Effects of Altered Intra-abdominal Pressure on the Upper Airway Collapsibility in a Porcine Model

Shu-Lin Ren¹, Yan-Ru Li², Ji-Xiang Wu¹, Jing-Ying Ye³, Rachel Jen⁴
¹Department of General Surgery, Beijing Tongren Hospital, Capital Medical University, Beijing 100730, China
²Department of Otolaryngology Head and Neck Surgery, Beijing Tongren Hospital, Capital Medical University, Beijing 100730, China
³Department of Otolaryngology Head and Neck Surgery, Beijing Tsinghua Changgung Hospital, Tsinghua University, Beijing 102218, China
⁴Department of Medicine, Respiratory Division, University of British Columbia, Vancouver, BC, Canada

Abstract

Background: Obstructive sleep apnea is strongly associated with obesity, particularly abdominal obesity common in centrally obese males. Previous studies have demonstrated that intra-abdominal pressure (IAP) is increased in morbid obesity, and tracheal traction forces may influence pharyngeal airway collapsibility. This study aimed to investigate that whether IAP plays a role in the mechanism of upper airway (UA) collapsibility via IAP-related caudal tracheal traction.

Methods: An abdominal wall lifting (AWL) system and graded CO₂ pneumoperitoneum pressure was applied to four supine, anesthetized Guizhou miniature pigs and its effects on tracheal displacement (TD) and airflow dynamics of UA were studied. Individual run data in 3 min obtained before and after AWL and obtained before and after graded pneumoperitoneum pressure were analyzed. Differences between baseline and AWL/graded pneumoperitoneum pressure data of each pig were examined using a Student’s t-test or analysis of variance.

Results: Application of AWL resulted in decreased IAP and significant caudal TD. The average displacement amplitude was 0.44 mm ($P < 0.001$). There were three subjects showed increased tidal volume (TV) ($P < 0.01$) and peak inspiratory airflow ($P < 0.01$); however, the change of flow limitation inspiratory UA resistance (Rua) was not significant. Experimental increased IAP by pneumoperitoneum resulted in significant cranial TD. The average displacement amplitude was 1.07 mm ($P < 0.001$) when IAP was 25 cmH₂O compared to baseline. There were three subjects showed reduced Rua while the TV increased ($P < 0.01$). There was one subject had decreased TV and elevated Rua ($P < 0.001$).

Conclusions: Decreased IAP significantly increased caudal TD, and elevated IAP significantly increased cranial TD. However, the mechanism of UA collapsibility appears primarily mediated by changes in lung volume rather than tracheal traction effect. TV plays an independent role in the mechanism of UA collapsibility.

Key words: Abdominal Obesity; Intra-abdominal Pressure; Lung Volume; Obstructive Sleep Apnea; Trachea Traction

Introduction

Obstructive sleep apnea (OSA) is characterized by repetitive upper airway (UA) collapse and obstruction during sleep. Epidemiological studies have suggested a strong relationship between obesity and OSA. The prevalence of OSA is high in overweight and obese population, particularly in male with central abdominal obesity.[¹-³] Previous studies have confirmed that an anatomically narrower UA, potentially resulting from increased fat deposition surrounding the UA lumen, contributes to the development of OSA in perhaps most obese OSA patients.[⁴] Furthermore, increased intra-abdominal pressure (IAP) may help explain the strong associations between central obesity and OSA. Increased IAP is a predominate characteristic of central obesity.[⁵,⁶] Chronically increased IAP may influence the diaphragm position as well as the lung volume, which might affect UA collapsibility. Acute abdominal compression in patients with OSA might be associated with increased UA closing pressure.[⁷]

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Address for correspondence: Dr. Ji-Xiang Wu, Department of General Surgery, Beijing Tongren Hospital, Capital Medical University, Beijing 100730, China
E-Mail: wjx95@hotmail.com

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Mechanisms by which increased IAP may contribute to UA instability have been debated. One of the main theories is that elevated diaphragm decreases caudal longitudinal traction of the trachea through mediastinal structure, leading to worsen UA patency and increased UA resistance (Rua).\(^8\)\(^9\) Conversely, caudal tracheal displacement (TD) was shown to decrease the UA extra-luminal tissue pressure and abolish flow limitation in rabbits.\(^10\)\(^11\) In short, the longitudinal traction effect of the trachea tends to keep the airway open. Another possible theory is that the increase in end-expiratory lung volume, which may lead to increased transmural pressure gradients, may be associated with better UA patency. Hillman et al.\(^12\) reported that UA collapsibility was attributable to lung volume-related changes in pressure gradients rather than caudal displacement of the diaphragm.

Altered IAP may influence the lung volume, the degree of caudal tracheal traction exerted on the UA and consequently the UA collapsibility. However, the relative contributions of these effects have not been determined. Moreover, few previous studies have assessed the influence of IAP (both increases and decreases) on TD and collapsibility of UA.

We aimed to investigate the trachea movement, the ventilation as well as the Rua changes in altered IAP using \(\text{CO}_2\) pneumoperitoneum and abdominal wall lifting (AWL) in pigs in order to determine the relative contributions of the tracheal traction effect and the lung volume to the UA collapsibility. Differentiating them may permit better targeting of other therapeutic modalities.

**METHODS**

**Subjects**

Studies were performed on four adult, female, supine, and spontaneously breathing Guizhou miniature pigs (Liulihe Kexing Experimental Animal Center, Beijing, China) in Animal Laboratory of Capital Medical University. The weights of pigs were 42.5 kg (24 months), 27.8 kg (15 months), 24.8 kg (12 months), and 32.6 kg (18 months), respectively. The protocol was approved by the Animal Experiments and Experimental Animal Welfare Committee of Capital Medical University.

**Anesthesia**

Anesthesia was induced with an initial intramuscular injection of ketamine hydrochloride (10 mg/kg) and xylazine hydrochloride (3 mg/kg), and maintained via a continuous intravenous infusion of ketamine hydrochloride (5 mg·kg\(^{-1}\)·h\(^{-1}\)) and xylazine hydrochloride (1.5 mg·kg\(^{-1}\)·h\(^{-1}\)) through an ear vein.

**Experimental establishment**

Animals were studied with the head and neck position controlled such that a line drawn from the eye to the external nares was at 45° to the horizontal plane [Figure 1]. The subject’s mouth was closed, and a facemask (sealed with petroleum jelly) was secured over the snout. A Foley catheter (Weigao Group Medical Polymer Company Limited, Weihai, China) was placed in the urinary bladder via the urethra and connected to a bladder pressure measuring device. IAP was measured using a standardized protocol based on the modified Kron’s methods.\(^13\) Mechanical AWL system is widely used in laparoscopic surgery to obtain a surgical view.\(^14\) We use this system to simulate the decrease of IAP. Conversely, \(\text{CO}_2\) insufflation, otherwise known as pneumoperitoneum, is the most common approach to obtain a surgical view in laparoscopic surgery. \(\text{CO}_2\) insufflation is used to increase the IAP.

**Measurement of tracheal displacement**

A midline incision was made over the anterior surface of the neck, and the trachea was exposed. The second tracheal cartilage ring was connected to a draw-wire displacement sensor (JLMOON Technology Ltd., Beijing, China), and its end-expiratory and end-inspiratory position (caudal TD) was monitored (the wire was pulled out about 9–16 mm length beforehand).

**Measurement of airflow and pressure**

Respiratory airflow (V) was measured using a heated pneumotachograph (NR6 Rhinomanometer, GM Instruments Ltd., UK) attached to the tightly sealed face mask. The mask pressure (PM) and supraglottic pressure (SGP) were measured using a pressure sensor (CP110 Pressure Sensor, KIMO Instruments, France) [Figure 1]. A port was made surgically on the front of the thyroid cartilage lamina about 1.5 cm below the superior thyroid notch. The diameter of the port is about 0.5 cm. One tube of the pressure sensor was inserted into the superglottic region through the port, and the other one tube of the pressure sensor was connected with the side port of the mask. The driving pressure (Pd) (Pd = PM − SGP) for V can be measured and recorded through the polysomnography [Figure 1].

**Experimental protocols**

Two separate protocols were performed. Protocol No. 1 (AWL experiment) started from spontaneous resting IAP in the supine posture, and then the abdominal lifting system was
applied. The associated physiological parameters were measured during both resting IAP and abdominal lifting: TD, respiratory airflow (V), and Pd (Pd = PM − SGP). The protocol was performed with the pig’s head positioned at 45° to the horizontal plane [as referenced to a line drawn from the eye to the external nares in Figure 1] using a specially designed restraint which allowed flexion in the sagittal plane only.

Protocol No. 2 (pneumoperitoneum experiment) started from spontaneous resting IAP in the supine posture, and then graded CO₂ pneumoperitoneum pressure (15–25 cmH₂O, in 5 cmH₂O increments) was applied. Each run of graded pneumoperitoneum pressure was applied for 3 min, and the physiological parameters (IAP, TD, V, Pd) were recorded synchronously.

**Data analysis**

The airflow signal from the sealed face mask was integrated to obtain tidal volume (TV). Peak inspiratory airflow was averaged over breaths in 3 min for each IAP. To measure Rua, power functions (Pd = aVᵇ + c, where a, b, and c are constants, where the exponent b can be used to describe the flow regime) were fitted to the inspiratory limb of pressure-airflow plots generated from dozens of steady-state breaths in 3 min at each IAP level over a respiratory airway range of 0–180 ml/s. The Rua measurement was performed at a relatively low inspiratory airflow level (the airflow level conditions of the four pigs are different because of the weight difference) as this measurement can only be performed over a nonflow-limited part of the pressure/flow relationship. Airflow rates (V) for the subjects were 170 ml/s (pig 1), 150 ml/s (pig 2), 100 ml/s (pig 3), and 130 ml/s (pig 4), respectively.

**Statistical analysis**

Individual run data in 3 min were expressed as mean ± standard deviation (SD). The two-group t-test for independent samples was used to compare values obtained before and after AWL in protocol No. 1. Differences between baseline and graded pneumoperitoneum pressure data of each pig were examined using analysis of variance (ANOVA) in protocol No. 2. We considered P < 0.05 to indicate statistical significance. All statistical analyses were performed using the SPSS software (version 13.0.0 for Windows; SPSS Inc., Chicago, IL, USA).

**Results**

**Abdominal wall lifting experiment**

Application of AWL resulted in obvious decrease in IAP in all pigs. Compared to baseline, the IAP after application of AWL decreased from 9.0 to 6.5 cmH₂O in average (from 8 to 5 cmH₂O in pig 1, 7 to 5 cmH₂O in pig 2, 11 to 8 cmH₂O in pig 3, and 10 to 8 cmH₂O in pig 4, respectively).

A typical raw data tracing from one pig (pig 4) is shown in Figure 2. With the decrease of IAP, increase in caudal TD, TV, and peak inspiratory airflow was found. However, the decreased IAP did not significantly affect the Rua.

There was significant caudal TD during the AWL experiment. The average displacement amplitude was 0.44 mm (P < 0.001). Three subjects showed increased TV (P < 0.01) and increased peak inspiratory airflow (P < 0.01); however, the change of flow limitation inspiratory Rua was not statistically significant [Table 1 and Figure 3].

**Pneumoperitoneum experiment**

Application of graded CO₂ pneumoperitoneum pressure resulted in progressive increases in IAP in all pigs. A typical raw data tracing from one pig (pig 4) are shown in Figure 4. With increase of IAP, cranial TD increased. The TV increased and the Rua decreased.

Experimental increased IAP by pneumoperitoneum resulted in significant cranial TD. The average displacement amplitude was 1.07 mm (P < 0.001) when IAP was 25 cmH₂O compared to baseline. There were three subjects showed reduced Rua while the TV increased (P < 0.01). There was one subject (pig 3) had decreased TV and elevated Rua (P < 0.001) [Figure 5]. Table 2 shows the respiratory variables during baseline and pneumoperitoneum.

**Discussion**

The study described the impact of change in IAP on TD and UA collapsibility. The major finding of this study is that decreased IAP increases cranial TD significantly, and vice
versa. Acute IAP change was associated with change in TV, which was correlated with Rua as well as peak inspiratory flow. We believe the data to be clinically relevant because obesity and, especially central obesity, is well-linked to chronic intra-abdominal hypertension.

Changes in IAP may play an important role in the mechanism of development of OSA.

### Table 1: Respiratory variables during baseline and application of abdominal wall lifting

| Pig number | Baseline | AWL | P  | Baseline | AWL | P  | Baseline | AWL | P  |
|------------|----------|-----|----|----------|-----|----|----------|-----|----|
| Pig 1      | 12.55 ± 0.03 | 12.76 ± 0.02 | <0.001 | 12.74 ± 0.05 | 12.99 ± 0.04 | <0.001 | 58.64 ± 2.49 | 59.73 ± 2.09 | <0.001 |
| Pig 2      | 11.20 ± 0.29 | 11.29 ± 0.04 | <0.001 | 11.69 ± 0.37 | 11.73 ± 0.08 | <0.001 | 30.62 ± 6.04 | 38.22 ± 2.28 | <0.001 |
| Pig 3      | 10.32 ± 0.03 | 11.67 ± 0.04 | <0.001 | 10.53 ± 0.09 | 11.93 ± 0.04 | <0.001 | 40.44 ± 2.68 | 40.75 ± 2.65 | 0.368 |
| Pig 4      | 9.77 ± 0.02 | 9.86 ± 0.02 | <0.001 | 9.98 ± 0.04 | 10.13 ± 0.03 | <0.001 | 43.96 ± 2.36 | 45.26 ± 2.79 | 0.001 |

Values are expressed as mean ± SD. TDex: Tracheal displacement of the 2nd cranial tracheal ring at the end expiration; TDin: Tracheal displacement of the 2nd cranial tracheal ring at the end inspiration; TV: Tidal volume; PIF: Peak inspiratory airflow; Rua: Flow limitation upper airway resistance; AWL: Abdominal wall lifting; SD: Standard deviation.

### Figure 3: Individual data during baseline and application of abdominal wall lifting

The change in caudal tracheal displacement (a), tidal volume (b), peak inspiratory airflow (c), and flow limitation upper airway resistance (d) with the application of abdominal wall lifting are shown. AWL: Abdominal wall lifting.

### Influence of intra-abdominal pressure on the tidal volume

Earlier studies have shown that thoracic pressure increases with the elevation of IAP. The change might be caused by diaphragmatic elevation, leading to reduced thoracic volume and lung compliance. Stadler et al. reported a decreased TV when abdominal compression applied. In
obese population, abdominal fat accumulation and increased IAP may compress the lungs and decrease functional residual capacity. Without positive airway ventilator support, many morbidly obese patients are forced to sleep in the sitting position, which probably helps to lower the compression of IAP on thoracic cavity. Whether normal-weight people have a similar regulation or not remains unclear.

Our results showed that TV increased in most of our animals during application of pneumoperitoneum. A discrepancy of the TV change was also noted between pig 3 and the other pigs. The exact reason is unclear; we suspected that it is possibly related to the baseline status of the subjects as well as the method to increase the IAP. Furthermore, decreased TV and consequently blood gas change may stimulate the chemical receptors, which in turn, increase the respiratory drive and TV. Perhaps this could explain the phenomenon of TV inconsistency.

In this study, all of the pigs showed elevated TV with the decrease of IAP. Decreased IAP-induced by AWL may reduce the abdominal fat encroachment upon the lungs.

**Influence of intra-abdominal pressure on the trachea position**

Decreased IAP might contribute to diaphragm descent-related mediastinal traction, while increased IAP may push the diaphragm superiorly, raise pleural pressure, and displaced the trachea cranially. During the application of pneumoperitoneum, the caudal TD decreases while the TV increases. Our results supported that mediastinal traction reduce the abdominal fat encroachment upon the lungs.

**Table 2: Respiratory variables of 4 pigs during baseline and pneumoperitoneum experiment**

| Items          | Baseline | IAP (cmH\(_2\)O) | F         | P      |
|----------------|----------|------------------|-----------|--------|
|                |          | 15               | 20        | 25     |
| TDex (mm)      |          |                  |           |        |
| Pig 1          | 12.58 ± 0.07 | 12.46 ± 0.21*    | 12.20 ± 0.04*      | 11.29 ± 0.07*  | 53144.559 | <0.001 |
| Pig 2          | 15.87 ± 0.18 | 15.69 ± 0.24*    | 15.72 ± 0.17*      | 15.61 ± 0.13*  | 170490.00 | <0.001 |
| Pig 3          | 14.03 ± 0.01 | 13.57 ± 0.04*    | 13.44 ± 0.06*      | 12.57 ± 0.03*  | 950494.251 | <0.001 |
| Pig 4          | 10.09 ± 0.04 | 9.68 ± 0.01*     | 9.27 ± 0.02*       | 8.83 ± 0.0*    | 877359.810 | <0.001 |
| TDin (mm)      |          |                  |           |        |
| Pig 1          | 12.59 ± 0.12 | 12.59 ± 0.25     | 12.25 ± 0.10*      | 11.28 ± 0.09*  | 1397741.00 | <0.001 |
| Pig 2          | 16.13 ± 0.15 | 15.98 ± 0.21*    | 16.25 ± 0.28*      | 15.98 ± 0.12*  | 93764.00 | <0.001 |
| Pig 3          | 14.04 ± 0.09 | 13.67 ± 0.18*    | 13.52 ± 0.14*      | 12.59 ± 0.12*  | 18108.00 | <0.001 |
| Pig 4          | 10.29 ± 0.06 | 9.78 ± 0.03*     | 9.40 ± 0.03*       | 8.87 ± 0.01*   | 1110994.70 | <0.001 |
| TV (ml)        |          |                  |           |        |
| Pig 1          | 66.46 ± 5.34 | 65.28 ± 5.28*    | 71.53 ± 7.19*      | 78.87 ± 7.73*  | 353578.00 | <0.001 |
| Pig 2          | 32.93 ± 14.21 | 32.98 ± 16.06  | 36.24 ± 16.76*     | 36.69 ± 14.55* | 3915.00 | 0.009 |
| Pig 3          | 46.78 ± 12.32 | 44.92 ± 8.79     | 41.51 ± 7.81*      | 39.32 ± 7.24*  | 13988.00 | <0.001 |
| Pig 4          | 44.07 ± 5.19  | 45.53 ± 5.04*    | 48.02 ± 5.11*      | 47.03 ± 5.52*  | 20228.00 | <0.001 |
| PIF (ml/s)     |          |                  |           |        |
| Pig 1          | 196.46 ± 10.28 | 165.26 ± 8.50*  | 184.62 ± 16.75*    | 220.15 ± 10.99* | 398073.00 | <0.001 |
| Pig 2          | 133.38 ± 23.13 | 134.60 ± 26.09  | 132.96 ± 27.12     | 134.30 ± 26.91 | 202.00 | 0.895 |
| Pig 3          | 106.18 ± 7.03  | 116.21 ± 10.91*  | 121.21 ± 13.83*    | 119.17 ± 10.54* | 40903.00 | <0.001 |
| Pig 4          | 132.85 ± 7.73  | 143.78 ± 7.28*   | 156.96 ± 5.21*     | 167.50 ± 7.67* | 258856.00 | <0.001 |
| Rua (cmH\(_2\)O·ml•1•s•10\(^{-3}\)) |          |                  |           |        |
| Pig 1          | 3.43 ± 0.70 | 5.28 ± 0.76* | 3.90 ± 1.30* | 2.41 ± 0.47* | 23153.00 | <0.001 |
| Pig 2          | 6.73 ± 2.49 | 6.58 ± 2.92 | 5.13 ± 1.90 | 4.14 ± 2.19* | 2638.06 | 0.07 |
| Pig 3          | 7.60 ± 1.73 | 10.07 ± 2.20* | 9.22 ± 1.18* | 8.75 ± 1.72* | 9215.00 | <0.001 |
| Pig 4          | 8.55 ± 0.85 | 8.08 ± 0.76 | 6.79 ± 0.65* | 6.85 ± 1.49* | 10663.00 | <0.001 |

*P<0.05 compared with baseline; †P<0.05 compared with IAP = 15 cmH\(_2\)O; ‡P<0.05 compared with IAP = 20 cmH\(_2\)O. Values are expressed as mean ± SD. TDex: Tracheal displacement of the 2nd cranial tracheal ring at the end of inspiration; TDin: Tracheal displacement of the 2nd cranial tracheal ring at the end of inspiration; TV: Tidal volume; PIF: Peak inspiratory airflow; Rua: Upper airway resistance; SD: Standard deviation; IAP: Intra-abdominal pressure.
rather than lung volume changes may contribute more to TD. We deduce that the chronically increased IAP may have a negative impact on UA patency.

Influence of intra-abdominal pressure on the upper airway resistance

The IAP may influence the Rua through different ways. Increased IAP may redistribute blood volume, and this may have had a direct effect on UA patency through alterations in central venous pressures.\cite{19,20} IAP may influence the Rua through the TD change as our study showed. Previous studies concluded that caudal tracheal traction could stabilize the UA by both reducing the extra-luminal tissue pressure and increasing longitudinal strain.\cite{10} Also, altered lung volumes may change surface tension in the airway, which could accentuate or attenuate pharyngeal collapsibility.\cite{21}

Interestingly, we found a positive relationship between the peak inspiratory airflow and the TV despite the TD changes. As the data showed, Rua reduced as the TV increase and vice versa, implying that Rua has a more close relationship to the lung volume compared to TD. The potential mechanisms may involve both the incensement of longitudinal strain and the reduction of the extra-luminal tissue pressure.

Kairaitis et al.\cite{16} found that increasing lung volume displaced the trachea caudally, reduced extra-luminal tissue pressure, abolished flow limitation, but had little effect on resistance or conductance, whereas decreasing lung volume resulted in cranial TD, increased extra-luminal tissue pressure and increased resistance. His study partially supports our view that Rua has a more close relationship to the lung volume compared to TD.

Clinical implications

Bariatric surgery has been proved to be effective in the treatment of OSA of morbidly obese patients. The indications for bariatric surgery as a treatment of OSA need to be further standardized and improved. In our study, we focused on the effects of IAP to the UA collapsibility; the outcomes supported that decreased IAP may be an important mechanism of improving sleep apnea and that the lung volume as a key targeting of this therapy. Considering the fact that not all the obese individuals with OSA have elevated IAP, for those patients who need bariatric surgery, IAP examine may be helpful.

Critique of methods

The study examined the effect of an acute alteration in IAP, which may not be representative of what happens in obesity group, where IAP increases chronically. Papavramidis et al. created a rabbit model in which IAP were chronically increased and maintained via an intra-abdominal balloon.\cite{22} More studies are needed to reveal the effect of chronic IAP change on UA collapsibility.
We did not measure the end expiration lung volume (EELV) in our study. EELV may have an important effect on UA collapsibility during expiration. In Stadler’s study,[7] pneumatic cuffs were inflated to the abdomen of the obese male OSA patients and abdominal compression significantly decreased EELV and UA closing pressure but not Rua.

Measurement of TD and SGP is an invasive process. Moreover, application of AWL and CO₂ pneumoperitoneum might cause pain; those may result in some disruption to the study. It should be noted that movements of the legs were not investigated during the experiment.

In conclusion, acute altered IAP results in significant TD generated by diaphragm position modification. However, the TD is so small that it may minimally change the Rua. The Rua has a more close relationship to the TV compared to TD. It is not possible to determine the precise mechanisms underpinning changes in Rua with AWL and CO₂ pneumoperitoneum in this study, but there is evidence that TV plays an independent role in the mechanism of UA collapsibility.

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**Conflicts of interest**

There are no conflicts of interest.

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