Intrathoracic Major Vessels, Trachea and Main Bronchi :
The Effect of Respiration on Size[1]

Kul Ho Jung, M.D., Byung Kook Kwak, M.D., Chi Hoon Choi, M.D.
Yong Ok Park, M.D., Hee Yeoun Goo, M.D., Shin Hyung Lee, M.D., Chang Joon Lee, M.D.

Purpose : To evaluate the effect of respiration on the sizes of intrathoracic vasculature, and the trachea, and the main bronchus.

Materials and Methods : Seventeen volunteers (10 males aged 20 — 39 years and 7 females aged 20 — 39 years) underwent spiral CT, between the apex and lowest base of the lung, collimation was 10 mm, pitch was 1, and images were obtained at breath hold forced end-inspiration and breath hold forced end-expiration. Cross secional areas or diameters were measured in each respiration state at the aorta (ascending, descending, lower thoracic) and great branches, the IVC (thoracic, abdominal), the SVC, pulmonary artery (right main, left descending) and the tracheobronchus (trachea, left upper bronchus). Changes in the size of vessels and airways between the respiration states were evaluated and compared between inspiration and expiration.

Results : During breath-hold forced end-inspiration CT, the ascending, descending, and lower thoracic aorta and its branches (brachiocephalic, left common carotid, left subclavian) as well as the thoracic IVC and SVC and the right main and left descending pulmonary arteries decreased in size : during breath-hold forced end-expiration CT, the size of all these vessels increased. For the trachea, left upper lobe bronchus and abdominal IVC, the situation was reversed. Statistically significant changes(p < 0.05) were noted in the ascending aorta and descending aorta, the lower thoracic aorta, the thoracic and abdominal IVC, the SVC, the right main and left pulmonary arteries, and the trachea.

Conclusion : During respiration, changes in the size of the thoracic vasculature and airways is probably due to changes in intrathoracic pressure. In the measurement and diagnosis of stenosis or dilatation in the intrathoracic vesculature and airways, respiration states should therefore be considered.

Index words : Computed tomography(CT), helical
　　　　　　　Aorta, CT
　　　　　　　Venae cavae, CT
　　　　　　　Trachea, anatomy
　　　　　　　Bronchi, CT

Computed tomography (CT) has become an accepted and widely available imaging technique for the evaluation of suspected major intrathoracic vascular and tracheobronchial diseases. With the introduction of technological improvements such as spiral CT and software, it is possible to evaluate the inspiratory and expiratory status of pulmonary diseases. CT has been used to establish the range of normal variation of aortic diameter (1) and to measure the diameter of the main pulmonary artery(2). Changes in thoracic great vessels especially the aorta and pulmonary artery, during respiration have not, however, been considered, though

the size and shape of the trachea and venae cavae during respiration have been reported(3—7). The purpose of this study was to determine changes in intrathoracic vascular and tracheobronchial size during respiration.

Material and Methods

As part of a study involving the measurement of lung volule, 17 normal volunteers(10 male, 7 female), aged 20—39 years who had undergone spiral CT were retrospectively evaluated(8). None suffured from hypertension, or cardiovascular, or renal disease, and pulmonary function measured by spirometry was normal. During each CT examination, for which a PQ 2000 (Picker International, Ohio, U.S.A.) was used, sequences were performed with the patient in the supine position from the lung apex to the base for one breath hold forced end-inspiration and one breath hold forced end-expiration. The parameters were as follows : 10-mm thickness, pitch 1, kVp/mA=130/150, matrix=512× 512, FOV=300 mm. Images were not contrast-enhanced. Raw data from spiral scans were reconstruc-ted at 5 mm intervals by overlapping each adjacent images by 5 mm to avoid image skipping.

The area or diameter of structures was measured by freehand tracing at window width/level of 300/40 HU, on a workstation(Voxel Q, Picker International, Ohio, USA). In each case, two observers (BK Kwak and KH Jung) measured the area or diameter of structures and calculated averages. The principles of measurement were, first, that during inspiratory and expiratory imaging phases, each structure was measured at the same level The ascending and descending aorta and SVC were measured at the level of the carina, where the ascending aorta is at its roundest (1) (Fig. 1) ; brachiocephalic, left common carotid and left subclavian artery, and the trachea at a point 2 or 3 slices (2—3 cm) above the top of the aortic arch (9) ; the right main pulmonary artery, at the level between the posterior wall of the superior vena cava and the anterior wall of the bronchus intermedius (2)(Fig. 1) ; left upper lobe bronchus and left descending pulmonary artery at the level of the left upper lobe bronchus, where it crosses the left descending pulmonary artery ; and the thoracic IVC and lower thoracic aorta at the level corresponding to the uppermost portion of the right diaphragm dome. In addition, abdominal IVC was measured below the caudate lobe of the liver.

Second, the area of all structures was measured at the level at which that structure was seen to be round, not oval.

Third, because cross-sectional areas at their point of origin vary slightly, branching structures such as the brachiocephalic, left common carotid and left subclavian arteries were measured slightly distal(2—3 cm) to their point of origin.

Fourth, cross sectional area better reflect change in the real size of a structure and for this reason, this was measured rather than diameter.

The significance of differences between measurements was determined at the $p < 0.05$ level using the paired-t test(SPSS for Windows, SPSS Inc., U.S.A.).

Results

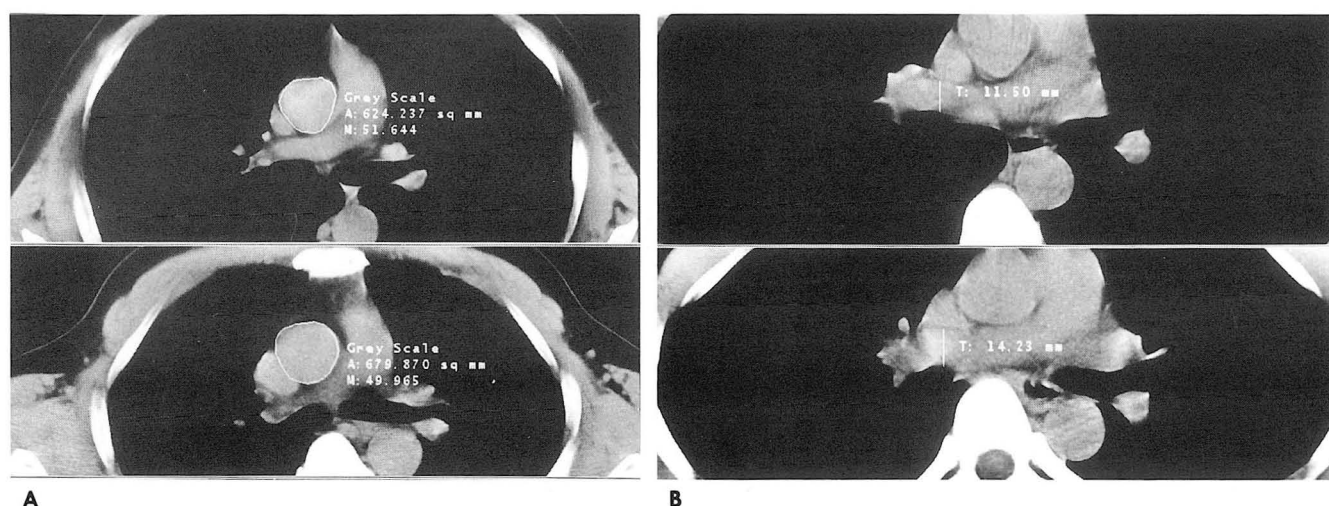Mean areas and diameters of all intrathoracic vascu-

A **B**

Fig. 1. The changes of the intrathoracic vasculatures between breath hold forced end-inspiration (upper) and breath hold forced end-expiration(lower).
A. Asce nding aorta, **B**. right main pulmonary artery.

lar structures, including aortas, venae cavae and pulmonary arteries, decreased during forced end-inspiration and increased during forced end-expiration. Statistically, the ascending and descending and lower thoracic aorta, SVC and thoracic IVC, and the right main and left descending pulmonary artery showed significant changes (p ⟨ 0.05) (Table 1). Major branches of the aorta (brachiocephalic, left common carotid and left subclavian artery) were not significant(p ⟩ 0.05).

On breath hold forced end-inspiration CT, the trachea and left upper-lobe bronchus increased in size, and on breath hold end-expiration CT, they decreased. The trachea showed significant change(p ⟨ 0.05), but the left upper-lobe bronchus did not(p ⟩ 0.05).

For the abdominal IVC, the situation was the reverse of that of the thoracic IVC ; that is, on breath hold forced end-expiration CT the area of the former decreased, and on breath hold forced end-inspiration CT it increased ; the differences were statistically significant(p ⟨ 0.05).

Discussion

Anatomically the cardiovacular system (heart, aorta, venae cavae and pulmonary vessels) and lungs are intimately associated within the thorax. Many of the effects of respiration are mediated through changes in left ventricular preload and afterload(10, 11). Lung CT was performed during breath-hold forced end-inspiration, a state similar to that existing during the Valsalva maneuver (forced expiration against a closed glottis after full inspiration) in the thorax. Shortly after the start of the Valsalva maneuver, the filling pressure of the heart is reduced by increased intrathoracic

pressure and remains low during the period of strain. Pressure in the intrathoracic great vessels such as the aorta and its branches, and the pulmonary artery falls and remains low(11, 12). The normal hemodynamic response to the Valsalva maneuver is a gradual decrease in arterial pressure, pulse pressure and stroke volume accompanied by tarchycardia(4). Although the clinical manifestations of the maneuver are well known, associated hemodynamic changes in the great vessels have not been extensively studied or documented. Breath hold forced end-expiration, on the otherhand, is similar to the Muller maneuver, which is associated with a decrease in intrathoracic pressure(13).

During the late strain phase of the Valsalva maneuver, a substantial reduction in the cross-sectional area of the SVC was noted. Although the form of the SVC during normal respiration is close to circular, during late strain the SVC is pressed against the aorta and it becomes more triangular(4). The Valsava maneuver has been shown to induce a 37 % decrease in the cross-sectional area of the SVC(4), a result similar to the 47.7 % decrease described in this report(Table 1).

The IVC is also highly compliant and its pressure is low ; its configuration varies with changes during ventilatory or circulatory events(5). The pattern of pressure distribution in the IVC is asymmetric and centered around the diaphragm with the pressure gradient in the abdominal IVC, results from decreased positive pressure, and in the thoracic segment from increased negative pressure(14). The abdominal IVC lumen decreased during the early inspiratory phase, reached a minimum at the end of the transient phase, as seen at a point 2 to 3 cm below the diaphragm during maximal inspiration. These ventilatory changes in the

Table 1. The Changes in the Size of Intrathoracic Major Vessels, Trachea and Bronchi at Breath Hold Forced End-inspiration and Breath Hold Forced End-expiration.

Structures	End-Inspiration(mm²)	End-Expiration(mm²)	p value	Increment(%)
Ascending aorta	572.9 ± 94.2	608.7 ± 106.3	⟨ 0.05	6.2
Descending aorta	362.5 ± 73.6	384.8 ± 78.9	⟨ 0.05	6.2
Lower thoracic aorta	299.2 ± 52.01	328.6 ± 52.6	⟨ 0.05	9.8
Brachi ocephalic artery	116.0 ± 19.7	124.1 ± 21.6	⟩ 0.05	7.0
Lt. common carotid a.	52.7 ± 8.4	58.4 ± 11.6	⟩ 0.05	10.8
Lt. Subclavian a.	58.3 ± 14.0	62.1 ± 17.4	⟩ 0.05	6.5
Superior vena cava	226.0 ± 50.0	333.7 ± 54.5	⟨ 0.05	47.7
Thoracic IVC	348.9 ± 98.4	430.3 ± 110.7	⟨ 0.05	23.3
Abdominal IVC	375.3 ± 149.5	221.9 ± 88.5	⟨ 0.05	−40.9
Trachea	272.7 ± 76.8	249.6 ± 82.3	⟨ 0.05	−8.5
Lt. upper lobe bronchus *	8.0 ± 1.5	7.5 ± 1.6	⟩ 0.05	−6.3
Rt. main pulmonary a. *	10.6 ± 1.7	13.1 ± 2.2	⟨ 0.05	23.6
Lt. descending pul. a.	154.5 ± 61.6	176.7 ± 63.7	⟨ 0.05	14.4

*, diameter(mm) ; Lt. : left ; Rt. : right ; IVC : inferior vena cava ; a. : artery

abdominal IVC reversed with increased intrathoracic pressure during the Valsalva maneuver and positive pressure ventilation (5). IVC response is a function of the interplay between intrathoracic and intra-abdominal pressure. During the Valsalva maneuver, increased intrathoracic pressure causes decreased abdominal IVC flow and resultant dilatation ; this is counteracted by increased intra-abdominal pressure, which causes collapse of the IVC(6).

In a previous study, MR angiography show that during maximum inspiration(breath hold forced end-inspiration), the AP diameter of the IVC was reduced by 21.7 %, and during Muller's maneuver by 35.7 %(7); because the current study measured area, not diameter, however, our data cannot be compared with that results. It was observed that the area of the abdominal IVC was reduced by 40.9 % during breath hold forced end-expiration, but that of the supradiaphragmatic thoracic IVC increased by 23.3 %(Table 1).

Only limited measurements of the aorta, right pulmonary artery and trachea have been published, and other than for the trachea, the measurement of structural change during respiration has not been reported (3, 9).

During the current study, certain principles of measurement were therefore applied. We wished to determine changes in each structure during respiration ; all measurements were therefore taken at the same level and configuration. In perpendicular structures, it is better to measure cross-sectional area than diameter, since the former better reflect changes in the real size of a struture(3, 9).

Our review of the world literature uncovered no report describing changes in the aorta and pulmonary artery during respiration. Shortly after the start of breath hold forced end-inspiration, the filling pressure of the heart is reduced by increased intrathoracic pressure, so the preload and the stroke volume of the heart are reduced. It therefore appears that during breath hold forced end-inspiration, the area of the aorta is reduced(4, 7, 10−13). Change in the area of the aorta was less than that of the venae cavae or pulmonary arteries(Table 1), though it was statistically significant. Because no previous reports have quatified the area of the aorta, we have no available comparable data. Normal thoracic aortic diameters reported by Aronberg et al. showed substantial variation according to age, sex, and thoracic vertebral body width(1). In addition to these results, respiratory states should be considered when evaluating the diameter of the aorta (1). The guideline for normal aortic size determined during inspiratory CT is unlikely to be the same during expiratory CT.

During late-stage Valsalva maneuver, a significant decrease was observed in the stroke volume, whereas the decrease in cardiac output was not significant. The heart rate increased significantly(4).

The mean cross-sectional area of aortic branches also changed according to respiration, but probably due to their small size and tortuosity, which made it difficut to precisely measure them, these changes were not significant.

The size of the pulmonary artery, already studied by O'Callaghan et al.(2), is a useful indicator of the presence or absence of pulmonary hypertension. The main pulmonary artery was measured at the intrapericardial segment between the posterior wall of the superior vena cava and the anterior wall of the intermedius bronchus. The mean size was 13.3 mm (SD, 1.5 mm) which is comparable with the current data, 10.6 mm. The slight difference seen here may be explained by the difference in age group between the two studies ; in ours, all voluteers were younger and - in addition - females were included ; a further difference is that we did not use contrast media. Among five pulmonary hypertensive patients previously studied(2), mean size ranged from 16.6 to 26.6 mm, which is well above the upper normal limit. They stated value was 16.3 mm + 2SD(95 % confidence limit for the upper limit of normal for normal patients, and probably at inspiration ; this last point was not-mentioned). In this current study, however, the diameter ranged from 9.9 mm to 17.6 mm at forced end-expiration, which is above the upper limit of normal ; to decide whether a patient is suffering from pulmonary hypertension, the state of respiration must therefore be considered.

It is well-known that pulmonary blood pressure, volume and flow change during each breath. Having found no significant differences between inspiration and expiration in pulmonary vascular resistance, characteristic impedance and overall impedance spectra in subjects free of cardiopulmonary disease, Murgo and Westerhof(15) concluded that quiet respiration has no effect on pulmonary arterial load, and that changes in pressure and flow must result from changes in right ventricular performance. Milnor(16, 17) states that total pulmonary vascular resistance increases slightly with inspiration, but no respiratory change in pulmonary vascular impedance has been reported.

On the other hand, the trachea and bronch increase in volume during sustained full inspiration. Stern et al (3, 9) found significant correlation between respiration and tracheal cross-sectional area, and showed that the trachea enlarges during inspiration. The change in tracheal cross-sectional area occuring during the respirat-

ory cycle is assumed to occur after anterior displacement of the posterior tracheal membrane, as is the observed decrease in the transverse diameter of the trachea(9). Stern et al reported the cross-sectional area of the trachea as $280 \, mm^2$ (range, $221-388 \, mm^2$; SD, 50.5) at end-inspiration and $178 mm^2$ (range, $115-236 \, mm^2$; SD 40.2) at end-expiration. These results are slightly different from ours, and this may be due to different levels during the two studies; in order to avoid the influence of continuing compression of the aortic arch, which enlarges during expiration, we did not select the level of aortic arch selected by Stern et al. Unlike in the trachea, change in the left upper-lobe bronchus is not significant; this is probably due to its small size, or compression of the adjacent pulmonary artery.

In conclusion, during respiration, change in the size of the thoracic vasculature and airways is changed probably due to changes in intrathoracic pressure. In the measurement and diagnosis of stenosis or dilatation in the intrathoracic vasculature and airways, respiration status should therefore be considered.

References

1. Aronberg DJ, Glazer HS, Madsen K et al. Normal thoracic aortic diameters by computed tomography. *J Comput Assist Tomogr* 1984;8:247-50

2. O'Callaghan JP, Heitzman FR, Somogygi JW, et al. CT evaluation of pulmonary artery size. *J Comput Assist Tomogr* 1982;6:101-4

3. Griscom NT, Wohl ME. Tracheal size and shape:effects of changes in intraluminal pressure. *Radiology* 1983;149:27-30

4. Eichenberger AC, Schwitter J, McKinnon GC, Debatin JF, von Schulthess GK. Phase-contrast echo-planar MR imaging:real-time quantification of flow and velocity patterns in the thoracic vessels induced by Valsalva maneuver. *J Magn Reson Imaging* 1995;5:648-55

5. Natori H, Tamaki S, Kira S. Ultrasonographic evaluation of ventilatory effect on inferior vena caval configuration. *Am Rev Respir Dis* 1979;120;421-7

6. Rak KM, Hopper KD, Tyler HN. The slit infrahepatic IVC: pathologic entity or normal variant? *J Clin Ultrasound* 1991;19:399-403

7. Carriero A, Iezzi A, Magarelli N, Severini S, Tartaro A. Whole body MR angiography:physiologic presaturation of the inferior vena cava. *Radiol Med* 1993;85:557-61

8. Kang HY, Kwak BK, Lee SY et al. Measurement of lung volumes:usefulness of spiral CT. 대한방사선의학회지 1996;35:709-14

9. Stern EJ, Graham CM, Webb WR, Gamsu G. Normal trachea during forced expiration:dynamic CT measurements. *Radiology* 1993;187:27-31

10. Abel JG, Salerno TA, Panos A, et al. Cardiovascular effect of positive pressure ventilation in humans. *Ann Thorac Surg* 1987;43:198-206

11. Fishman AP. *Dynamics of the pulmonary circulation.* In:Circulation(Vol. II). American Physiological Society 1963:1667-743

12. Sharpey-Schafer EP. Effects of Valsalva maneuver on the normal and failing circulation. *Br Med J* 1995;19:693-5

13. Mellins RB, Levin OR, Skalak R, Fishman AP. Interstial pressure of the lung. *Circ Res* 1969;24:197-212

14. Rabinovici N, Navot N. The relationship between respiration, pressure and flow distribution in the vena cava and portal and hepatic veins. *Surg Gynecol Obstet* 1980;151:753-63

15. Murgo JF, Westerhof N. Input impedance of the pulmonary arterial system in normal man:effects of respiration and compar-ision to systemic impedance. *Circ Res* 1884;54;666-73

16. Milnor WR. *Hemodynamics.* 2nd ed. Baltimore, Maryland, Williams & Wilkins, 1989

17. Castiglioni P, Di Rienzo M, Grossoni M, Tommasini R, Morpurgo M. Does spontaneous respiration alter pulmonary artery input impedance? *Eur Respir J* 1996;9:2328-34

호흡에 따른 흉곽내 대혈관 및 기관기관지의 크기변화[1]

[1]국립의료원 진단방사선과

정걸호 · 곽병국 · 최치훈 · 박용옥 · 구희연 · 이신형 · 이창준

목 적 : 최대 흡기와 호기시 흉곽내 대혈관및 기관지의 크기변화를 알아보고자 하였다.

대상 및 방법 : 정상 성인 남자 10명, 여자 7명(평균 연령 : 남자 30.9, 여자 26.1세)을 대상으로 하였다. 나선식 단층촬영을 single spiral(10mm thickeness, pitch 1)로 시행하여, 최대흡기와 호기시 흉곽내 중심혈관과 기관 및 주기관 지의 단면적 혹은 직경을 측정 하였다.

결 과 : 상행대동 맥, 하행대동맥, 상완두동맥간, 좌측 총경동맥, 좌측 쇄골하동맥, 상대정맥, 흉부 하대정맥, 우폐동맥 그리고 좌 폐동맥은 최대흡기시 크기가 감소하였고, 최대호기시 증가하였다. 기관, 좌측 주기관지는 최대흡기시 크기가 증가하였고, 최대호기시 그 크기가 감소하였다. 그러나 값의 변화가 통계학적으로 의미있는 것은 상행대동맥(p = 0.001), 하행대동맥(p=0.002), 상대정맥(p = 0.000),흉부 하대정맥(p = 0.01), 기관(p = 019), 좌우 폐동맥(p=0.01)이었다.

결 론 : 흡기시 증가된 흉곽내 압력은 정맥과 기관이나 기관지 뿐만아니라 동맥에도 영향을 미친다. 흉부 CT 판독시 혈관이나 기관이나 기관지의 협착 혹은 확장진단은 호흡상태를 고려하여야 한다.