyperventilation. Subject inhaled through mouth before holding her breath.

Among the four duration times under different conditions, the forced inhalation is observed as the longest one which is 84.43 seconds, and followed by normal inspiration duration, 46.31 seconds, which is more than 40 seconds drop than that of forced inhalation. The normal expiration, 35.85 seconds, is observed slightly around 10 seconds drop compared with that of normal inspiration, but almost 1 time higher than that of the forced exhalation, which is 16.11 seconds. (Figure 3)

Figure 3. Effects of healthy human subject’s lung volume on duration of breath hold. The four lung volumes were created using: normal expiration, normal inspiration, forced inhalation, and forced exhalation. Subject breathed through mouth, and nose was clipped.

As observed, all of these indexes have an increase generally from rest condition to 2 KPa during exercise. TV increases from 1.14 L/breath to 2.25 L/breath, which is a 97.4% increasing. And although there is a slight drop at 0.5 kPa which is 14 breathes/min, RR still increases from 16 breathes/min to 33 breathes/min indicating an increase of over one time during the whole process. As a result, the ventilation has a
significant increase from 18.24L/min to 74.25L/min. It is 3 times higher at the 2 kPa workload than that at rest condition. The fraction of expired CO₂ (FE CO₂) increased constantly from 4.92% to 6.67%. There is a steady increase of about 0.2%-0.3% every 0.5 workload. And at 2.0 kPa workload, the FE CO₂ is 1.75% higher than that at rest. As a result, with the increased VE and FE CO₂, the minute CO₂ increases. It increased from 0.90 to 4.95 L/min constantly, and indicating that at 2.0 kPa, the exhaled CO₂ amount per minute is about 4.5 times higher than that at rest. (Figure 4)

Figure 4. Relationships between workload and TV, RR, VE, FECO₂, and minute CO₂ for the male subject are plotted. The male subject sat quietly on exercise bike for 2 minutes and started biking from 0 kilopond (KPa) workload. Workload increased by 0.5 KPa at every 2 minutes until 2.0 KPa. Subject’s nose was clipped and breathed through mouth.

Discussion:

The respiratory centers in the brain stem establish a rhythmic breathing pattern. The rhythmic pattern of breathing including inspiratory and expiratory is established and
controlled by medullary respiratory center via cyclic neural activity to the respiratory muscles (Sherwood 2010, Pg. 498). During rhythmically breath in and out at rest, the alternate contraction and relaxation of diaphragm and external intercostals muscle occurs with the supplying of phrenic nerve and intercostals nerves respectively. Impulses originating in the medullary center end on the motor-neuron cell bodies which compose the phrenic nerve and intercostals nerves. When the motor neurons are activated, the inspiratory muscles are stimulated, leading to the inspiration, and when these neurons are not firing, the inspiratory muscle relax causing the expiration.

The medullary respiratory center is divided into two groups, Dorsal Respiratory Group (DRG) and Ventral Respiratory Group (VRG). The DRG contains mostly of inspiratory neurons which connects with motor neuron and innervate the inspiratory muscle. The inspiration takes place once DRG inspiratory neurons are stimulated, and expiration takes place once DRG inspiratory neuron firing is inhibited. DGR inspiratory neurons rhythmically fire is believed to be driven by the synaptic input from Pre-Botzinger complex which is region locating in the medullary respiratory center. This complex region’s neuron networks act as the pacemaker of breath activity, undergoing self-induced action potentials. Meanwhile, the pneumotaxic center sends the impulses to DRG to help inhibit the inspiratory neurons, and limit the inspiration duration. And apneustic center in contrast to prevent the inspiratory neurons from inhibiting, and drives an extra boost of inspiration. The VRG, contains inspiratory and expiratory neurons, is only activated during forced breathing (Sherwood 2010, Pg. 500). Breathing is modified by peripheral chemoreceptors, central chemoreceptors, and stretch receptors (Hering-Breuer Reflex).
The breathing modification by peripheral and central chemoreceptors work with three chemical factors: O₂ pressure, CO₂ pressure, and H+ concentration to adjust the magnitude of ventilation. Pressure of O₂ (PₐO₂) and CO₂ (PₐCO₂) of the systemic arterial blood leaving the lungs are held constant, indicating that arterial blood-gas content is precisely regulated. To meet the body’s needs for O₂ uptake and CO₂ removal, the arterial blood gases are maintained within the normal range by varying the magnitude of ventilation like rate and depth of breathing (Sherwood 2010, Pg. 501).

To balance the gases in arterial blood, the peripheral chemoreceptor that is located in the carotid bodies and aortic arch detect the decreased PₐO₂ which is also known as hypoxia. Once a PₐO₂ drop of 40-60 mmHg is detected by the peripheral chemoreceptor, as a result, ventilation rate increases to increase the inhaling of O₂. The central chemoreceptor which is the main regulator is located in the medulla and senses the increased PₐCO₂ known as hypercapnia, and the decreased PH indicating acidosis in the cerebral spinal fluid by signaling to the medulla via afferents of glosspharyngeal nerve and vagus nerve for the carotid bodies and aortic bodies respectively. CO₂ produced by the metabolic of organs binds with water in the body and yields carbonic acid (H₂CO₃) which is broken down to H⁺ and bicarbonate (HCO₃⁻). This can be expressed as the chemical equation: CO₂ + H₂O ↔ H⁺ + HCO₃⁻. When PₐCO₂ increases, chemical equation shifts to the right, yielding more H⁺ and thus lowers the PH value. Therefore central chemoreceptor senses increased PₐCO₂ by detecting the drop in PH leading to the increased ventilation so that to increase PₐO₂ and PH, and decrease in PₐCO₂. As a result the excess CO₂ is got rid of the body. And once PₐCO₂ constantly decreases to the central chemoreceptor threshold, the ventilation will be reduced in response to CO₂ (Mohan, Liu, P.11).
Amara, and et al., 1999). Overall, hypoxia, hypercapnia, and acidosis would cause an increase of firing rate of chemoreceptors to increase the ventilation.

Other than chemoreceptors, stretch receptors also manipulate ventilation rate by detecting stretch of smooth muscle in air way. It is known as Hering-Breuer Reflex. When the smooth muscle of air way is stretched at large tidal volumes, action potential from these stretch receptors travel through afferent nerve fibers to the medullary center to inhibit the inspiratory neurons. The inhibited inspiratory neurons help to inhibit breathing to prevent the lung from overinflated (Sherwood 2010, Pg. 500). On the contrary, if air is expired, stretch receptors detect the deflation of lung and send signals to the medulla to allow more air into the lung.

During exercise, ventilation rate is proportional to metabolic activity. As body organ’s metabolic activity rate increases, more CO₂ is released by organs and accumulates in the body leading to the increased demand of O₂. Body tries to remove excess CO₂ by increase in ventilation rate. During exercise, it is more efficient to increase tidal volume (TV) than increasing respiratory rate (RR) to elevate minute ventilation (Vₑ). When deeper breathes are taken (increase in TV), more air is transported to the alveolar for O₂/CO₂ exchanged. If RR increases faster, more energy needs to be used, and more air is moved into the alveolar and dead space, so more air is wasted since there is no gas exchange within dead space.

In part one of lab, the female subject’s static lung volume was tested. Under normal breathing, the subject’s TV was 0.46L (Table 1). TV is the amount of air a person inspired and expired on a normal breath. When the subject inhaled as deeply as possible, he was using forced breathing. (Sherwood, 2010, Pg. 480) Forced breathing is accomplished by maximal
contraction of the diaphragm, and external intercostals muscles. The extra volume of air inspired (IRV) by the subject was 1.10 L (Table 1). This is the amount of air inspired on top of his regular inspiratory volume. For expiration, the subject took an exhale as deeply as possible. The extra amount air (ERV) pushed out by the lung was 1.91 L (Table 1). As a result, both of IRV and ERV are greater than TV due to the extra inspiration and expiration. It was observed that subject’s ERV was 0.81 greater than IRV (Table 1). However, physiologically, EVR should be less than IRV, because the lungs can never empty out all the air. To explain specifically, if all the air inspired into the lung is expired, fluctuation in CO₂ and O₂ in blood will be wide. Moreover, it is more efficient to inflate partially filled alveolus than a totally deflated one. Additionally, the reserved volume can keep the lung from collapsing. The error here might be caused by that the subject didn’t inhale as much as possible. The amount of air inspired and expired per minute by the subject was 7.36 L/min indicating $V_E$ (Table 1). $V_E$ describes how deeply and frequently the subject took her breathes per minute. It can be adjusted by the respiratory rate and tidal volume through exercise. It will be discussed further in experiment part three in discussion.

The total amount of air inspired and expired per minute doesn’t mean the actual amount of air participating in gas exchange. The actual participated air for gas exchange is alveolar ventilation ($V_A$) which is 5.28 L/min (Table 1). It is smaller than $V_E$, because when air enters into the respiratory tract, the air cannot have an exchange at the part of conducting zone. The gas exchange can only take place at alveoli where contain capillaries allowing O₂ and CO₂ to exchange in and out of blood. As a result, there is always a part of air that has no gas exchange being reserved in the conducting zone. This conducting zone is also called dead
space, and the volume of this zone is dead space volume ($V_{DS}$) which is 2.08 L/min for the female subject. The dead space volume value equals to the subject’s actual weight in lb. Thus, the different volume of $V_A$ and $V_E$ is $V_{DS}$, which also can be expressed as the equation: $V_E - V_A = V_{DS}$.

In part two, the effects of inspired gas composition and lung volume were tested, the female subject exhaled the first half of gas in dead space before and after breath hold so that to measure the actual CO2 percentage. It was observed that all CO2 contents after breath hold are higher than that of before breath-hold (Figure 1). It is because body was accumulating CO2 and was not releasing it. Even subject was not breathing, her body was still metabolizing and generating CO2 by organs. The main function of the lung is to get CO2 out of body and get more O2 into the body. When the female subject held her breath, her body was not releasing any of the accumulated CO2 out.

In the re-breathing test, before breath-hold, the CO2 contents were the highest compared to the other testes. To explain this, the subject’s body was not effectively releasing all the expired CO2 because she re-breathed in the CO2 in a volume limited bag. When the subject took her last inhaled from the bag, she inspired all the CO2 accumulated in the bag into her body. Thus at the end of breathing in and out the bag for 3 minutes, the amount of CO2 in her body was highest.

For the hyperventilation test, it was observed that the CO2 content is the lowest that is 3.44% before breath hold (Figure 1), because under deeper and more frequent breathes, the subject’s body was able to release CO2 more efficiently and inhale more O2. As a result the CO2 concentration decreased greatly. It agrees with the trend that before breath-hold, the CO2
content in hyperventilation group is much lower than that of re-breathing group which is 6.57% and moderately lower than the control group of normal breathing which is 5.71%.

To compare the %CO₂ after breath-hold, it was observed that the CO₂ content had a clearly dropping from re-breathing to normal breathing to hyperventilation with the number of 7.10%, 6.56%, and 6.11% respectively (Figure 1.). However, physiologically all of these three breathing tests should have the same amount of CO₂ contents, because the central chemoreceptor in medulla always senses the same CO₂ level to decide the discomfort level for the subsequent exhaling. Thus, after breath-hold, the subject should hold the breath to the same level of discomfort to exhale in all of these three tests. Therefore, to explain the test result error, the subject didn’t hold her breath to the same discomfort level when she did this part of test.

Comparing the duration of breath-hold under the three breathing types, subject held her breath for 57.65 seconds under hyperventilation which is longer than that of 33.2 seconds under re-breathing, but shorter than that of 60.54 seconds under normal breathing. However, physiologically, the hyperventilation breath hold duration should be moderately longer than that of normal breathing and greatly longer than that of re-breathing. Central chemoreceptor in the medulla senses the drop in brain fluid’s PH. CO₂ can cross blood vein barrier, so when the amount of CO₂ increases in our body, more H⁺ is generated. Longest duration of hyperventilation is due to the increased lung volume and decreased PₐCO₂ (higher initiated brain fluid PH). This leads CO₂ to take more time to diffuse between alveolus and blood, and more CO₂ was needed to be accumulated in the body to drop the PH low enough to trigger the central chemoreceptor. As a result, a longer time was needed to hold to accumulate enough
CO₂. For re-breathing, the body started with a high level of CO₂ (6.57%) (Figure 1), thus it took shorter time for the subject to accumulate enough CO₂ or H⁺ to activate central chemoreceptor. And the high level of CO₂ percentage needs a shorter time to have CO₂ diffusion. During breath-hold, CO₂ content is not the only factor to initiate ventilation, O₂ content also plays a role in ventilation. As the higher content of CO₂ during breath-hold, O₂ content runs low, thus the peripheral chemoreceptor is triggered to increase the ventilation rate.

In the test for the effect of lung volume and duration of breath-hold in Part 2, the female subject held her breath for 35.85 seconds after normal expiration. She was able to hold her breath for a longer time of 46.31 seconds after normal inspiration (Figure 3). To explain this, the P_{CO₂} or CO₂ concentration plays a big role. The lungs volume increases during inspiration leading to the decrease in P_{CO₂} in the lungs. Thus CO₂ need to take a longer time to diffuse between the alveoli and blood capillaries to reach CO₂ equilibrium. In a contrast, the lungs volume decreases during expiration leading to an increase in P_{CO₂} in the lungs. As a result, CO₂ needs a shorter time to diffuse between alveoli and capillaries to reach the equilibrium. Therefore the diffusion time of CO₂ before chemoreceptors senses the CO₂ threshold decides the breath-hold duration time. Moreover, Hering-Breuer Reflex also affects the duration time. The Hering-Breuer Reflex is caused by the increased stretch of lungs. When the subject inhaled, the lung volume increased, and increased stretch of her airways triggered Hering-Breuer reflex, which inhibited inspiratory neurons to prevent her lung from overinflating. This decreases the drive for her breath, thus she was able to hold her breath for longer period of time. On contrary, when subject decreased her lung’s stretch or lung volume
by exhaling, her inspiratory neurons were activated to initiate breathing. This result agrees with the study conducted by V. Chan and A. Green in 1992, on how Hering-Breuer reflex function in newborns. Chan and Green found that the Hering-Breuer reflex is initiated via stretch receptors in the lung. Hering-Breuer reflex increases when there is a low compliance (ability to stretch) by inhalation in the lung (Chan and Green, 1992).

For maximum inhalation and exhalation, the phenomenon describe is put to a larger scale. The forced inhalation has longest breath-hold duration with 84.43 seconds, and the forced exhalation has shortest breath-hold duration with 16.11 seconds (Figure 3). Subject took in maximum amount of air, so her lung was stretched to its maximum volume causing a bigger decrease in $P_{CO_2}$ in alveoli, leading to a much longer CO$_2$ diffusion time to reach CO$_2$ equilibrium between alveoli and capillaries before the activation of central chemoreceptor. And also, under maximum stretch, Hering-Breuer reflex was triggered to prevent inhalation. Therefore the subject was able to hold her breath for a longest period time of 84.43 seconds. When subject exhaled to her maximum or decrease her lung volume to minimum, the $P_{CO_2}$ in alveoli has bigger increase leading to a very short time for CO$_2$ to diffuse between alveoli and capillaries to reach equilibrium. As a result, much shorter breath-hold duration takes place.

In part three of the lab, as workload was increased, the pre-Botzinger complex is more active leading to the increase in TV, RR, FECO$_2$ and $V_E$ generally. At rest, the male subject’s TV was 1.14 L per breath, and at workload of 2.0 KPa, the male subject’s TV was at 2.25 L per breath which means the subject was taking deeper breath as the workload increased. As workload increased, subject’s RR value overall increased from 16 breaths per minute to 33 breaths per minute, although there was a slightly decrease of 14 breathes per minute at 0.5
Since the increase in TV and RR, the male subject’s $V_e$ also increased. At 0 KPa, his $V_e$ was 26.69 L per minute, and at 2.0 KPa, his $V_e$ was 74.25 L per minute.

This trend shows that the subject was taking in more air every minute as exercise workload increased. It is more efficient for the body to increase in TV than to increase RR, because rather than wasting air in dead space by increase RR, deeper breath would be more efficient, so after each breath, air reaches the alveolus and participate in gas exchange much better. It is also agrees with the equation: $V_e = TV \times RR$ from which it’s easy to see that the change in TV is easier to have a bigger change overall, but RR change cannot cause a bigger overall change. Amount of CO$_2$ in subject’s breath had minimal change since the beginning of exercise. It was observed that FECO$_2$ at rest was 4.92% and was 6.67% at 2.0 KPa (Figure 4). The fluctuation was only almost 2%. Despite there is a slightly increase in CO$_2$ production during exercise, arterial $P_{CO2}$ does not increase but remains normal or decrease slightly because the extra CO$_2$ is removed as rapidly or even more rapidly than it is produced by the increase in ventilation (Sherwood 2010, Pg. 504). Likely wise, during exercise, despite the increased use of O$_2$, arterial $P_{O2}$ does not decrease but remains normal or may actually increase slightly because the increase in alveolar ventilation keeps pace with or even slightly exceeds the stepped-up rate of O$_2$ consumption. And the H+ concentration also doesn’t increase as expected, because H+ - generating CO$_2$ is held constant (Sherwood 2010, Pg. 54). Therefore, these three chemical factors: decreased $P_{O2}$, increased $P_{CO2}$, and increased H+ cannot well explain the phenomenon of increased ventilation during exercise. However some factors were suggested to play a role in the increased ventilation during exercise, such as the reflexes originating from body movements, increase in body temperature, epinephrine
release, and impulses from the cerebral cortex (Sherwood 2010, Pg. 504). Specifically, during exercise, joint and muscle receptors excited during muscle contraction reflexly stimulate the respiratory center, and the respiratory activity is also coordinated with the increased metabolic requirements of active muscles, as a result the ventilation is abruptly increased. Moreover, during exercise, energy usually is converted to heat causing sweat frequently, but the conversion of energy cannot pace with the increased heat production as increased physical activity, so body temperature usually increases to stimulate the ventilation. Additionally, as exercise workload increases, the circulating epinephrine is released by adrenal medulla more which also stimulates ventilation. Lastly, the motor areas of cerebral cortex can stimulate the medullary respiratory neurons and activate the motor neurons of exercising muscles. As a result, the motor region of the brain calls forth increased ventilation (Sherwood 2010, Pg.504).

This also agrees with the statement by David J. Paterson in “Defining the Neuro-Circuitry of Exercise Hyperpnoea”, where he mentions that central command in the brain control heart rate, arterial blood pressure and ventilation during exercise. Central command does so by sensing tendon vibration on the triceps or biceps muscle during exercise (Paterson, 2013).

It was also observed that subject’s minute CO$_2$ increased from about 0.90 L per minute to about 4.95 L per minute during the whole exercise process from rest condition to 2.0 KPa workload. According to Stato K et al., in their study of the influence of central command on exercising women’s cerebral blood flow, they found that central command along with mechanoreflex in the body is responsible for variation in the respiratory patterns (Stato K et al., 2009). The body does not choose to increase the concentration of CO$_2$ in each breath but increases the ventilation rate. This phenomenon is also caused by central command in the
brain. Overall, when subject is at a rest condition, the control of respiration stays steadily, but in exercise condition, almost all of the factors would have a bigger activation or increase to mainly increase the ventilation.

Generally speaking, two chemical receptors with three chemo factors: \( P_{CO2} \), \( P_{O2} \), and \( H^+ \), and one stretch receptor known as Hering-Breuer Reflex control and modify the respiratory system. Medulla is the primary control center with the central chemoreceptor sensing the increased \( P_{CO2} \) by detecting \( H^+ \) concentration. As the increase of \( P_{CO2} \), \( H^+ \), and the decrease of \( P_{O2} \), the chemoreceptors send the signal to promote the inspiration. And at the same time, Hering-Breuer Reflex also work to control the respiration as the lungs expand by inhalation to inhibit the continually inspiration aiming at preventing overinflating of lungs. And due to the central command by brain, the ventilation increase as the exercise workload increases.
Calculation

Average = (n1 + n2 + n3)/3
= (1.26 + 1.00 + 1.16)/3
= 1.14 L/breath

STDEV = \sqrt{\frac{\sum(X-\text{averageX})^2}{n-1}}
= \sqrt{\frac{(1.26-1.14)^2 + (1.00-1.14)^2 + (1.16-1.14)^2}{3-1}}
= 0.0757

V_E = TV * RR
= 1.14 L/breath * 16 breathes/min
= 18.24 L/min

Minute CO2 = V_E * FE\text{CO}_2
= 18.24 L/min * 0.0492
= 0.090 L/min

1 lb body weight = 1 ml V_DS
V_DS = DS * RR
= 132 ml * 16
= 2112 ml
= 2.11 L

V_A = V_E - V_DS
= 18.24 L/min – 2.11 L
= 16.13 L/min
Reference


