Does Chronic Low Back Pain Influence Breathing Mechanics and Diaphragm Positioning? A Pilot Study

Lindsey Wensel

Brigham Young University
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A Pilot Study

Lindsey Wensel

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Brigham Young University
in partial fulfillment of the requirements for the degree of

Master of Science

Ulrike H. Mitchell, Chair
Dustin A. Bruening
Anton E. Bowden

Department of Exercise Sciences
Brigham Young University

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ABSTRACT

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Lindsey Wensel
Department of Exercise Sciences, BYU
Master of Science

Background and objective: Chronic low back pain (CLBP) is a complex musculoskeletal condition and often the source of the pain is not clear. A correlation has been found to exist between incidences of low back pain (LBP) and those with respiratory diseases such as COPD. This could give reason to believe that the sequence of events could be reversed, and LBP could elicit changes in respiratory function. The purpose of this study is to investigate if CLBP has an influence on breathing mechanics and the positioning of the diaphragm in the trunk.

Methods: Volunteers were recruited between the ages of 35-65 years old with and without CLBP. All subjects underwent an MRI for imaging of their diaphragm to find the position of the diaphragm at the end of exhalation and inhalation. The height of the diaphragm at the end of exhalation and inhalation was then measured. Respiratory values were measured that included forced expiratory volume in 1 second (FEV1), and respiratory amplitude at the thoracic and abdominal level both at rest and after a series of functional exercises. Respiratory amplitude was used to measure average displacement of the abdomen and mid-ribs during normal breathing. FEV1 was measured using a handheld spirometer and respiratory amplitude was measured using band-like respiratory sensors that were wrapped around the participants.

Results: A total of 36 participants were recruited for this study (n = 21M; n = 15F), with 18 controls (n = 11M; n = 7F) and 18 with CLBP (n = 10M; 8F). Eleven variables were assessed to compare the results from the control group and CLBP group and see if there were any differences. No statistically significant differences were found for all variables assessed.

Conclusion: There was no significant evidence there was a difference in diaphragm positioning and breathing mechanics in those who have CLBP. For future testing, we would want to change categorization to CLBP groups based on pain severity or compare the differences between acute LBP and CLBP. We would also consider changing our method for measuring the diaphragm such as measuring the percent change in area of the diaphragm between inhalation and exhalation. We would also consider other parameters to test that could include more use of the information given in the PROMIS questionnaire or looking at the amount of lumbar lordosis as seen on the MRI and how those values compare to among the different groups.

Keywords: back pain, chronic, breathing, mechanics, functional movement, imaging, diaphragm, spine
ACKNOWLEDGEMENTS

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Introduction

Low back pain (LBP) is a complex condition and there can be many reasons for why individuals experience it. Some causes for LBP could be related to structure such as facet impingement, disc degeneration or muscle spasms. Another possible cause for LBP could be related to respiratory dysfunction or disease. Those who suffer from chronic respiratory conditions, such as asthma and chronic obstructive pulmonary disease (COPD), have been reported to have a higher prevalence of LBP and higher levels of pain than those who do not have respiratory pathologies (Chen et al., 2017; Ferreira et al., 2013). There are often postural changes associated with chronic respiratory diseases (HajGhanbari et al., 2012), and these changes can lead to altered muscular dynamics and limited range of motion (ROM) of the rib cage and lumbar area. These altered muscular functions and ROMs result in higher muscular fatigue of spine stabilizing muscles which includes the diaphragm. This fatigue of spine stabilizers could result in further injury to the lumbar area and worsen any pre-existing inflammation of the connective tissue of the low back (Bordoni et al., 2018) which would then result in more instances of pain.

The diaphragm plays an important role in respiration and stabilizing the spine during different functional movements. The diaphragm has various attachments along the ribcage and thoracic vertebrae which allows it to work in a coordinated manner with the lungs during breathing. During inhalation, the diaphragm flattens and moves caudally allowing for greater expansion of the ribcage and thus allowing for greater expansion of the lungs (Nelson, 2012); this creates more efficient airflow during breathing. The diaphragm also has attachments on the lumbar spine at the anterior aspect of the L1 and L2 vertebrae via the left and right crura. This allows it to directly act on the upper lumbar vertebrae and stabilize it (Nelson, 2012). It also stabilizes the spine through increasing intrabdominal pressure by acting as the roof to the abdominal cavity when the transverse abdominus and pelvic floor contract together with the diaphragm (Akuthota & Nadler, 2004). This increase in intrabdominal pressure helps to stiffen the spine and prepares it to handle changes in movement and loading. A prime example of diaphragm utilization to increase intrabdominal pressure is during weightlifting. Intrabdominal pressure rises at a greater
magnitude during strenuous weightlifting than any other non-respiratory exercise (Al-Bilbeisi & Mc, 2000). The utilization of the diaphragm is so great during weightlifting that it could possibly be a method of increasing diaphragm strength (Al-Bilbeisi & Mc, 2000). If not functioning properly—be it due to fatigue or improper positioning (e.g., it is flatter or positioned higher in the trunk)—the diaphragm cannot contract with the needed force to stabilize the upper lumbar spine and generate adequate abdominal pressure. This can lead to spinal instability which subsequently can result in LBP. If not corrected, it can become a chronic issue.

Those with chronic respiratory diseases have altered postures which can contribute to diaphragm fatigue and decreased function. Those with chronic low back pain (CLBP) also have altered postures which would give reason to believe that the positioning and function of the diaphragm could be altered. If this is the case, then those with CLBP would present with respiratory dysfunctions seen in COPD populations such as altered breathing mechanics (i.e., shallow breathing, using accessory breathing muscles, decreased rib expansion (Beeckmans et al., 2016)), changes in diaphragm positioning (Janssens et al., 2013; Vostatek et al., 2013) and changes in vital lung capacity. Few studies have investigated incidences of respiratory dysfunction in those with CLBP. A relationship appears to exist between changes in respiratory function and instances of LBP. This relationship could be attributed to changes in diaphragm function and positioning. The aim of our study was to see if there is a difference in breathing mechanics and diaphragm function and positioning in those who CLBP compared to healthy individuals. We hypothesized that those with CLBP would have diaphragms that are positioned higher in the trunk and would have greater displacement through the ribcage compared to the abdomen when breathing.

**Methods**

**Participants**

Participants were recruited by word of mouth and advertising at local pain clinics, physical therapy clinics, chiropractic facilities and orthopedic offices throughout Utah County. The targeted age range was adults between the ages of 35-65 years old. We selected this age range
because it has the highest incidences of CLBP (Freburger et al., 2009). Those over the age of 65 were excluded to control for other potential disabilities or comorbidities that could contribute to alterations in their breathing mechanics.

Those who were categorized as being asymptomatic did not have CLBP but could still have LBP for which they did not seek treatment or take medicine to ease symptoms within the last 6 months prior to data collection. Those who were categorized as having CLBP had LBP every day for the last 3 months or 50% of the time within the last 6 months for which they sought treatment.

Individuals were excluded if they were diagnosed with scoliosis with a Cobb angle ≥ 35°, had previous lower extremity injuries or surgeries within the last 6 months prior to data collection, had pain radiating beyond the knee or any current respiratory conditions or diseases (i.e., COPD, asthma, etc.). Documentation of opioid use by the participants was taken since these drugs have been known to influence breathing.

**Data Collection**

Participants came to the McDonald Research Facility located at Brigham Young University. They consented to be a participant via a consent form prior to data collection. Participants provided their demographic information, medical history and filled out a PROMIS form for us to later assess their average pain and functional activity based on their group classification.

Imaging of the diaphragm was done using a Siemens TIM-Trio 3.0T MRI scanner. Participants were oriented in a supine position with their heads facing the bore. A Siemens Body 13 coil combined with the Spine 18 coil was used to image the diaphragm. The Body 13 coil was placed on the participant over the location of their diaphragm. Imaging of the diaphragm included its distal attachments at the first lumbar vertebrae via the diaphragmatic crura and its most superior point at the level of T6. It also included its anterior and lateral muscle bundles or “diaphragmatic slips” that attach proximally to the lower six ribs and costal cartilages and the xiphoid process.
Participants were asked to do breath holds at the end of inhalation and the end of exhalation for two separate imaging sequences. The length of each breath hold sequence was 19 seconds. The projection plane was placed sagittally in the axial topogram going paravertebrally on the right side, midway through the center of the vertebral body and edge of the thoracic wall (Vostatek et al., 2013). Slice thickness was 3.0 mm for inhalation and exhalation. The VIBE dynamic sequence for inhalation was as follows: 96 slices per slab, TR 4.0 ms, TE 1.74 ms, FOV 400 mm. The VIBE dynamic sequence for exhalation was as follows: 96 slices per slab, TR 4.2 ms, TE 2.09 ms, FOV 300 mm. Six more sequences were done to image the diaphragm during regular respiration. Three sequences used the TRUFI dynamic sequence, and three sequences used the GRE dynamic sequence. The sequence length was 21 seconds and slice thickness 8.0 mm for the TRUFI dynamic sequence. The TRUFI dynamic sequence was configured as follows: 1 slice per slab, TR 4.2 ms, TE 2.09 ms, FOV 300 mm. The sequence length was 19 seconds and slice thickness 5.0 mm for the GRE dynamic sequence. The GRE dynamic sequence was configured as follows: 1 slice per slab, TR 5.1 ms, TE 2.00 ms, FOV 300 mm.

Respiratory parameters in this study were measured using Bio-Medical Instruments’ Respiration Sensor SA9311M and SpiroLink’s handheld Spirometer Model B1. The respiratory sensors were used to measure anterior and posterior displacement of the chest and abdomen in a standing position. The spirometer was used to measure forced expiratory volume in one second (FEV1), also in a standing position.

Before donning the sensors, the participants were asked to do the FEV1 test. Participants were instructed on how to do the FEV1 test by a research assistant. The instructions given were as follows: (1) plug your nose, (2) take a deep breath in, (3) place mouth over the mouthpiece, (4) exhale as hard and as quick as you possibly can and (5) relax. They performed this test three times and the FEV1 displayed on the screen of the spirometer was recorded each time to get an average FEV1. Once they finished this test, the respiratory sensors were donned.
The respiratory sensors were placed on the participants at the thoracic and abdominal level. The thoracic sensor was placed at midsternal level and the abdominal sensor was placed at the midline of the abdominal wall just above the umbilicus (Figure 1.) (Gastinger et al., 2010). While fastening the sensors, the participants were asked to fully inhale and exhale. At the end of full exhalation, the sensors were quickly fastened so that they were fully secured and with slight tension on the band. The bands were checked to make sure the sensors were not too loose, but still comfortable for the participants (Ltd., 2021) and that the band remained level around the circumference of their trunk once it had been fastened. Sensor signaling was recorded using v6.0 Infiniti Software with the ProComp 2 encoder on a DELL Inspiron 13-7353 model P57G.

Once the sensors were on, we recorded their resting breath 3 times for 30 seconds. After these measurements were taken, the sensors were taken off and the participants performed 20 minutes of functional movements as part of another study they were selected to participate in (Baker et al., 2023). After they performed the final movement, the respiratory sensors were put back on the participants to record their breathing. This time their breathing was recorded one time for 2 minutes. This period allowed for the recording of their breath recovery.

**Data Analysis**

All MRI scans were analyzed using the OsiriX Lite © Pixmeo Sarl – Switzerland (software version UDI-PI: 13.0.2) program. This program is a storage and processing software that allowed for visualization of the images in a DICOM format. One point of interest in this study was the position of the diaphragm at rest (at the end of exhalation) and at the end of inhalation. The sagittal view of the diaphragm was used to obtain these measurements. Using the OsiriX
software, a point was positioned on the spinous process of the L1 vertebra on each image as a reference point. A vertical line was then drawn from this reference point posterior to anterior to the anterior wall of the abdomen. After this line was drawn, another line was drawn perpendicular to it, inferior to superior to the apex of the diaphragm (the highest point in which it sat) (Figure 2.). The length of the latter line, representing the height of the diaphragm was measured and recorded.

The respiratory sensors visually provided the amount of chest and abdominal wall expansion through a waveform. The amplitude of each waveform for thoracic and abdominal expansion were measured. The Infiniti software provided the average displacement for the thoracic and abdominal sensors. These averages were then used to determine the differences between thoracic and abdominal displacement. The average distance between each wave peak was also measured at both the thoracic and abdominal level. We defined distance as the time between each peak. We found this average by using the MATLAB® version R2022b program. The data from the 2-minute breathing trial were exported from the Infiniti software to a spreadsheet. This spreadsheet was then saved as a file and uploaded to the MATLAB® program, so it could read the file. We then used the findpeaks() function within MATLAB to locate all peaks using the [pks, locs] function and the [dist = mean(diff(x))] function to determine the average distance between each of the located peaks.

**Statistical Analysis**
We used an independent group t-test to analyze our data. A p-value <0.05 was considered statistically significant. Our independent variable was group type—symptomatic or asymptomatic—and our dependent variables were: (1) the difference of thoracic displacement from abdominal displacement, (2) average thoracic displacement for all 30-second trials, (3) average abdominal displacement for all 30-second trials, (4) average thoracic displacement during the 2-minute respiratory trial, (5) average abdominal displacement during the 2-minute respiratory trial, (6) the difference found in average displacement during the 2-minute trial, (7) average spirometry values, (8) positioning of the diaphragm at the end of exhalation, (9) the change in diaphragm positioning from inhalation to exhalation, (10) the time between peaks for the abdominal waveform and (11) the time between peaks for the thoracic waveform.

Results

We recruited 36 participants (n = 21M; n = 15F), of whom 18 were controls (n = 11M; n = 7F) and 18 were CLBP (n = 10M; 8F) subjects. The average age of our participants was 48.42 ± 7.67 years, the average height was 173.99 ± 11.43 cm. There was no significant difference between groups for age, weight, height, and BMI (Table 1). One thing to note when analyzing data for the average time between breaths, the recorded session was not saved for 5 of the controls. Therefore, their data could not be included in that particular analysis because we could not go back and extract the data from that session like we did for all other participants.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Control (n=18)</th>
<th>CLBP (n=18)</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>48.78 ± 9.01</td>
<td>48.06 ± 6.26</td>
<td>0.78</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>174.84 ± 10.09</td>
<td>173.14 ± 12.87</td>
<td>0.66</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>86.46 ± 21.56</td>
<td>80.13 ± 16.96</td>
<td>0.35</td>
</tr>
<tr>
<td>BMI (kg/cm)</td>
<td>28.07 ± 5.89</td>
<td>26.47 ± 3.08</td>
<td>0.31</td>
</tr>
<tr>
<td>Pain Level†</td>
<td>1 ± 1.17</td>
<td>4.28 ± 1.91</td>
<td>&lt; 0.001*</td>
</tr>
<tr>
<td>PROMIS Score**</td>
<td>55.91 ± 7.69</td>
<td>44.74 ± 6.2</td>
<td>&lt; 0.001*</td>
</tr>
</tbody>
</table>

Table 1. Data are presented as mean ± standard deviation.  
*Tested variable had significant statistical difference with a p-value < 0.001  
**Score presented as a t-score on a scale from 0-100. A lower score indicated lower functional activity.  
†Pain was scored on a scale of 1-10 with 10 being the highest level of pain
There was no significant difference among the different measurements for respiration and diaphragm positioning (Table 2.). Abdominal displacement during the 30-second trial, thoracic displacement during the 2-minute trial and time between breaths at the abdominal level had smaller p-values compared to the other respiratory measurements. Representative waveforms from which we got the average displacement and time between breaths for our data analysis are provided for both a CLBP and control subject (Figure 3a-d. & Figure 4a-d.).

<table>
<thead>
<tr>
<th>Variable</th>
<th>Control (n=18)</th>
<th>CLBP (n=18)</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Abdominal and thoracic displacement difference (mm)</td>
<td>-0.56 ± 4.94</td>
<td>-1.44 ± 3.89</td>
<td>0.56</td>
</tr>
<tr>
<td>30-second trial: thoracic displacement (mm)</td>
<td>6.27 ± 2.77</td>
<td>5.96 ± 2.31</td>
<td>0.71</td>
</tr>
<tr>
<td>30-second trial: abdominal displacement (mm)</td>
<td>5.72 ± 4.50</td>
<td>4.52 ± 2.83</td>
<td>0.34</td>
</tr>
<tr>
<td>2-minute trial abdominal and thoracic difference (mm)</td>
<td>-1.09±5.75</td>
<td>-0.80 ± 4.32</td>
<td>0.87</td>
</tr>
<tr>
<td>2-minute trial: thoracic displacement (mm)</td>
<td>6.89 ± 4.10</td>
<td>5.56 ± 3.23</td>
<td>0.29</td>
</tr>
<tr>
<td>2-minute trial: abdominal displacement (mm)</td>
<td>5.79 ± 4.87</td>
<td>4.76 ± 2.91</td>
<td>0.44</td>
</tr>
<tr>
<td>Time between breaths (s): thoracic</td>
<td>4.51 ± 1.72*</td>
<td>4.74 ± 1.83</td>
<td>0.72</td>
</tr>
<tr>
<td>Time between breaths (s): abdominal</td>
<td>4.80 ± 1.48*</td>
<td>5.34 ± 1.66</td>
<td>0.36</td>
</tr>
<tr>
<td>FEV1 (L/s)</td>
<td>3.68 ± 0.02**</td>
<td>3.68 ± 0.02**</td>
<td>0.97</td>
</tr>
<tr>
<td>Diaphragm position at rest (cm)</td>
<td>0.59 ± 0.27**</td>
<td>0.59 ± 0.19**</td>
<td>0.99</td>
</tr>
<tr>
<td>Inhalation and exhalation difference (cm)</td>
<td>0.06 ± 0.04**</td>
<td>0.07 ± 0.06**</td>
<td>0.77</td>
</tr>
</tbody>
</table>

Table 2. Data are presented as mean ± standard deviation.

* n = 13 for the statistical analysis for this tested variable

** Data normalized by height
**Figure 3a-d.** Respiratory waveform result from a representative CLBP subject showing the results for CLBP subjects. A and B represent the waveform during the 2-minute trial and both the abdominal (A) and thoracic (B) level. C and D represent the waveform from one of the 30-second trials and both the abdominal (C) and thoracic (D) level.
We did not find any significant difference for diaphragm positioning among the two groups. All variables that tested for diaphragm positioning had p-values that showed little to no difference between those with CLBP and those without. The different positions and measurement values of the diaphragm at the end of inhalation and exhalation are provided for a representative subject with CLBP (Figure 5a-b.) and a control (Figure 5c-d). There was no significant difference between the two groups for FEV1 (p-value = 0.97).

Figure 4a-d. Respiratory waveform from a representative control showing what the results looked like for control subjects. A and B represent the waveform during the 2-minute trial and both the abdominal (A) and thoracic (B) level. C and D represent the waveform from one of the 30-second trials and both the abdominal (C) and thoracic (D) level.
Figure 5a-d. A and B show the positioning of the diaphragm in a representative CLBP subject. A represents the diaphragm at the end of inhalation. B represents the diaphragm at the end of exhalation. C and D show the positioning of the diaphragm in a representative control. C represents the diaphragm at the end of inhalation. D represents the diaphragm at the end of exhalation.
Discussion

The purpose of this study was to see if there was a difference in diaphragm positioning and breathing mechanics in those with CLBP. Our hypothesis was not supported because we did not find any significant differences between those with CLBP and healthy individuals for all tested variables. Our hypothesis was based on previous studies that found the diaphragm was positioned higher in the trunk for individuals with CLBP when compared to healthy individuals (Kolar et al., 2012) and studies that saw changes in breathing mechanics in those with LBP during different functional tasks (Hagins & Lamberg, 2011; Lamberg & Hagins, 2012; Shah et al., 2019; Smith et al., 2005). Our study was unique as we looked at respiratory characteristics in those with CLBP whereas the other studies primarily looked at different respiratory characteristics for those who had acute LBP. There are additional psychosocial problems associated with CLBP such as changed movements and behavior and we wanted to see if these changes would be observed during our investigation. This was thus an exploratory study to see if the changes that the other studies found in those who had LBP could also be found in those who had CLBP. There are a few explanations for why we did not see any differences.

We expected the diaphragm positioning to be similar to Kolar et al. (2012) findings in which they reported that their subjects who had CLBP had diaphragms that were positioned higher in the trunk than those who did not have CLBP. Kolar et al. (2012) discussed that one potential reason for why they saw the diaphragm positioned higher in those with CLBP is because those with LBP have been noted to have limited movement of the anterior and middle portions of the diaphragm which then leads to the diaphragm becoming weak. A possible reason for why we did not see these same differences in our study was we had our participants lying still while getting an MRI whereas Kolar et al. (2012) had their participants do isometric contractions of the lower and upper extremities. The abdominal muscles were more engaged at this point resulting in a different effect on the diaphragm compared to just lying relaxed in a supine position. We felt that by us not having our subjects do an isometric contraction of the limbs while scanning we would get a better representation of how the diaphragm moves during breathing and how it is positioned at rest.
Diaphragm positioning can also be affected by anything that affects intra-abdominal pressure such as high amounts of abdominal fat, accumulation of fluid in the peritoneal cavity or an enlarged liver (Nason et al., 2012). We did not control for BMI, so anyone with higher levels of abdominal fat could have presented with a diaphragm in an elevated position, especially considering that we had them laying in a supine position. When lying down, the additional weight across the abdomen pushes down on it which in turn displaces the diaphragm into a more elevated position (Qureshi, 2009). This would then mean they would have measurements that look no different from those with CLBP. There is a chance that some of our participants could have presented with an enlarged liver without our knowledge since we did not control for instances of liver disease or other diseases that could cause an enlarged liver. The right hemisphere of the diaphragm sits on top of the liver making this side sit slightly higher than the left side (O’Brien, 1928). When we did our measurements, we made them on the side where the liver was, so if a participant had an enlarged liver, it would have a significant influence on our outcome measures. We could have then had some controls with diaphragms positioned higher in their trunks than what we would have expected. For future research, we would control for BMI and change the side on which we measured the diaphragm, so we do not get any influence from the liver.

Hagins and Lamberg (2011) reported differences in breathing characteristics during a functional task in those who had LBP compared to healthy individuals thus we expected to find similar results with our CLBP subjects. One likely explanation for why we did not see similar changes could be due to the type of functional tasks. Hagins and Lamberg (2011) had their subjects perform various lifting tasks that progressively got more difficult. This could have then lead to greater exacerbation on those who had LBP leading them to change how they breathe to provide greater spinal stability during these lifting tasks. None of our functional movements included a lifting task, but rather they were very simple and most likely did not elicit any type of fatigue that would then require them to adjust how they breathe even after they completed the tasks and were in recovery. We also were not monitoring our subject’s breathing during the different functional tasks whereas Hagins and Lamberg (2011) did. Since the functional movements were part of another study they could not have been adjusted for this current study. However, for a future study we would include functional movements that included various lifting tasks and
increasing the weight of the object being lifted to elicit greater engagement of the abdomen and spine stabilizing muscles of the lower back. One thing to consider when doing lifting tasks is the current pain status of the participant. We would want to not increase their pain to the point to where their pain and discomfort is worse than what they were already experiencing.

Another reason for us not seeing any differences between the two groups could be due to the placement method we used for the respiratory devices. The respiratory sensors we used measured anterior/posterior (A/P) and lateral movement. However, chest breathing is more often associated with a greater superior rising of the chest (Nelson, 2012) which we did not capture. Alternative methods that could possibly capture this superior movement and provide significant results would be the use of optoelectronic plethysmography. This method can assess absolute chest wall volumes and how they vary in the lower and upper rib cage and abdomen (Houssein et al., 2019). This method works by placing a certain number of points on the outer surface of the chest wall and measuring the amount of displacement points (Layton, 2013; Parreira, 2012). Having the ability to choose where these points are placed and having more points of measurement could provide more information on the movement of the ribcage compared to the abdomen when measuring breathing mechanics.

The length of each trial may not have been long enough which could provide another explanation for why we did not find any significant differences. The 30-second trials may not have been long enough to get an accurate representation of the participant’s breathing pattern. The longer breathing trials somewhat indicate this. The p-value for the 2-minute trial at the thoracic level was lower than the 30-second trial. The p-values for the 2-minute and 30-second trial at the abdominal level did not vary much. However, the 2-minute trials still may have not been long enough. If done again, the 30-second trials would be eliminated from the protocol and trials longer than 2 minutes would be implemented.

We did not find any significant differences in the time between peaks at both the abdominal and thoracic level. We thought that there would be a shorter time between peaks for those with CLBP due to Glynn et al. (1981) finding that those experiencing pain have shorter and shallower breaths (i.e., hyperventilating) than those who do not have pain. The explanation they provided
as to why those with pain hyperventilate is most likely not because of the pain itself but rather from the individual’s anxiety towards the pain. Those who experience chronic pain develop changes in behavior that lead to fear of movement/reinjury, catastrophizing of their pain, and a general fear of pain (Asmundson et al., 1999; Linton, 2000; Vlaeyen & Linton, 2000). We did not see this pattern of behavior exhibited in our participants due to us not seeing significant changes in time between breaths. It is likely that the subjects we had were not experiencing high levels of pain at the time of testing. Therefore, they most likely did not have high levels of anxiety of their pain getting worse during testing thus resulting in no significant changes in their respiratory rate. One of the difficulties when testing CLBP populations is their pain fluctuates, so on the day of testing the pain they are experiencing that day may not accurately represent the pain they experience from day-to-day. If we were to do this again, we would want to create different groups within the CLBP population based on the severity of their pain to then see if there are differences between those with severe, moderate, and mild CLBP.

A key finding, based on previous studies, appears to be differences in acute LBP and CLBP cohorts. Many of the studies that we looked at found differences in breathing mechanics and positioning of the diaphragm, but they were looking at those who were experiencing acute LBP. It is likely that the changes made during acute bouts of pain are not long term and it is possible that over time the individual returns to having normal breathing mechanics.

Conclusion

There was no significant evidence there was a difference in diaphragm positioning and breathing mechanics in those who have CLBP. For future testing, we would want to change categorization to CLBP groups based on pain severity or compare the differences between acute LBP and CLBP since previous studies did find changes in breathing mechanics and diaphragm positioning in individuals with acute LBP. We would also consider changing our method for measuring the diaphragm such as measuring the percent change in area of the diaphragm between inhalation and exhalation in either the A/P or superior/inferior direction. We would also consider other parameters to test that could include more use of the information given in the PROMIS
questionnaire or looking at the amount of lumbar lordosis as seen on the MRI and how those values compare to among the different groups.
References


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