Standard Cardiopulmonary Resuscitation Generates a Compression (Narrowing) of the Left Ventricular Outflow Tract in Humans

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This certifies that the master's thesis of Pei Ge Zhao is approved

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Abstract

Standard Cardiopulmonary Resuscitation Generates a Compression (Narrowing) of the Left Ventricular Outflow Tract in Humans

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Objectives: Little is known about how external chest compressions influence cardiac structures during standard cardiopulmonary resuscitation (CPR) in humans. The aim of this study is to investigate morphologic cardiac changes generated by external chest compression and the influence of these changes on the efficacy of CPR.

Subjects and methods: Thirty-four patients with non-traumatic cardiac arrest (24 males, mean age: 56 years old) were enrolled. Multi-plane transesophageal echocardiography (TEE) was performed during standard CPR, and a 135° longitudinal view was chosen to observe the left ventricular outflow tract (LVOT). An area of maximal compression (AMC) on LVOT was sought, and the degree of compression at the AMC (DEG_{com}) was calculated from the diameters of the LVOT or the aorta at the maximal compression during compression systole and compression diastole. Left ventricular areas and left ventricular volumes were measured by perimeter and area-length method respectively. Left ventricular stroke volume (LV_{SV}) was calculated by subtracting end-systolic volume from end-diastolic volume. We also investigated the correlation

between left ventricular stroke volumes and the degree of compression.

Results: The AMC was identified at the aorta including the aortic valve area in 20 patients (59%) and at the LVOT in 14 patients (41%). The AMC was located within 2 cm from the aortic valve in 79% of the patients. Compression of the AMC was noted in all patients and the degree of the compression at the AMC ranged from 19% to 83% (mean: 49±19%). Compared with the patients having the AMC at the aorta, the patients having the AMC at the LVOT had higher calculated stroke volume and larger left ventricular systolic and diastolic areas (44±15 vs. 29±16 ml, p=0.047; 14±7 vs. 6±3 cm^2, p=0.00; 22±10 vs. 10±7 cm^2, p=0.00).

Conclusion: Standard cardiopulmonary resuscitation generates variable degrees of compression of the LVOT or the aortic root. Narrowing of aortic root resulted in more adverse effect to the efficacy of CPR compared to that of LVOT. Lower hand placement of sternum for external chest compression might decrease this adverse effect and result in better hemodynamic outcomes of CPR.

Keywords: cardiopulmonary resuscitation; external chest compressions; left ventricular outflow tract

Ⅰ. Introduction

In 1960, Kouwenhoven et al. first reported the use of closed-chest cardiopulmonary resuscitation (CPR) in 20 patients [1]. Since then, CPR has been widely implemented in the world and documented to be effective to rescue patients with cardiac arrest. Many investigations have been carried out in order to investigate the physiologic mechanism by observing the opening or closure of the cardiac valves during external chest compression and to develop new methods of CPR [2-6]. Morphologic changes of the cardiovascular structures during external chest compression have been sought by using of two-dimensional echocardiography. Most of these previous studies focused on motions of cardiac valves [7-10] or diameter of descending aorta [11]. However, little has been known about how external chest compressions influence cardiac structures during standard CPR in humans. The aim of this study is to investigate the changes of cardiac structure generated by external chest compression and the influence of these changes on the efficacy of CPR.

Ⅱ. Subjects and methods

1. Subject

Thirty-four patients with non-traumatic cardiac arrest (24 males and 10 females, mean age: 56 years old) were enrolled in the study. Patients who had structural heart diseases or aortic disease on transesophageal echocardiography (TEE) were excluded. Patients who had destructive changes of the lungs on chest X-ray were also excluded. The study was reviewed and approved by the Human Investigation Committee of our institution. Informed consent could not be obtained from the patients, but verbal communication was made with a family member present.

2. Methods

2.1. Cardiopulmonary resuscitation

The team performed CPR as soon as the patient arrived at the emergency room or just after the cardiac arrest developed. CPR was performed in accordance with 2000 American Heart Association (AHA) guidelines for CPR and ECC [12]. The rate of chest compression was 100 per min. External chest compressions were done by one of emergency residents. Ventilation with 100% oxygen was provided with a self-inflating bag. One milligram of epinephrine was injected into the antecubital vein in every 3 minutes.

2.2. Transesophageal echocardiography

TEE was performed during CPR just after the completion of tracheal intubation and injection of first dose of epinephrine. A multiplane transesophageal probe (5 MHz, Ultramark-9, Advanced Technology Laboratories Inc., USA) was introduced into the esophagus to observe the heart and the ascending aorta. Immediately after the

transesophageal probe was inserted, the heart was briefly examined to search for possible causes of cardiac arrest. Any patient who had a morphologic abnormality of the heart or the aorta on TEE was excluded from the study. Left atrium–left ventricle two-chamber views at 90° and longitudinal view at 135° were used to inspect opening or closure of the mitral and aortic valve, and a horizontal four-chamber view to evaluate the extent of left ventricle compression. A 135° longitudinal view was taken to observe the left ventricular outflow tract (LVOT) and the ascending aorta. Images of the left ventricular outflow tract were obtained at the point of the maximal compression site. M-mode tracing was done at the area of maximal compression (AMC). All echocardiographic images were recorded with s-VHS videotape.

2.3. Analysis of recorded images

The images recorded during CPR were analyzed by a cardiac image analysis system (Imagevue, Novamicrosonics Inc., USA). After freezing recorded images from the cardiac analysis system, calibration was made by an internalized calibrator. The exact position of AMC was identified. Next，the distances from AMC to the aortic valve were measured in every patient. The maximal and minimal diameters of the LVOT or the aorta during external compression were measured. The ratio of maximal diameter to minimal diameter was calculated as the degree of the compression at the AMC (Fig. 1). The cross-sectional area of LVOT was also measured during compression systole and diastole. Left ventricular volume was calculated by the area–length method [12], by subtracting end-systolic volume from end-diastolic volume to acquire left ventricular stroke volume (LV_{SV}). Left ventricular systolic and diastolic areas were measured by perimeter method from the view at end-systole and end-diastole. Each measurement was calculated by taking the average value of five consecutive cycles from the recorded images. Analyses were performed and reviewed by an emergency physician and two cardiologists. If there were any disagreements, the image was reanalyzed.

According to the difference of the location of maximal compression (as shown in the section 3.1 location of the area of maximal compression), we divided all patients into two groups: LVOT and aorta group.

2.4. Statistical analysis

Data were coded into computerized data processing software (SPSS for windows 12.0, SPSS Inc., Chicago, IL). Continuous variables were analyzed by paired t-test or Mann–Whitney *U*-tests if appropriate, and statistical significance was noted at a *P*-value less than 0.05. All data are presented as mean±SD. Stroke volume, left ventricular systolic area and left ventricular diastolic area between the aorta group and LVOT group were compared by Student t-test.

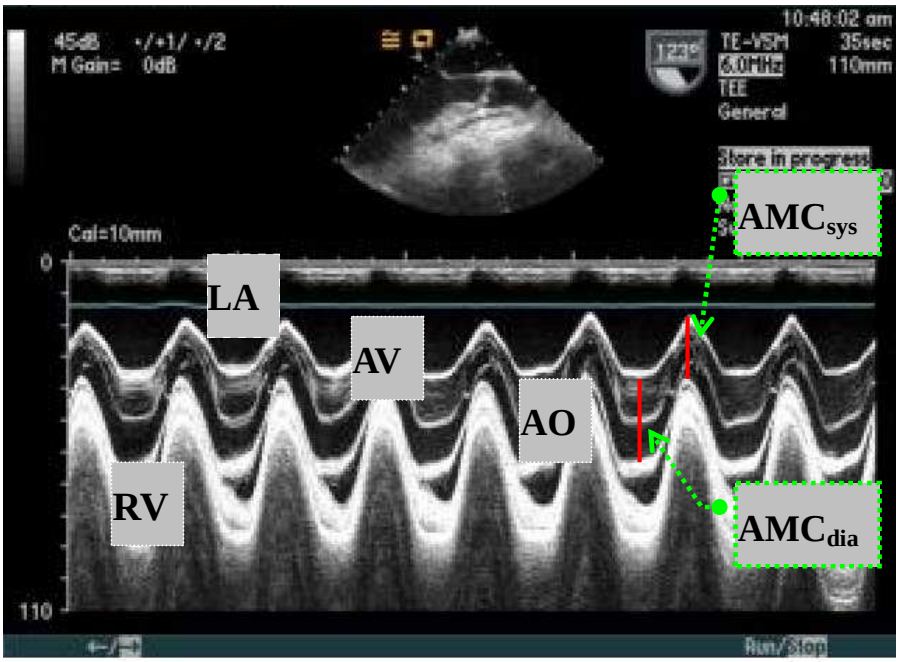

Fig. 1. Measurement of the diameter at an AMC on M-mode tracing. LA: left atrium; RV: right ventricle; AV: aortic valve (opening); AO: aortic root; AMC_{sys}: diameter of AMC at compression systole; AMC_{dia}: diameter of AMC at compression diastole.

Ⅲ. Results

1. General characteristics of the patients

In this study, 34 patients who suffered non-traumatic cardiac arrest received TEE. Twenty-four patients were males. The mean age was 56 years old. Twenty-eight patients had arrests in the field and six patients had arrests in the emergency department. The total arrest time ranged from 1 to 120 minutes, mean value is 31.2±10.3minutes. Total compression time ranged from 20 to 92 minutes, mean 44.7±13.9 minutes. With regard to the initial rhythm of cardiac arrest, ventricular fibrillation (or pulseless ventricular tachycardia) is in 4 patients, PEA in 10 patients, and asystole in 20 patients. Return of spontaneous circulation (ROSC) occurred in 8 patients after CPR. Only 4 patients were discharged with survival. Another 2 died within 24 hours. In one patient the information is not clear in age, arrest time, compression time and rhythm. The average time from patient arrival to completion of probe insertion was 8±7 min. There was no difficulty in introducing the TEE probe into the esophagus.

Characteristics of the patient were shown in Table 1. There is no significant difference in age, arrest time and CPR time between LVOT and aorta group.

Table 1. Characteristics of patients and comparison between LVOT and aorta group

Characteristics	Total	LVOT group	Aorta group	*P* value
Cases	34	14	20	
Age, yrs	56.3±11.5	55.2±13.6	57.4±10.8	.443
Sex				
Male	24	10	14	
Female	10	4	6	
Arrest time (min)	31.2±10.3	30.3±8.6	33.6±11.7	.327
CPR time (min)	44.7±13.9	46.8±17.4	43.4±14.8	.418
Rhythm				
Asystole	20	9	11	
VF/ Pulseless VT	4	1	3	
PEA	10	4	6	
Outcome				
Survival	4	2	2	
No Survival	30	12	18	

Arrest time: elapsed time from collapse to ER visit

CPR time: in-hospital CPR time (elapsed time from ER visit to ROSC or termination of CPR)

VF/ Pulseless VT: ventricular fibrillation / pulseless ventricular tachycardia

PEA: pulseless electrical activity

2. Morphologic changes of the heart

AMC was sought with TEE observation. We found the base of the heart, including the LVOT, the aortic root, and the right ventricular outflow, subjected to external chest compression. The base of the heart was compressed significantly during external chest compression. In all patients, a significant narrowing of either the LVOT (Fig. 2) or the aortic root (Fig. 3) was observed in the 135° longitudinal view of TEE. The mitral valve was closed during compression phase and open during relaxation phase. The aortic valve was open during compression phase and closed during relaxation phase.

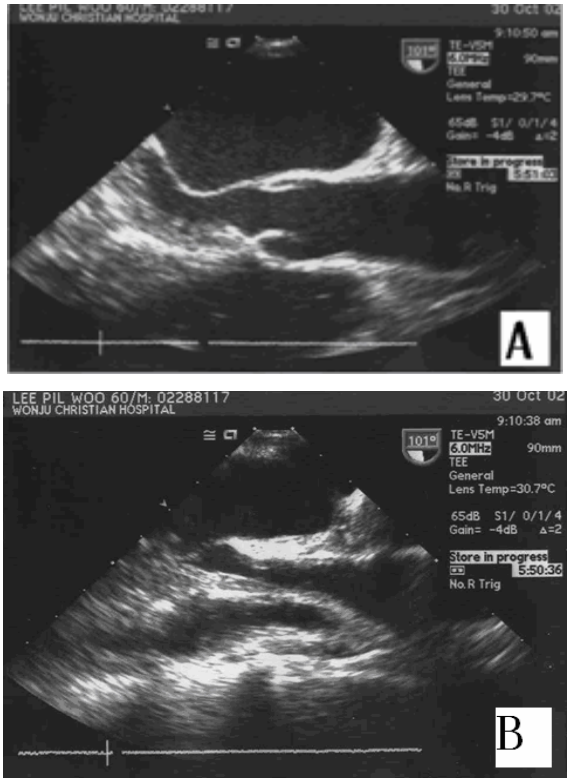

Fig. 2. AMC at LVOT. Compared with TEE image in 135° longitudinal view recorded in early phase of compression (A), both the LVOT and the aortic root became narrowing at the end of compression phase (B). The area of maximal narrow was obviously at the LVOT. The right ventricle was also deformed remarkably.

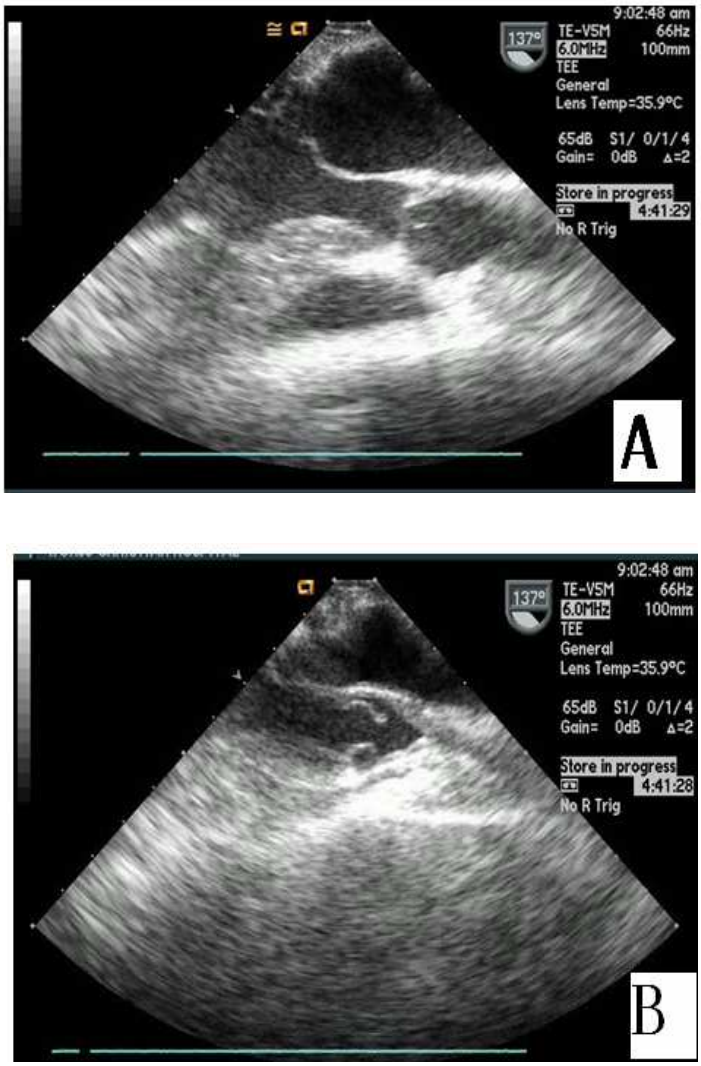

Fig. 3. AMC at aortic root. Compared with TEE image in 135° longitudinal view recorded in early phase of compression (A), the ascending aorta became narrowing at the end of compression phase (B). The area of maximal narrow was obviously at the aortic root.

3. The AMC; the location and the degree of compression

3.1 The location of the AMC

The AMC was identified at the aorta including the aortic valve in 20 patients (59%, aorta group) and at the LVOT in 14 patients (41%, LVOT group). The distance from the aortic valve to the AMC (D_{AMC-AV}) ranged from 0.09 to 3.62cm (mean: 1.71±0.34cm) in LVOT group and 0.25 to 3.67cm (mean: 1.58±0.47cm) in aorta group. The AMC was located within 2cm from the aortic valve in 79% of the patients (Fig. 4).

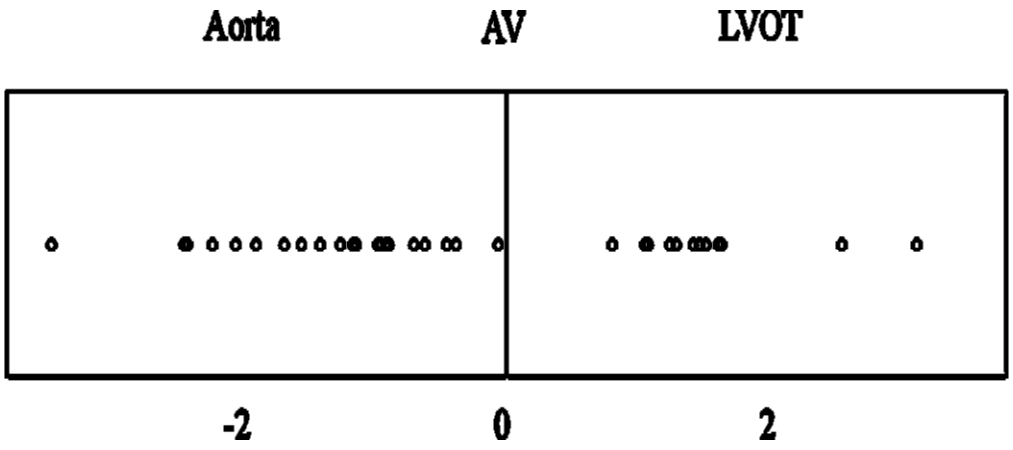

Fig. 4. Distribution and the distance of AMC from the aortic valve (cm)

3.2 changes of the diameter of the LVOT or aorta

Compression of the AMC was noted in all patients. Mean diameter of the AMC was 2.08±0.50 cm at compression diastole and 1.04±0.42 cm at the compression systole ($p<0.01$, Fig. 5). The degree of the compression at the AMC ranged from 19% to 83% (mean: 49±19%).

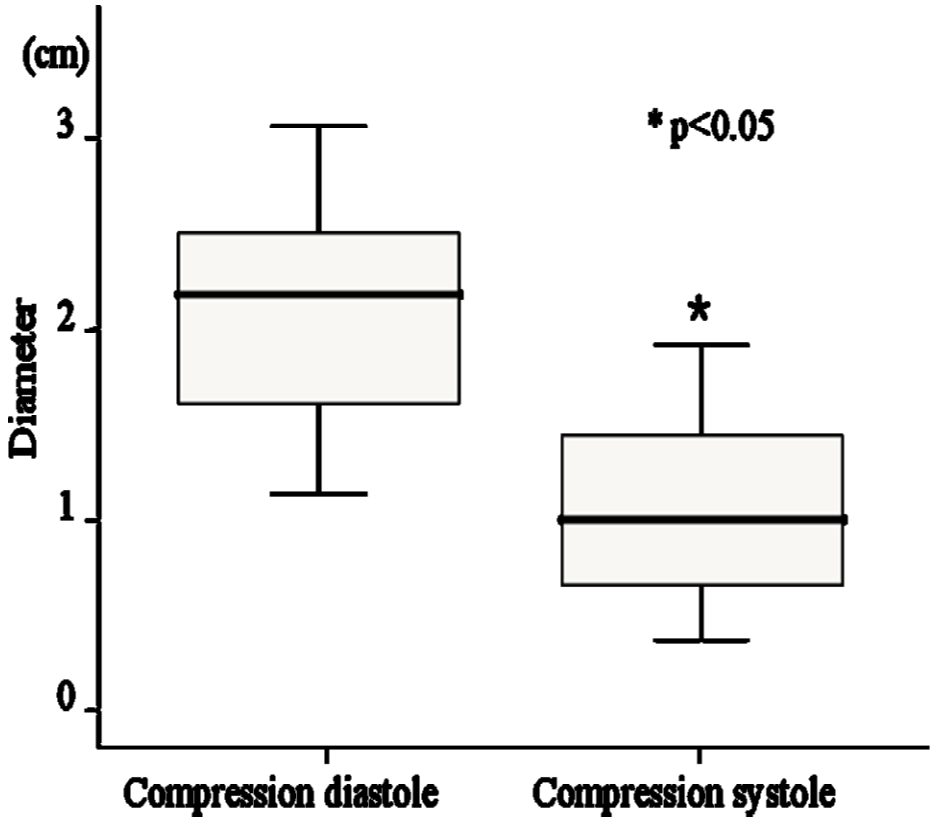

Fig. 5. Diameter of the LVOT or aorta

The degree of compression in aorta group was significantly higher than LVOT group, which suggested that higher degree of a narrowing of the outflow tract can be developed when the AMC is located above the aortic valve (Table 2).

Table 2. Comparison of dimensions of the AMC between LVOT group and aorta group

Group	AMC_{sys} (cm)	AMC_{dia} (cm)	AMC_{com} (%)	DEG_{com} (%)
LVOT	1.32±0.13	2.14±0.24	61.6±10.3	38.3±9.4
Aorta	^{a}0.86±0.09	^{b}2.01±0.19	^{c}42.8±12.1	^{d}57.6±11.5

P value: a<0.01; b<0.05; c<0.01; d<0.01

AMC_{sys}: diameter of AMC at compression systole

AMC_{dia}: diameter of AMC at compression diastole

AMC_{com} : compression of AMC= AMC_{sys}/AMC_{dia} (%)

DEG_{com} : degree of compression= $(AMC_{dia}-AMC_{sys})/AMC_{dia}$ (%)

3.3 Correlation between degree of compression and LV stroke volume

There was no correlation between degree of compression and LV stroke volume. Analysis of linear regression showed there was a positive correlation (the coefficient of correlation ***r*** =0.673) between the degree of compression and LV stroke volume in LVOT group. On the contrary, a negative correlation was shown in the aorta group (***r*** =−0.749) (Fig. 6).This finding suggested that the higher possibility of cardiac compression is present when the AMC is located at the LVOT while the lesser possibility is present when the AMC is located at the aorta.

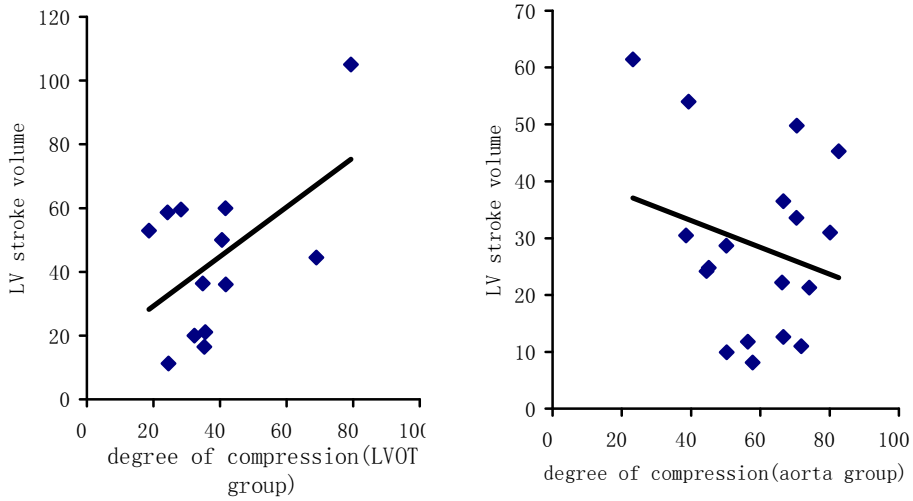

Fig. 6. Correlation between degree of compression and LV stroke volume in LVOT and aorta group.

3.4 Location of AMC and changes in the left ventricular dimensions and LV stroke volume

LV stroke volumes and LV systolic and diastolic areas were calculated in 31 patients. Unfortunately, in three patients we couldn't acquire good image plane to measure left-ventricular volume. Compared with the patients who had the AMC at the aorta, the patients having the AMC at the LVOT had higher calculated stroke volume and larger left ventricular systolic and diastolic areas (44±15 vs. 29±16 ml, p=0.047; 14±7 vs. 6±3 cm^2, p=0.00; 22±10 vs. 10±7 cm^2, p=0.00) (Fig. 7).

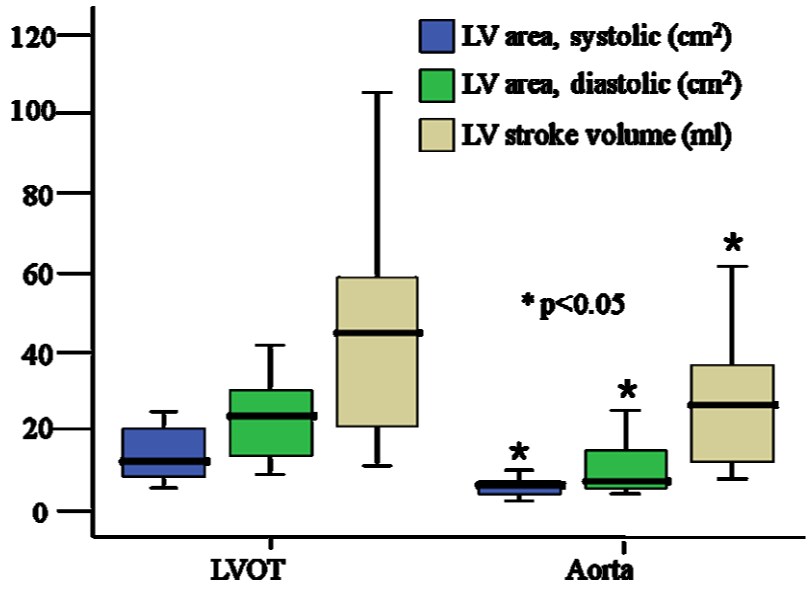

Fig. 7. LV systolic area, LV diastolic area, and LV stroke volume (LVOT vs. Aorta group)

Ⅳ. Discussion

The major findings of our study are as follows: (1) Standard cardiopulmonary resuscitation generates variable degrees of compression at the outflow tract of the left ventricle including the LVOT or the aortic root. (2) Compared with the patients who had the AMC at the aorta, the patients having the AMC at the LVOT had higher calculated stroke volume and larger left ventricular systolic and diastolic areas.

In our study, external chest compression was performed according to 2000 American Heart Association (AHA) guidelines for CPR and ECC, in which the rescuers were suggested to compress the lower half of the victim's sternum in the middle of the chest between nipples with a compression depth of 4 to 5 cm [11]. The rescuer should place the heel of the hand on the sternum in the center (middle) of the chest between the nipples and then place the heel of the second hand on top of the first so that the hands are overlapped and parallel [11].

However, same hand placement and same compression depth of chest compression during CPR may result in different changes of cardiac structures in different patients. Various configurations of thorax and morphologies of intrathoracic structures may account for these changes. Thoracic configurations are attributed to various body types, sexes, and ages of individuals [13-17]. Some pleura and pulmonary diseases can also change the relative position of heart and sternum [15, 18, 19]. For example, pulmonary atelectasis shifts heart to the affected side; pleural diseases such as pneumothorax or pleural effusion shift it to the opposite side. Various types of cardiovascular diseases may produce its own shape and position [20].

In our study, we reviewed chest radiograph or computed tomography (CT) before or after CPR for every patient. Combined with transesophageal echocardiography, we excluded all patients with significant morphologic abnormalities from thoracic or cardiovascular diseases.

It is easy to understand which part of the cardiac structure will be subjected by external

chest compression if we take a look at a cross-sectional anatomy of the thorax at the level of the position for external chest compression. Chest CT scan in a patient of our study shows that the cardiac base is located at the position of external chest compression (Fig. 8). For this reason, the compression force does not directly work on left ventricle. The axis of compression force exerted by external chest compression will traverse as following: compression point of sternum →right ventricle →aortic root (or LVOT) →left atrium →vertebral column. External compression force derived from CPR providers is indirectly transmitted to aortic root (or LVOT) passing through right ventricle. That is the reason why LVOT or aorta root is compressed during external chest compression in our study.

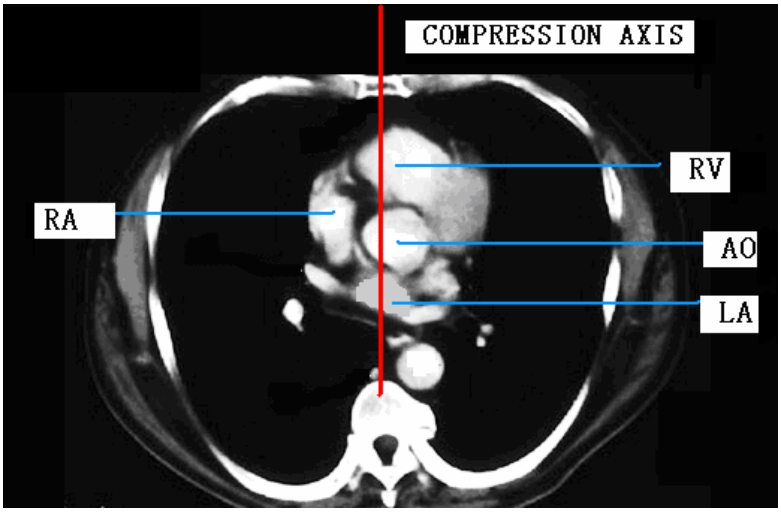

Fig. 8. Axis of compression traversing the heart. RV: right ventricle; RA: right atrium; AO: aorta; LA: left atrium

We noticed that the area of maximal compression was identified at the aorta above the aortic valve in 59% patients and at the LVOT in 41% patients. In one of the latest studies of radiological assessment of the chest in patients without cardiac arrest, Pickard et al. reported that the left ventricle was presented at the middle of the lower half of the sternum

in 3% of cases, the left atrium in 23%, the top of the left atrium in 36% and the ascending aorta in 38% of cases [21]. The differences of the results in the reports between ours and Pickard's study could be due to two factors as following: firstly, all the patients in our study were of cardiac arrest. Secondly, the means of imaging and methods of measurement are different. We used transesophageal echocardiography and just observed the AMC of the outflow tract of the left ventricle including the LVOT or the aortic root during CPR, while they used chest CT scans to investigate anatomical structures below the mid-point of the lower half of the sternum [21]. Nevertheless, we may infer that if the patients enrolled in Pickard's study were performed CPR, the most common AMC of LVOT would be at aortic root. The variation of AMC in patients with cardiac arrest probably resulted from different relative positions between standard compression point of sternum and LVOT (including aortic root) which might be due to aberrance and diseases to some extent.

There was a large variation of degree of compression at AMC in this study. The degree of compression should be due to several factors. First, the relative position between LVOT (or aortic root) and sternum must be the primary determinant. Generally speaking, aortic root is located just below the sternum. However, LVOT lies in the left lateral under the sternum. So, equipollent force of compression will produce higher degree of deformation at AMC in aortic root than LVOT. Second, the distance from the middle point of lower half of sternum to LVOT (or aortic root) may be another determinant. The longer the distance is, the lesser degree of compression is developed. In addition, the result of our study showed the lumen of LVOT was wider than aortic root, which might lead to its lower degree of deformation.

Compression of LVOT (or aortic root) will result in narrowing of its lumen which would cause increase in resistance to forward blood flow from left ventricle [22]. Compression (or narrowing) of LVOT or aortic root should be regarded as an adverse effect to the efficacy of CPR. It seems that LV stroke volume should be decreased if we increase the degree of compression at AMC of LVOT or aortic root to every patient

during CPR. However, our results showed no correlation between LV stroke volume and the degree of compression at AMC in all patients enrolled in this study. Quite differently, there was a positive correlation in LVOT group or a negative correlation in aorta group between LV stroke volume and the degree of compression at AMC. In other words, increasing the degree of compression at LVOT or aortic root may induce different effect for LV stroke volume. It is a very interesting phenomenon.

The patients in LVOT group had higher calculated LV stroke volume and larger left ventricular systolic and diastolic areas than the patients in aorta group. This is another valuable finding in our study. It has not been seen in literatures to describe these interesting phenomena. Larger changes in the calculated LV stroke volume and ventricular area suggest that the left ventricle is more compressed in the LVOT group than the aorta group. In the view of cardiac pump theory, external chest compression should produce significant volume change of the left ventricle by direct compression of the cardiac chambers. Our finding revealed that direct cardiac compression is likely to occur when the caudal portion of the sternum is compressed by external chest compression.

To a healthy person, the process of LV ejection can be described as following: During the phase of isovolumetric contraction, left ventricular pressure rises rapidly with its myocyte contraction. During the phase of rapid ejection, when the intraventricular pressures exceed the pressures within the aorta, the aortic valve is pushed to open, followed by rapid ejection of blood into the aorta from left ventricle [23]. Blood is ejected because the total energy of the blood within left ventricle exceeds the total energy of blood within the aorta. In other words, there is an energy gradient to propel blood into the aorta from left ventricle [24]. But under the condition of cardiac arrest with external chest compression, the energy that accounts for the ejection of blood from LV to aorta derives from rhythmic squeeze to left ventricle between sternum and spine because there is no intrinsic myocardial contractility [25].

In LVOT group, the position of left ventricle is closer to the axis of compression force. Besides, LVOT is not located just below the sternum and the lumen of LVOT is wider. Therefore, equipollent force of compression can result in lower degree of compression at AMC and yield higher intra-LV pressure in LVOT group. More blood will be propelled from LVOT to aorta during early phase of compression. Consequently, more LV stroke volume will be yielded in LVOT group than in aorta group. With regard to the different correlation between the degree of compression at AMC and LV stroke volume in the two groups, we may make a supposition as following: if we increase the force of compression in patients having AMC at LVOT (i.e. increase the degree of compression at AMC), the increase of blood energy in left ventricle will be higher than the increase of resistance in LVOT. On the contrary, in aorta group, the increase of resistance of aortic root will be higher than the increase of blood energy in left ventricle, even the lumen of aortic root occluded during the end phase of compression. So, increasing compression force may produce higher LV stroke volume in LVOT group and lower LV stroke volume in aorta group.

Since LV stroke volume in aorta group is lower than that of LVOT group, a question would be put forward with regard to how we could improve the outcome of CPR in the aorta group. In this study, Analysis of linear regression showed there was a negative correlation between the degree of compression and LV stroke volume in the aorta group. Then, how about to reduce compression depth? If we do this way, the degree of compression and the resistance of outflow tract will be decreased. However, the force of compression to LV will also decline. Moreover， the biggest problem is that we can hardly judge whether the location of AMC is in aorta root or LVOT before CPR. Besides, in a prospective and multi-center study of adult in-hospital and out-of-hospital cardiac resuscitations, Edelson et al reported that shallow chest compressions were associated with defibrillation failure [26]. Therefore, decreasing compression depth might not be a considerable way to improve outcome of CPR. From this we hypothesized if we shift the location of AMC from aortic root to LVOT or lower place, all problems seem to be

solved. If so, as long as we keep or increase the force of compression, LV stroke volume will be likely to be increased.

Since both other study [21] and ours indicated that most AMC during standard CPR were located at aortic root, if we lower hand placement for chest compression, most AMC will be located at LVOT and higher LV stroke volume will be acquired. So it is supposed that the currently recommended position of external chest compression during standard CPR might not be proper，hand placement of the sternum caudal to current position seems to be more rational in evidence. In other words, more patients of cardiac arrest would benefit from this position with higher efficacy of CPR.

The alternative position we suggest is located between the standard position and xiphoid process. If compression is centered too low, the xiphoid process may be driven into the left lobe of the liver, resulting in liver laceration [27]. If the compression point is shifted laterally, the costochondral junctions may be subluxed or ribs may be broken [25].

There are several limitations to this study. First, we could not assess changes in the LVOT (including aortic root) as well as in left ventricular chamber simultaneously. Simultaneous observation of LVOT (including aortic root) and left ventricular may be needed to find the relationship between changes of LVOT (including aortic root) and left ventricle. It is very hard to observe left ventricular and LVOT (including aortic root) at the same moment with TEE. Second, we could not measure intracavitary pressures in the left ventricle and/or the LVOT (including aortic root). Therefore, we could not investigate actual pressure gradient through the narrowing segment during compression or the time sequences of pressure rise in the left ventricle and the LVOT (including aortic root), which may help to clarify the mechanism that the patients in LVOT group had higher LV stroke volume than the patients in aorta group. Third, we could not measure blood flow (this is not feasible during CPR in humans), so that we could not correlate morphologic changes of the cardiac structures with blood volume transferred from the left ventricle to the aorta. Fourth, the compression force could not be measured during CPR. Higher compression force might produce higher degree of compression at AMC of LVOT

(including aortic root) and different changes of LV stroke volumes between LVOT and aorta groups. However, excessive compression forces must not be administered because residents of emergency departments perform precordial compression according to AHA guidelines. Fifth, we did not have the patients performed chest compressions at the hand placement caudal to the standard position so that we could not confirm another hand placement is more proper than the standard position. Further studies are needed to confirm this assumption.

Ⅴ. Conclusion

Standard cardiopulmonary resuscitation produces variable degrees of compression of the cardiac base and. generates variable degrees of narrowing at the outflow tract of the left ventricle including the LVOT and the aortic root, which might affect adversely to the hemodynamic effect of external chest compression. Hand placement caudal to the position currently recommended for external chest compression might decrease the narrowing of the left ventricular outflow tract and avoid compression of the cardiac base. Further studies are needed to make this assumption more confirmed.

References

1. Kouwenhoven W, Jude J, Knickerbocker G. Closed-chest cardiac massage. JAMA. 1960; 173: 1064-67.

2. Rudikoff M, Maughan W, Effron M, Freund P, Weisfeldt M. Mechanisms of blood flow during cardiopulmonary resuscitation. Circulation. 1980; 61:345-52.

3. Mackenzie GJ, Taylor SH, McDonald AK, Donald KW. Hemodynamic effects of external cardiac compression. Lancet. 1964; 1:1342-45.

4. Thomson J, Stenlund R, Rowe G. Intrathoracic pressure during closed chest cardiac massage. JAMA. 1968; 205:116-8.

5. Raessler KL, Kern KB, Sanders AB, Tacker WA, Ewy GA. Aortic and right atrial systolic pressures during cardiopulmonary resuscitation: a potential indicator of the mechanism of blood flow. Am Heart J. 1988; 115:1021-29.

6. Babbs CF, Blevins WE. Abdominal binding and counterpulsation in cardiopulmonary resuscitation. Crit Care Clin. 1986; 2:319-32.

7. Werner J, Greene H, Junko C, Cobb L. Visualization of cardiac valve motion in man during external chest compression using two-dimensional echocardiography. Circulation. 1981; 63:1417-21.

8. Rich S, Wix H, Shapiro E. Clinical assessment of heart chamber size and valve motion during cardiopulmonary resuscitation by two-dimensional echocardiography. Am Heart J. 1981; 102:368-73.

9. Deshmukh HG, Weil MH, Trevino RP, Bisera J. Echocardiographic observations during CPR: a preliminary report. Crit Care Med. 1985; 13:904-6.

10. Deshmukh HG, Weil MH, Gudiputi CV, Trevino RP, Bisera J, Rackow EC. Mechanisms of blood flow generated by precordial compression during CPR, I: studies on closed chest precordial compression. Chest. 1989; 95:1092-99.

11. Hwang SO, Lee KH, Cho JH, Yoon J, Choe KH: Changes of aortic dimensions as evidence of cardiac pump mechanism during cardiopulmonary resuscitation in humans. Resuscitation. 2001; 50: 87-93.

12. International Guidelines 2000 for CPR and ECC. Emergency Cardiac Care Committee and Subcommittee, American Heart Association. Part 3. Adult Basic Life Support . JAMA 2000; 102[Suppl I]: I-22 - I-59.

13. Bellemare F, Jeanneret A, Couture J. Sex differences in thoracic dimensions and configuration. Am J Respir Crit Care Med. 2003;168:305–12.

14. Bellemare JF, Cordeau MP, Leblanc P, Bellemare F. Thoracic dimensions at maximum lung inflation in normal subjects and in patients with obstructive and restrictive lung diseases. Chest 2001; 119:376–86.

15. Wilson TA, Rehder K, Krayer S, Hoffman EA, Whitney CG, Rodarte JR. Geometry and respiratory displacement of human ribs. J Appl Physiol. 1987; 62:1872–77.

16. Dansereau J, Stokes IAF. Measurements of the three-dimensional shape of the rib cage. J Biomech. 1988; 21:893–901.

17. Abiru H, Ashizawa K, Hashmi R, Hayashi K. Normal radiographic anatomy of thoracic structures: analysis of 1000 chest radiographs in Japanese population. Br J Radiol.

2005 May;78:398-404.

18. Knisely BL, Broderick LS, Kuhlman JE. MR imaging of the pleura and chest wall. Magn Reson Imaging Clin N Am. 2000; 8:125-41.

19. Cassart M, Gevenois PA, Estenne M. Rib cage dimensions in hyperinflated patients with severe chronic obstructive pulmonary disease. Am J Respir Crit Care Med. 1996; 154:800–5.

20. Jeudy J, Waite S, White CS. Nontraumatic thoracic emergencies. Radiol Clin North Am. 2006 Mar;44:273-93.

21. Pickard A, Darby M, Soar J. Radiological assessment of the adult chest: implications for chest compressions. Resuscitation. 2006;71(3):387-90.

22. Hrovatin E, Piazza R, Pavan D, Mimo R, Macor F, Dall'Aglio V, et al. Dynamic left ventricular outflow tract obstruction in the setting of acute anterior myocardial infarction: A serious and potentially fatal complication? Echocardiography. 2002; 19: 449-55.

23. Emrys K. Mechanical events and the pressure–volume relationships. Anaes &Inten Care Med.2006;7: 313-16.

24. Romero T, Covell J, Friedman WF. A comparison of pressure-volume relations of the fetal, newborn, and adult heart. Am J Physiol. 1972; 222:1285-90.

25. Babbs CF**.** Technique of chest compression in CPR. in: Joseph P Ornato**;** Mary Ann Peberdy(ed). Cardiopulmonary resuscitation. Humana Press. Totowa, New Jersey, 2005;162-166.

26. Edelson DP, Abella BS, Kramer-Johansen J, Wik L, Myklebust H, Barry AM, et al.

Effects of compression depth and pre-shock pauses predict defibrillation failure during cardiac arrest. Resuscitation. 2006; 71:137-45.

27. Vitello JM, Hartung R. Splenic laceration secondary to closed chest massage: successful recognition and management—case report. J Trauma. 1991; 31:426-28.

Abstract in Korean

표준 심폐소생술에 의한 좌심실 유출로의 협착
– 경식도 심초음파에 의한 관찰 –

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목적: 심폐소생술 중 흉부압박이 흉곽내 구조에 미치는 영향에 대한 연구는 거의 없다. 현재의 흉부압박 위치는 과학적 연구의 근거없이 정하여졌으며, 현재의 압박위치가 심폐소생술에 의한 혈역학적 효과를 최대화 시킬 수 있는지 알려지지 않았다. 이 연구는 현재 여러 지침에서 권장되고 있는 흉부압박이 심장에 미치는 영향을 확인함으로써, 현재 권장되고 있는 흉부압박의 위치를 평가하는 것이다.

연구대상 및 방법: 34 명의 비외상성 심정지환자(남자 24 명, 평균연령 56 세)가 연구에 포함되었다. 흉곽의 변형 또는 흉곽내에서 심장의 위치에 영향을 줄 정도의 심각한 심장질환이 있는 환자는 연구에서 제외되었다. 심폐소생술이 시작되면 경식도 심초음파를 사용하여 심장을 관찰하였다. 135 도 종단면 관찰과 횡단면 관찰을 통하여 좌심실, 좌심실 유출로, 대동맥 부위를 관찰하였다. 심폐소생술이 종료될 때까지 경식도 심초음파 관찰을 계속하였으며, 심초음파 영상은 녹화되어 저장되었다. 저장된 영상을 사용하여 분석을 하였다. 흉부압박에 의하여 가장 변형이 심하게 발생하는 부위를

확인하여, area of maximal compression (AMC)으로 정하였다. AMC 가 관찰되는 부위에서 압박기와 이완기의 직경을 측정하였으며, 이를 사용하여 압박률을 계산하였다. Area-length 법으로 좌심실 1 회 박출량, 압박기와 이완기의 좌심실 면적을 계산하였다. 연구대상은 AMC 의 위치에 따라 좌심실유출로 군과 대동맥 군으로 구분하였다.

결과: AMC 의 위치는 20 예(59%)에서 대동맥판 상방(대동맥 군)에 있었으며, 14 예(41%)는 대동맥판 하방(좌심실유출로 군)에 있었다. AMC 가 대동맥판을 중심으로 2 cm 이내에 있었던 경우가 79%로서 흉부압박에 의하여 가장 많이 압박되는 심장구조는 대동맥판 부근임을 알 수 있었다. AMC 에 위치한 구조물은 모두 압박되었으며, 압박률은 19% - 83% (평균: 49±19%)였다. 좌심실유출로 군이 대동맥군에 비하여 계산된 좌심실 1 회 심박출양, 압박기 및 이완기 좌심실 면적이 컷다 (44±15 vs 29±16 ml, p=0.047; 14±7 vs 6±3 cm^2, p=0.00; 22±10 vs 10±7 cm2, p=0.00).

결론: 현재 지침에서 권장하고 있는 위치에서의 흉부압박은 심장의 저부를 압박하여 좌심실 유출로 또는 상행 대동맥의 일시적이고 반복적인 협착을 초래한다. 심장의 저부의 압박을 줄이려면, 현재 권장되는 위치보다 미골부 방향으로 흉부압박의 위치를 옮겨야 할 것이다.

Keywords: cardiopulmonary resuscitation; external chest compressions; left ventricular outflow tract