

# The Effect of Upper Chest Wall Restriction on Diaphragmatic Function

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**Abstract.** [Purpose] The purpose of the present study was to determine whether transient upper chest wall restriction would enhance diaphragmatic contribution to tidal volume in healthy subjects. [Methods] Changes of diaphragm thickness ( $\Delta Tdi$ ) were studied by ultrasound in 24 healthy male subjects in the supine position. Tidal volume ( $V_t$ ) and respiratory rate were measured by spirometer.  $\Delta Tdi$  with each breath was expressed as percentage of the thickness at the end of expiration ( $\Delta Tdi\%$ ) and the ratio of  $\Delta Tdi\%$  to tidal volume was calculated ( $\Delta Tdi\%/V_t$ ). The upper rib cage was compressed with a sphygmomanometer cuff to restrict its motion. All parameters were measured in three conditions: during rib cage compression, prior to the compression, and subsequent to the compression. [Results] When upper rib cage compression was applied,  $\Delta Tdi\%$  significantly increased, whereas it returned to baseline levels upon release of the compression.  $\Delta Tdi\%/V_t$  tended to increase, however there was no significant difference. Subjects were divided into those who showed an increase in  $\Delta Tdi\%/V_t$  (group 1:  $n=16$ ) and those who showed a decrease (group 2:  $n=8$ ). Prior to the compression,  $\Delta Tdi\%$  and  $\Delta Tdi\%/V_t$  of group 1 were higher than those of group 2. [Conclusion] These results suggest that in healthy male subjects transient upper rib cage restriction may enhance diaphragmatic contribution to tidal volume, particularly in those subjects with low initial diaphragm recruitment.

**Key words:** Diaphragm, Chest Wall, Ultrasonography

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## INTRODUCTION

In ankylosing spondylitis, diaphragm motion is increased due to restriction of the chest wall. Ankylosing spondylitis, a chronic inflammatory disease, affects not only the sacroiliac joints and the longitudinal ligament of the vertebral column but also the costovertebral joints and costochondral junctions, which may result in limited motion of the chest wall. Ventilation becomes dependent on the diaphragm whose excursion is greater than

normal<sup>1-3)</sup>. It was reported recently that in ankylosing spondylitis, the upper chest wall motion is reduced while there is a normal range of lower chest wall and abdominal motion<sup>4)</sup>. It has been indicated that ankylosing spondylitis patients are able to compensate for their limited upper chest wall motion by increasing diaphragm motion<sup>4,5)</sup>. However, it is unclear whether in healthy subjects such a compensatory breathing pattern is induced by upper rib cage restriction. Previous studies as to whether upper chest wall restriction induces

increased diaphragm motion are few, a only a limited amount of previous study has been done on the effect of chest wall restriction<sup>6-8</sup>). Deschamps et al.<sup>8</sup>) showed that external force on the front of the rib cage produced a decrease in abdominal volume at a given value of esophageal pressure, but they gave no description of the diaphragmatic contribution to ventilation that was supplied by the external force.

To evaluate respiratory muscle function, invasive methods using a balloon catheter system and electrophysiological techniques have generally been employed. However, it is not easy to use these methods. Ultrasound provides a viable tool for non-invasive measurement of muscle functions, particularly those of deep muscles. It is useful for the evaluation of the respiratory function of the diaphragm<sup>9-21</sup>). Ultrasound imaging of the diaphragm is evaluated by measuring the thickness and length of the zone of apposition against the rib cage, and the displacement of the dome of the diaphragm. Previous studies have shown that diaphragm thickness changes with lung volume and during maximum inspiratory pressure maneuver<sup>13,14</sup>), and that this technique is useful as a means of diagnosing of diaphragmatic function<sup>16,19,20</sup>).

The purpose of the present study therefore was to determine by ultrasound, whether transient upper chest wall restriction enhances diaphragmatic contribution to tidal volume in healthy subjects.

## SUBJECTS AND METHODS

### *Subjects*

The subjects of this study were 24 healthy males with an average age of  $21 \pm 1$  yr, an average height of  $1.73 \pm 0.06$  m, and an average weight of  $61.0 \pm 11.0$  kg (mean  $\pm$  standard deviation [SD]). Subjects with a history of respiratory, circulatory, or neurological disorders were excluded. The study was approved by the faculty's ethical committee, and all subjects gave their informed consent to participate in this study.

### *Methods*

To restrict upper rib cage motion, the upper rib cage was compressed with a sphygmomanometer cuff (length: 24 cm, width: 13 cm), which was placed on the front rib cage between the axillae and the xiphoid process and was held in place by an

inelastic band that was wrapped around the chest. The cuff was inflated to about 40 mmHg at functional reserve capacity. This compression was set at a pressure forcing slow exhalation by compressing the upper chest wall without being uncomfortable for subjects.

To assess diaphragmatic function, diaphragm thickness was measured by ultrasound. The diaphragm in the zone of apposition was imaged by B-mode ultrasound. To image the diaphragm, a 6.0 MHz ultrasound linear probe (Mirucube, Global Health Co., Ltd., Kanagawa, Japan) was placed on the chest wall at the 8th or 9th right intercostal space, between the antero- and mid-axillary lines<sup>13</sup>). The probe was held perpendicular to the chest wall. The diaphragm was clearly outlined by the pleural and peritoneal membranes as two bright parallel lines. On the diaphragm image, diaphragm thickness (Tdi) was measured from the middle of the pleural line to the middle of peritoneal line. Ultrasound images were taken with the transducer head in the same position, using skin landmarks to minimize repositioning errors, and the ultrasound images were recorded as a video file and stored for offline analysis. Ultrasound still images were extracted offline. Image measurement was conducted using a software package (Scion Image Beta 4.03 for Windows, National Institutes of Health, Frederick, MD). Three ultrasound images were obtained at the end of inspiration (Tdi.in: maximum of Tdi) and at the end of expiration (Tdi.ex: minimum of Tdi), from which mean values were calculated. Measurements were rounded to the nearest 0.1 mm. The change in Tdi ( $\Delta$ Tdi) with each breath was calculated as absolute values of difference between the thickness at the end of inspiration and the thickness at the end of expiration.  $\Delta$ Tdi was expressed as percentages of the thickness at the end of expiration ( $\Delta$ Tdi%). Furthermore, to express the diaphragmatic contribution to tidal volume, the ratio of  $\Delta$ Tdi% to tidal volume was calculated ( $\Delta$ Tdi%/Vt).

While capturing ultrasound images, Vt and respiratory rate (RR) were additionally measured by spirometer (HI-801, Chest MI Inc, Tokyo, Japan) during one-minute breathing with a face mask connected to the transducer.

Measurements were made under three conditions: prior to rib cage compression, i.e., pre-condition; during upper rib cage compression, i.e., compression-condition; and subsequent to upper rib

**Table 1.** Changes in diaphragmatic function and respiratory parameters (n=24)

	Pre	Compression	Post
Vt (l)	0.60 ± 0.11	0.59 ± 0.13	0.56 ± 0.13
RR (breaths/min)	15.8 ± 3.7	14.8 ± 2.5	15.0 ± 2.9
Tdi,in (mm)	1.89 ± 0.32	2.02 ± 0.36*	1.80 ± 0.33†
Tdi,ex (mm)	1.54 ± 0.30	1.53 ± 0.33	1.50 ± 0.30
ΔTdi (mm)	0.35 ± 0.17	0.49 ± 0.25*	0.30 ± 0.17†
ΔTdi%(%)	23.7 ± 13.4	34.1 ± 19.7*	20.9 ± 12.2†
ΔTdi%/Vt (%/l)	41.2 ± 26.0	59.1 ± 32.8	38.7 ± 25.3†

Values are expressed as mean ± SD. Vt: tidal volume, RR: respiratory rate, Tdi,in: diaphragm thickness at the end of inspiration, Tdi,ex: diaphragm thickness at the end of exhalation, ΔTdi: Tdi,in minus Tdi,ex, ΔTdi%: ΔTdi relative to Tdi,ex, \*: comparison with pre condition (p<0.05), †: comparison with compression condition (p<0.05).

cage compression, i.e., post-condition. After donning a facemask, the subject was asked to lie supine with a pillow under the head and to relax. Upon confirmation of steady breathing, ultrasound imaging began, and respiratory parameters were measured. The cuff was then wrapped around the upper chest wall, and this state was maintained for 3 minutes, while similar measurements were made. Finally, within 5 minutes of removal of the cuff, the measurements were repeated in a similar manner. Ultrasound measurements were conducted by the same operator, who was experienced and blinded to our hypothesis.

Results are given as means ± SD. The ultrasound and spirometer recorded variables during the three conditions were analyzed using Tukey's multiple comparison test. Statistical analyses were performed using SPSS14.0 (SPSS Japan Inc., Tokyo, Japan). Values of p less than 0.05 were taken as significant.

To assess intraoperator variability for measurements of ΔTdi and ΔTdi%, 10 different subjects were examined twice by the same operator on the same day. Between a given pair of measurements, subjects were re-positioned in the resting supine position. Bland-Altman plotting was performed for the assessment of repeatability. There were no systematic differences between the 2 ultrasound measurements at rest. The intraclass correlation coefficients (ICC) for ΔTdi and ΔTdi% was 0.925 and 0.944, and the standard errors of measurement (SEM) for ΔTdi and ΔTdi% was 0.07 mm (95% confidence interval [CI]: 0.03–0.12) and 3.0% (95% CI: 1.5–5.6), respectively. This result suggests that there is good short-term intraoperator

repeatability for diaphragm thickness measurements using ultrasound imaging. The SEM is given by the squares root of the within subject residual mean squares error. The SEM is used to determine the degree of change required in a given individual's measure to establish that change over and above measurement error. The minimum detectable change (MDC), which is called the reliable change index, is useful for interpreting the relevance of any changes recorded after an intervention. The MDC is calculated as follows:  $MDC = z\text{-score} \times \sqrt{2 \times SEM^2}$ . At the 95% confidence level, the MDCs for ΔTdi and ΔTdi% were 0.22 mm and 9.6%, respectively.

## RESULTS

The results of diaphragm thickness and respiratory parameters are summarized in Table 1. Compared to pre-condition, ΔTdi and ΔTdi% significantly increased in compression-condition, and returned to baseline levels in post-condition. ΔTdi%/Vt tended to increase in compression-condition (p=0.09), but significantly decreased in post-condition. Tdi,in increased in compression-condition and then decreased in post-condition, similar to ΔTdi and ΔTdi%. On the other hand, Tdi,ex did not significantly change among the three conditions. RR tended to decrease in compression-condition but Vt did not show any significant differences in any of three conditions (p=0.06).

In eight of the 24 subjects ΔTdi%/Vt decreased in compression-condition. We decided to divide subjects into those who showed an increase in ΔTdi%/Vt (group 1: n=16) and those who showed a

**Table 2.** Comparisons of diaphragmatic function and respiratory parameters in group 1 (n=16) and group 2 (n=8)

	Pre		Compression		Post	
	Group 1	Group 2	Group 1	Group 2	Group 1	Group 2
Vt (l)	0.61 ± 0.12	0.57 ± 0.11	0.59 ± 0.10	0.59 ± 0.18	0.57 ± 0.09	0.54 ± 0.19
RR (breaths/min)	15.9 ± 3.6	15.6 ± 4.0	14.4 ± 2.4	15.6 ± 2.8	14.8 ± 3.1	15.4 ± 2.6
Tdi,in (mm)	1.86 ± 0.37	1.93 ± 0.21	2.03 ± 0.38	2.01 ± 0.35	1.83 ± 0.36	1.75 ± 0.29
Tdi,ex (mm)	1.59 ± 0.33	1.42 ± 0.21	1.45 ± 0.31	1.71 ± 0.34	1.51 ± 0.30	1.48 ± 0.30
ΔTdi (mm)	0.26 ± 0.13	0.52 ± 0.10*	0.58 ± 0.25	0.30 ± 0.14*	0.32 ± 0.16	0.28 ± 0.19
ΔTdi%(%)	16.9 ± 8.6	37.5 ± 10.5*	41.8 ± 19.1	18.6 ± 9.3*	21.3 ± 11.3	20.0 ± 14.7
ΔTdi%/Vt (%/l)	28.1 ± 14.3	67.5 ± 24.6*	71.1 ± 31.4	35.1 ± 21.0*	37.5 ± 20.8	41.1 ± 34.1

Values are expressed as mean ± SD. Group 1: ΔTdi%/Vt increased by compression, Group 2: ΔTdi%/Vt decreased by compression, \*: comparison with group 1 (p<0.05).

decrease in (group 2: n=8) the inter-group differences in characteristics, since the variables in each condition were analyzed using the unpaired t-test (Table 2). The comparison revealed that although there were no significant differences in Vt or RR, in pre-condition, group 2 in ΔTdi, ΔTdi%, and ΔTdi%/Vt were significantly higher than group 1. In contrast, in compression-condition, ΔTdi, ΔTdi%, and ΔTdi%/Vt in group 2 were significantly lower than in group 1 and Tdi.ex tended to be higher in group 2 (p=0.07).

## DISCUSSION

In this study, we investigated, by ultrasonography, whether transient upper rib cage compression enhanced the diaphragmatic contribution to tidal volume in healthy male subjects. Transient upper chest wall compression resulted in a significant increase in ΔTdi and ΔTdi%. Though ΔTdi%/Vt also increased, there was no significant difference. It was also noted that eight of the 24 subjects showed a decrease in ΔTdi%/Vt during upper rib cage compression and that they had significantly higher values of ΔTdi%/Vt prior to the compression than the other subjects. These results may indicate that transient upper chest wall restriction elicited diaphragm recruitment only in those healthy male subjects who did not have relatively high in diaphragm recruitment to begin with.

In previous research into ventilation in ankylosing spondylitis, diaphragm contribution was estimated by the ratio between the chest wall and abdominal wall motion which was measured by magnetometer<sup>3</sup>, optoelectronic plethysmography<sup>5</sup>,

and laser techniques<sup>4</sup>). These methods are indirect assessments of diaphragmatic function. In the present study, we used ultrasound to assess diaphragmatic function more directly by measuring the thickness of the diaphragm. The reproducibility of the assessment of diaphragm thickness by this method has been found to be satisfactory<sup>23</sup>). We confirmed in our present study that short-term intraoperator repeatability for diaphragm thickness measurement is good (ICC = 0.944), and the MDC for ΔTdi% was 9.6%.

When the upper chest wall was compressed, ΔTdi% significantly increased and in pre-condition, ΔTdi%/Vt in group 2 was significantly higher than in group 1. This suggests that there was predominance of diaphragmatic contribution to tidal volume in group 2. In compression-condition, although the values of Tdi.in were comparable between the two groups, Tdi.ex in group 2 tended to be higher than in group 1. This was due to the way the two groups reacted to compression, i.e., the mean value in Tdi.ex increased in group 2 while it decreased in group 1.

The change in Tdi is closely associated with lung volume<sup>9,15</sup>). Cohn et al.<sup>15</sup>) demonstrated that Tdi increased with increasing lung volume. Because Tdi.ex in group 2 was increased by compression, we reasoned that functional residual volume was elevated by compression. In this condition, the respiratory variables were comparable between the two groups. The result therefore implied that the inspiratory rib cage muscle was recruited to compensate for a decrease ΔTdi%, while tidal volume was kept constant. Furthermore, we speculate that subjects in group 2 might have had to recruit the inspiratory rib cage muscles to avoid

unacceptable upper chest wall depression. If unchecked, compression and greater action on the part of the diaphragm would necessarily stretch the inspiratory rib cage muscle: with diaphragm shortening, the inspiratory rib cage muscle is passively stretched through the reduction of intrathoracic pressure<sup>24)</sup>. As we did not examine the rib cage muscle recruitment this time, it was beyond the scope of our study to determine the rib cage muscle contribution to ventilation.

In post-condition,  $\Delta Tdi\%/Vt$  significantly decreased reflecting a significant decrease in  $Tdi.in$ . The value of change in  $\Delta Tdi\%$  was above the MDC. These results suggest that the effects of enhancing diaphragm recruitment by upper rib cage compression cannot be sustained. Additionally, when the two groups were compared in post-condition, there was no significant difference in any of the variables of diaphragm thickness, unlike in the other conditions. These results must reflect that  $\Delta Tdi\%/Vt$  in group 2 did not return to the baseline after the application of upper chest wall compression. Also, diaphragmatic contribution to tidal volume in group 2 was close to that in group 1. A possible explanation for these results is that subjects in group 2 were conscious of their breathing pattern and were unconsciously using diaphragmatic breathing at rest before compression. Another explanation may be that there are differences in breathing pattern between individuals. A wide variation has in fact been reported for breathing patterns of normal supine subjects<sup>25)</sup>. Gottesman et al.<sup>16)</sup> showed that  $\Delta Tdi\%$  was  $37 \pm 9\%$  in normal subjects, which was similar to the result of group 2, but their result was calculated as change in diaphragm thickness from functional residual volume to total lung volume in the standing position. It is therefore impossible to compare the value calculated in our study with that reported by Gottesman<sup>16)</sup> because of the different measurement conditions. However, given that  $\Delta Tdi\%$  and  $\Delta Tdi\%/Vt$  in group 2 were more than twice as large as those of group 1, it may be argued that the values in group 2 were too high. Based on the above, it may be appropriate to say that the breathing patterns of group 2 were somewhat unnatural before compression.

We designed this study as an attempt to investigate whether transient upper rib cage compression would enhance diaphragmatic function in healthy male subjects. Although there

were individual variations, we found that the diaphragmatic contribution tended to be increased by upper rib cage compression. In order to effect a restriction on the upper chest wall, we applied compression with a sphygmomanometer cuff. This maneuver restricts the upper chest wall movement during inspiration but not expiration. In this regard, the upper chest wall restriction that we applied would be different from the way the upper chest wall is restricted in ankylosing spondylitis. However, we believe that transient upper chest wall restriction enhances the diaphragmatic contribution to tidal volume if not markedly exaggerating diaphragm breathing. In other diseases with upper chest wall restriction, even if it is temporary, it may in fact be the case that the diaphragmatic contribution to tidal volume is enhanced.

## REFERENCES

- 1) Josenhans WT, Wang CS, Josenhans G, et al.: Diaphragmatic contribution to ventilation in patients with ankylosing spondylitis. *Respiration*, 1971, 28: 331–346.
- 2) Hauge BN: Diaphragmatic movement and spirometric volume in patients with ankylosing spondylitis. *Scand J Respir Dis*, 1973, 54: 38–44.
- 3) Grimby G, Fugl-Meyer AR, Blomstrand A: Partitioning of the contributions of rib cage and abdomen to ventilation in ankylosing spondylitis. *Thorax*, 1974, 29: 179–184.
- 4) Ragnarsdottir M, Geirsson AJ, Gudbjornsson B: Rib cage motion in ankylosing spondylitis patients: a pilot study. *Spine J*, 2008, 8: 505–509.
- 5) Romagnoli I, Gigliotti F, Galarducci A, et al.: Chest wall kinematics and respiratory muscle action in ankylosing spondylitis patients. *Eur Respir J*, 2004, 24: 453–460.
- 6) Forkert L: Effect of regional chest wall restriction on regional lung function. *J Appl Physiol*, 1980, 49: 655–662.
- 7) Estenne M, Yernault JC, De Troyer A: Rib cage and diaphragm-abdomen compliance in humans: effects of age and posture. *J Appl Physiol*, 1985, 59: 1842–1848.
- 8) Deschamps C, Rodarte JR, Wilson TA: Coupling between rib cage and abdominal compartments of the relaxed chest wall. *J Appl Physiol*, 1988, 65: 2265–2269.
- 9) Wait JL, Nahormek PA, Yost WT, et al.: Diaphragmatic thickness-lung volume relationship in vivo. *J Appl Physiol*, 1989, 67: 1560–1568.
- 10) McKenzie DK, Gandevia SC, Gorman RB, et al.: Dynamic changes in the zone of apposition and diaphragm length during maximal respiratory efforts. *Thorax*, 1994, 49: 634–638.



- 11) Houston JG, Angus RM, Cowan MD, et al.: Ultrasound assessment of normal hemidiaphragmatic movement: relation to inspiratory volume. *Thorax*, 1994, 49: 500–503.
- 12) Cohen E, Mier A, Heywood P, et al.: Excursion-volume relation of the right hemidiaphragm measured by ultrasonography and respiratory airflow measurements. *Thorax*, 1994, 49: 885–889.
- 13) Ueki J, De Bruin PF, Pride NB: In vivo assessment of diaphragm contraction by ultrasound in normal subjects. *Thorax*, 1995, 50: 1157–1161.
- 14) Wait JL, Nahormek PA, Yost WT, et al.: Diaphragmatic thickness-lung volume relationship in vivo. *J Appl Physiol*, 1989, 67: 1560–1568.
- 15) Cohn D, Benditt JO, Eveloff S, et al.: Diaphragm thickening during inspiration. *J Appl Physiol*, 1997, 83: 291–296.
- 16) Gottesman E, McCool FD: Ultrasound evaluation of the paralyzed diaphragm. *Am J Respir Crit Care Med*, 1997, 155: 1570–1574.
- 17) Gerscovich EO, Cronan M, McGahan JP, et al.: Ultrasonographic evaluation of diaphragmatic motion. *J Ultrasound Med*, 2001, 20: 597–604.
- 18) Yamaguti WP, Paulin E, Shibao S, et al.: Ultrasound evaluation of diaphragmatic mobility in different postures in healthy subjects. *J Bras Pneumol*, 2007, 33: 407–413.
- 19) Yoshioka Y, Ohwada A, Sekiya M, et al.: Ultrasonographic evaluation of the diaphragm in patients with amyotrophic lateral sclerosis. *Respirology*, 2007, 12: 304–307.
- 20) Summerhill EM, El-Sameed YA, Glidden TJ, et al.: Monitoring recovery from diaphragm paralysis with ultrasound. *Chest*, 2008, 133: 737–743.
- 21) Kaneko H, Yamamura K, Mori S, et al.: Ultrasonographic evaluation of the function of respiratory muscles during breathing exercises. *J Phys Ther Sci*, 2009, 21: 135–139.
- 22) Haley SM, Fragala-Pinkham MA: Interpreting change scores of tests and measures used in physical therapy. *Phys Ther*, 2006, 86: 735–743.
- 23) Enright SJ, Unnithan VB, Heward C, et al.: Effect of high-intensity inspiratory muscle training on lung volumes, diaphragm thickness, and exercise capacity in subjects who are healthy. *Phys Ther*, 2006, 86: 345–354.
- 24) Mortola JP, Sant'Ambrogio G: Motion of the rib cage and the abdomen in tetraplegic patients. *Clin Sci Mol Med*, 1978, 54: 25–32.
- 25) Tobin MJ, Chadha TS, Jenouri G, et al.: Breathing patterns. 1. Normal subjects. *Chest*, 1983, 84: 202–205.