

Lab 6: Spirometry and Respiratory Regulation

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Introduction

The purpose of this laboratory exercise was to examine the various mechanisms of respiration, how the various volumes and phenomena can be measured, and how the respiratory system responds to changes via various stimuli. In particular we sought to investigate the control of the respiratory system and the response of the respiratory system control to changes in base metabolism rate, oxygen in the air, carbon dioxide in the air, and dead space.

The respiratory system is principally controlled by involuntary autonomic systems in the nervous system. These systems are driven by receptors that sense the chemical balance in the blood and cerebro spinal fluid with respect to oxygen concentration, carbon dioxide concentration, and pH, as well as mechanical receptors that sense the distension of the chest during respiration. The breathing cycle is mainly determined by inspiration rate. Expiration signals are constantly sent and periodic inspiration signals override the expiration stimulus. Inspiratory signals are unregulated by a decrease in oxygen concentration in the blood, and an increase in carbon dioxide concentration in the blood and cerebro spinal fluid (which corresponds to a decrease in pH). These chemical changes are sensed by chemical sensing cells in the aortic arch and carotid arteries. By contrast, deep or rapid inspirations result in stimuli from stretch receptors that would inhibit inspiration. Because there are many signals that must be integrated to regulate autonomic breathing this regulation must be controlled centrally at the brain stem, specifically the medulla oblongata. There is of course manual override of breathing control, but the scope of this manual control is limited. Of the stimuli that affect the rate of respiration, the concentration of carbon dioxide in the blood is the strongest effector, even more so than oxygen concentration. While some of these biochemical regulation methods are not fully understood, it is known that carbon dioxide is directly related to the pH buffering of the blood, as carbon dioxide dissolves into carbonic acid into the blood which is essential to the blood pH buffering system. Additionally the cerebro spinal fluid is poorly buffered and thus its pH can be affected greatly by changes in carbon dioxide concentration.

Metrics of breathing usually involve measuring several different respiratory volumes. Such volumes like tidal volume, which measures the volume of air moved during rest breathing, as well as expiratory reserve (volume of breath that can be expired after a rest expiration), inspiratory reserve (volume of air that can be inspired after a rest inspiration), vital capacity (volume that can be expired after a maximum inspiration), residual volume (volume of air that cannot be expired), total lung capacity, and dead space (amount of air that does not move during respiration), are all used to diagnose various respiratory conditions. During this laboratory exercise we investigated all of these volumes as well as changes to respiration by various stimuli.

Methods

Spirometry

The spirometer used was calibrated using a known volume of air pressed into the device. Several values of known volume were used and the deflection in mV seen on the recording equipment was recorded. These volumes and changes in voltage were used to determine a conversion factor from mV to mL of air moved. The subject was then instructed to breathe at rest into the device, followed by a maximal inspiration, a maximal forced expiration, and again normal breaths. With these breathing patterns we were able to calculate all the necessary lung volumes.

Respiratory Rate

We then examined respiratory rate under the conditions of rest, mild exercise, and increased dead space. Heart rate and starting SPO_2 were measured followed by a minute of breathing rate recording. This was done at rest, after a brief run around the building, and again at rest but the subject was breathing through three feet of tubing. The three feet of tubing add dead space to the subject's respiratory system.

Breath Holding

The subject was then asked to hold breath as long as possible at rest, after hyperventilation, and after exercise.

Physical Models

We then investigated a couple of physical models of alveolar filling. We investigated filling one balloon of the alveolar model more than the other then letting their pressures interact. We then used a lung model to observe the filling of the lungs under different pressure scenarios.

Re-Breathing

We then allowed the subject to re-breathe air under two conditions, CO_2 scrubbing and O_2 rich. Under CO_2 conditions the air that the subject was re-breathing was had the CO_2 removed constantly while the O_2 was allowed to be metabolized normally. In the O_2 trial an excess of O_2 was sent into the system and the CO_2 was allowed to accumulate naturally. During each trial the subject rebreathed the air while breath rate, inspired and expired CO_2 and O_2 , heart rate, and SPO_2 were measured. The trial was terminated if the patient became too uncomfortable, or if SPO_2 fell too low or CO_2 concentrations rose too high.

Results

Spirometry

The spirometry results are summarized with an example plot in Figure 1 and the measurements in Table 1. When compared to the measurements taken by the dedicated FEV_1 device we

find agreement with our measurements. The device showed a FEV_1 of 3.54 Liters (3540 mL) which agrees with our measured volume of 3.095 L. when compared to documented volumes of Wikipedia we find some agreement. It is stated in the Wikipedia article we were instructed to compare against that the volumes are typically larger in taller persons, at higher altitude, and when the subject is fit. In our case our subject is over 6 ft tall, lives at moderately high elevation, and is fit. This explains why the volumes we record are larger than expected. The only exception is inspiratory capacity which is reported at 3.5 Liters, which is close to our measured value of 3.639 Liters.

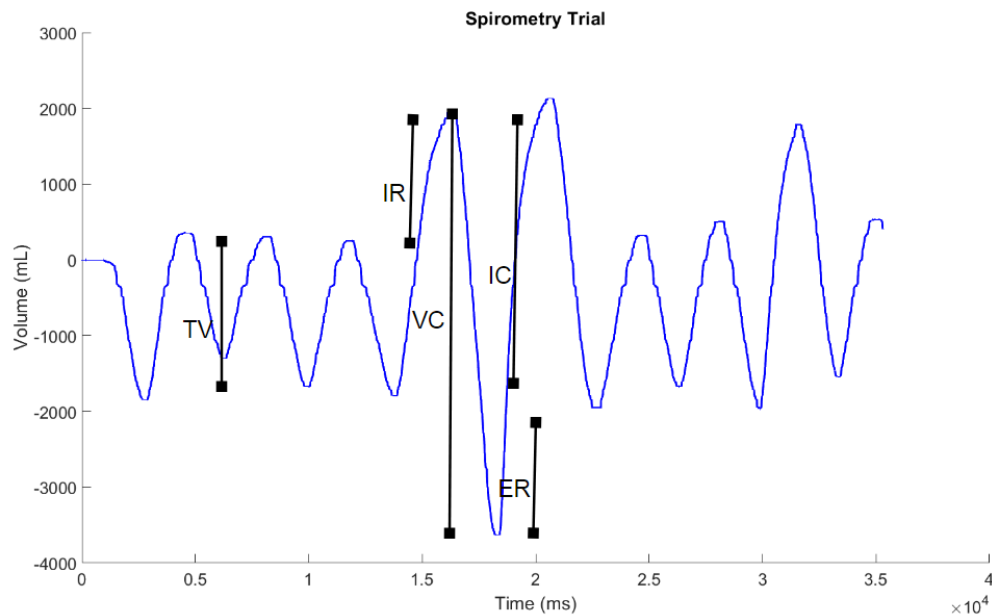


Figure 1: Sample of breath volume measurement trial. Shown are measures of tidal volume (TV), inspiratory reserve (IR), vital capacity (VC), inspiratory capacity (IC), and expiratory reserve (ER).

Table 1: Breaths per minute under exercise, rest, and added dead space conditions.

	Volume (mL)	STD (mL)
TV	1965	216
IR	1673	259
ER	1976	331
IC	3639	125
VC	5615	264
FEV_1	3095	258

Respiratory Rate

The numerical results for the breaths per minute trials are shown in Table 2. The two rest periods did not result in different numbers and are displayed together. During the extra dead space trial where the subject was breathing through the tube an increased depth of breathing was noted. The breathing seemed more labored and was much deeper than at rest.

Table 2: Breaths per minute under exercise, rest, and added dead space conditions.

	Breath Per Minute	Start HR	Start SPO ₂
Rest	14	84	96
Exercise	17	151	98
Extra Dead Space	7	101	98

Breath Holding

The numerical results from the breath hold trial are shown in Table 3. As can be seen the subject struggled to hold their breath after exercise.

Table 3: Breath hold measurements. Time given in seconds. Start HR and SPO₂ taken right before breath hold initiated. End SPO₂ and HR taken when breath hold terminated. During exercise trial the heart rate was too high to measure well on the pulse oximeter.

	Time (s)	Start HR	Start SPO ₂	End HR	End SPO ₂
Rest	75	101	98	88	96
Hyperventilate	105	101	98	91	98
Exercise	8	too high	69	too high	94

Physical Models

When using the alveolar two balloon model we found that inflating one balloon more than the other then allowing the pressures to interact resulted in the less inflated balloon becoming even less inflated and the more inflated balloon becoming more inflated. With the lung model we found that when we pulled on the 'diaphragm' and released the valve the balloons inflated.

Re-Breathing

The rebreathing of carbondioxide scrubbed vs oxygen rich air are shown in the following figures. Figure 2 shows the parameter measurements from CO₂ scrub rebreathing while Figure 3 shows the instantaneous tidal volume during this trial. As can be seen the tidal volume did not change much. The subject did not report any distress during this trial despite the continuous decrease in SPO₂ and O₂ concentration. By contrast during the oxygen rich trial the subject reported difficulty breathing. These results are shown in Figure 4 and Figure 5. Here we see an increase in instantaneous tidal volume as the CO₂ concentration increases as well as a slight increase in HR. All this despite no change in SPO₂. In both trials the breaths per minute tracking was occasionally faulty and thus is missing some time instants.

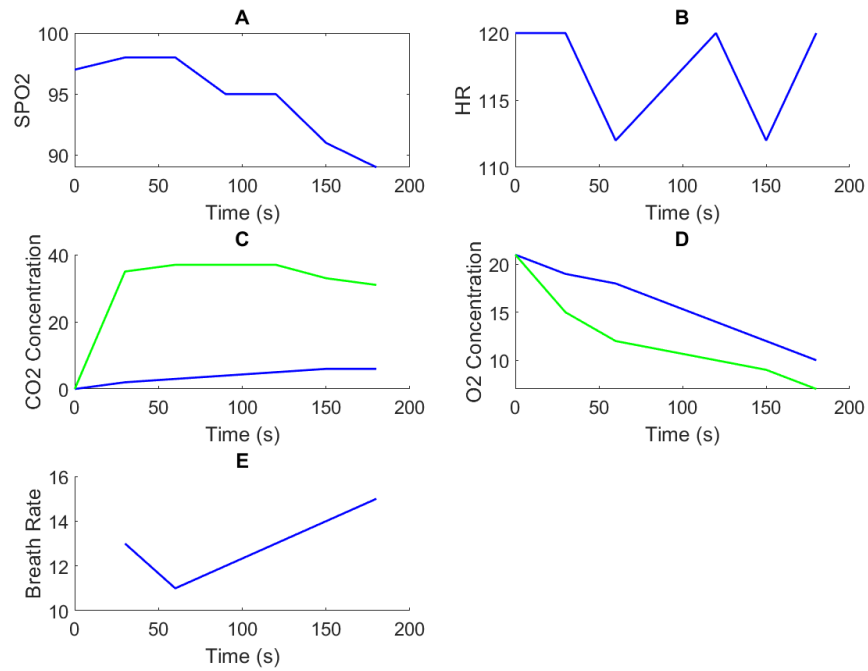


Figure 2: Measurements from rebreathing room air with CO₂ scrub. SPO₂ over time (a), heart rate (B), inspired CO₂ (C, blue), expired CO₂ (C, green), inspired O₂ (D, blue), expired O₂ (D, green), breathing rate (E).

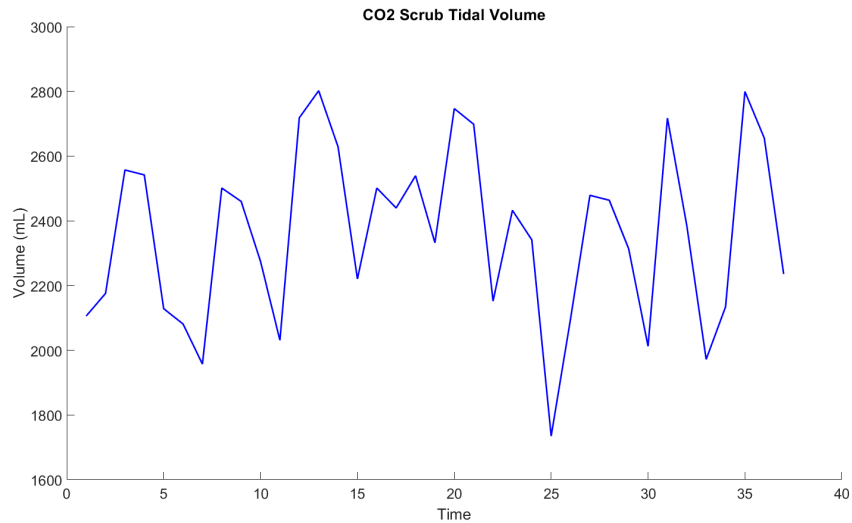


Figure 3: Tidal volume over time during the room air with CO₂ scrub rebreathing. Time instants represent one breathing cycle.

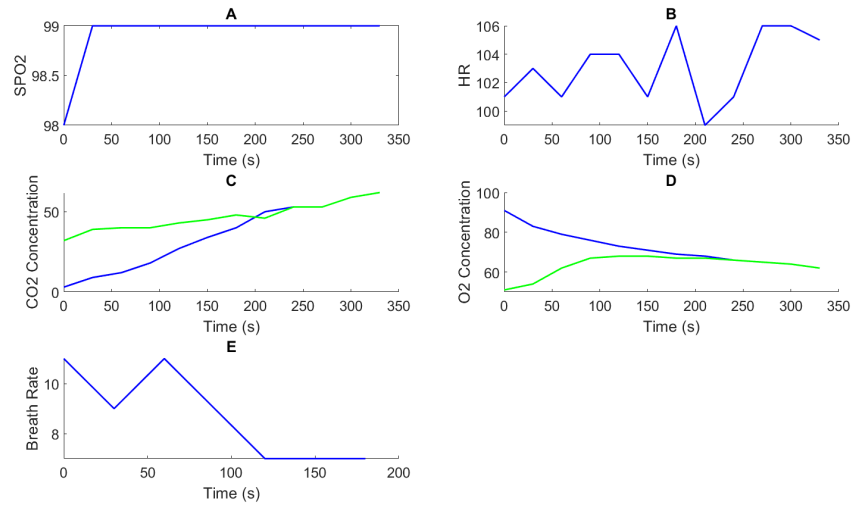


Figure 4: Measurements from rebreathing high oxygen concentration air. SPO₂ over time (a), heart rate (B), inspired CO₂ (C, blue), expired CO₂ (C, green), inspired O₂ (D, blue), expired O₂ (D, green), breathing rate (E).

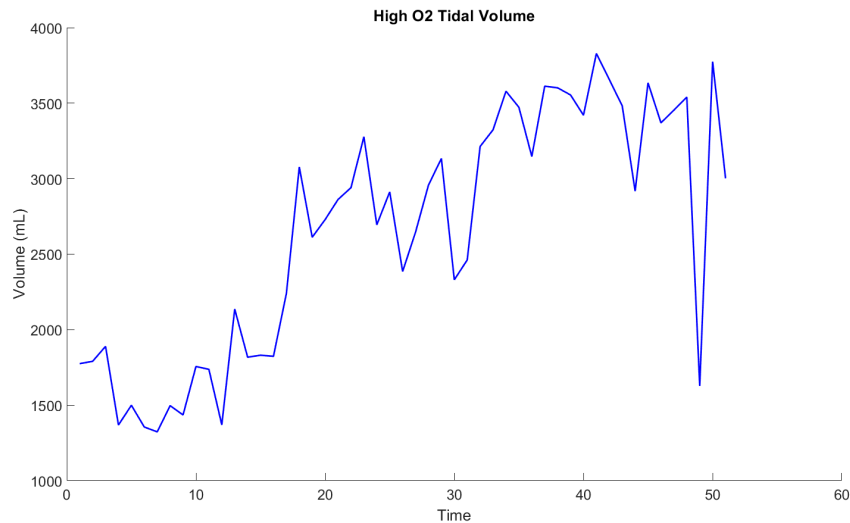


Figure 5: Tidal volume over time during the high oxygen concentration rebreathing. Time instants represent one breathing cycle.

Discussion

Spirometry

During this section we measured the specific lung volumes of the subject (Table 1 and Figure 1). We found that these volumes generally tended to be larger than expected reported volumes, however given that the subject is over 6ft tall and lives at high altitude this makes sense. Larger

bodies result in larger lungs, and at higher altitude a larger volume of air flow is necessary to maintain proper blood gas exchange. During the measure of FEV₁ we found that both the direct spirometry measure and the dedicated device measure produced similar results (3.095 L and 3.54 L respectively). Measuring FEV₁ is important for understanding the health and functioning of the lungs. A lowered FEV₁ indicates pulmonary obstructions or other diseases that affect the lung's ability to move air.

Respiratory Rate

During this trial the subject was allowed to breathe freely and the rate of breathing as well as any other qualitative measures were made during rest, post exercise, and with additional dead space added. At rest the subject showed a basal breaths per minute of 14 (Table 2) This jumped to 17 post exercise with an elevated heart rate of 151 beats per minute. To add extra dead space to the system the subject was instructed to breathe through a 3 ft length of tubing. It was important that this tubing be exposed to open air to not confound these results with the processes present when the subject rebreathes room air. The subject needed to be breathing, not rebreathing, so that CO₂ concentrations would not increase and O₂ concentrations would not decrease. These factors would likely confound our analysis of the additional dead space. While it is true that using the spirometer also adds dead space due to the tubing, these other confounding factors lead us to use a simple open ended tube. During this trial the breaths per minute dropped dramatically to 7 while the breaths themselves became much deeper and labored. The subject reported difficulty breathing. This is likely due to the extra effort needed to breathe with extra dead space. The increase volume of breaths was also due to the increased dead space, and these increased volumes of breaths resulted in decreased breathing rate due to negative feedback by stretch receptors in the chest.

Breath Holding

During this trial the subject was instructed to hold their breath as long as possible at rest, after exercise, and after hyperventilating. At rest this time was 75 seconds with a basal heart rate of 101 beats per minute, and SPO₂ of 98. Post breath hold the heart rate had dropped to 88 beats per minute while the SPO₂ did not change much (Table 3). The reduction in heart rate is directly related to the reduction in respiration. After hyperventilation the subject was able to hold their breath much longer, up to 105 seconds. Again we saw a similar drop in heart rate during the breath hold. Hyperventilation over-oxygenates the blood and drives off excess CO₂, thus the urge to breathe is delayed by the reduced CO₂ concentration in the blood. After exercise the subject was hardly able to hold their breath at all, only managing 8 seconds. This makes sense as the metabolic demand by the body is high such that the urge to breathe overrides the conscious control of the breathing.

Physical Models

During the examination of the physical models of the lung we found that by inflating one alveolar balloon to a higher extent than the other and allowing the pressures to interact we saw the more inflated balloon become even more inflated while the less inflated balloon became less inflated. This relates to the material properties of the balloons and similarly to the alveolar sacs. The more stretched balloon is more compliant while the less stretched one requires more energy to induce any more stretch. This is important to understand in the lungs as it implies that the

lungs must inflate uniformly to ensure that every alveolar sac is inflated properly. This is more easily accomplished in healthy tissue by negative pressure than forced positive pressure. The demonstration of the negative pressure lung model showed that when we applied a negative pressure by pulling of the diaphragm both 'lung' balloons inflated equally.

Re-Breathing

The two rebreathing protocols produced different results both numerically and qualitatively. Despite the continuous decrease in oxygen concentration during the CO₂ scrub protocol, the subject did not report any trouble breathing or discomfort. We also did not see any change in heart rate or tidal volume indicating no mechanical changes in breathing were detected (Figure 2 and Figure 3). However when ample O₂ was provided but CO₂ was allowed to accumulate the subject reported difficulty breathing quickly, tidal volume increased, and heart rate slightly increased despite a constant and high SPO₂. This phenomena while initially surprising, should be expected. We know that breathing regulation is primary driven by sensing of CO₂ levels rather than O₂ levels. Thus when CO₂ is scrubbed, even though O₂ falls to levels that would be problematic for the body it does not sense a problem, whereas when O₂ is abundant but CO₂ is not cleared the breathing regulation systems sense a problem resulting in increased tidal volume, and a sensation of a need to breath or difficulty breathing.

Overall we found that, as expected, CO₂ is the principle driver of respiratory regulation as can be seen in the final experiments. A limitation of our study is the fact that the spirometers we used add a large amount of dead space to our subject's respiratory system. This could likely reflect in the increased tidal volumes we recorded as compared to literature values, and we have no good way of separating these effects from other explanations. Additionally we found that the pulseoximeter was limited especially post exercise in providing accurate measurements. To improve this we would likely utilize more direct measures such as an ECG and spirometry data as is done in a typical exercise lab.