タイトル	Mechanophysiological Analysis of Active Compression-Decompression Cardiac Massage Technique Using the Physical Heart Simulator
作成者（著者）	Goto, Ai / Kambayashi, Ryuichi / Chiba, Koki / Nunoi, Yoshio / Izumi-Nakaseko, Hiroko / Kawai, Shinichi / Takei, Yoshinori / Matsumoto, Akio / Lurie, Keith G. / Sugiyama, Atsushi
公開者	The Medical Society of Toho University
発行日	2022.12.01
ISSN	21891990
掲載情報	Toho Journal of Medicine. 8(4). p.154-160.
資料種別	学術雑誌論文
内容記述	Original Article
著者版フラグ	publisher
JaLCDOI	info:doi/10.14994/tohojmed.2022-015
メタデータのＵＲＬ	https://mylibrary.toho-u.ac.jp/webopac/TD22294587

Original Article

Mechanophysiological Analysis of Active Compression-Decompression Cardiac Massage Technique Using the Physical Heart Simulator

Ai Goto[1)] Ryuichi Kambayashi[1)] Koki Chiba[2)]
Yoshio Nunoi[3)] Hiroko Izumi-Nakaseko[1)] Shinichi Kawai[4)]
Yoshinori Takei[1)] Akio Matsumoto[5)] Keith G. Lurie[6)]
and Atsushi Sugiyama[1, 4, 5)]*

1) Department of Pharmacology, Faculty of Medicine, Toho University, Tokyo, Japan
2) Department of Traditional Medicine, Faculty of Medicine, Toho University, Tokyo, Japan
3) Department of Cardiovascular Surgery, Faculty of Medicine, Toho University, Tokyo, Japan
4) Department of Inflammation & Pain Control Research, Faculty of Medicine, Toho University, Tokyo, Japan
5) Department of Aging Pharmacology, Faculty of Medicine, Toho University, Tokyo, Japan
6) Department of Emergency Medicine, University of Minnesota Medical School, Minneapolis, USA

ABSTRACT

Introduction: Active compression-decompression cardiac massage (ACD-CM) technique can achieve higher cardiac output than standard cardiac massage (S-CM). We explored unknown mechanisms of ACD-CM leading to its efficacy other than the enhancement of venous return and cardiac filling.

Methods: We assessed the effects of ACD-CM on pressure waves of the aorta, left ventricle, and left atrium compared with those of S-CM using the physical heart simulator. Cardiac massage was performed at a rate of 105 strokes/min with a depth of 5 cm.

Results: Cardiac output was 1.72 times greater with ACD-CM than with S-CM. The mitral valve closed just before the end of compression, and the aortic valve shut around the end of decompression with both maneuvers, indicating occurrence of mitral and aortic valve regurgitation. The opening duration of the mitral valve during compression and that of the aortic valve during decompression were significantly shorter with ACD-CM than with S-CM.

Conclusions: ACD-CM results in smaller time window of regurgitation for mitral and aortic valves, possibly contributing to the augmentation of cardiac output.

Toho J Med 8 (4): 154–160, 2022

KEYWORDS: active compression-decompression, cardiac massage, heart simulator, mechanophysiology, pressure waveform

---

Ai Goto and Ryuichi Kambayashi contributed equally to this work.

*Corresponding Author: Atsushi Sugiyama, 5-21-16 Omori-nishi, Ota-ku, Tokyo 143-8540, Japan, tel: +81–3–3762–4151 (Ext 2361)
e-mail: atsushi.sugiyama@med.toho-u.ac.jp

DOI: 10.14994/tohojmed.2022-015
Received June 29, 2022: Accepted Sept. 2, 2022
Toho Journal of Medicine 8 (4), Dec. 1, 2022.
ISSN 2189–1990, CODEN: TJMOA2

Introduction

While active compression-decompression cardiac massage (ACD-CM) was shown to provide better hemodynamics under cardiac arrest when compared with standard cardiac massage (S-CM) in an animal model,[1)] the efficacy of ACD-CM in clinical studies remains controversial.[2–4)] ACD-CM increases the negative intrathoracic pressure during the decompression phase, augmenting venous return and cardiac filling, which may increase in cardiac output during the compression phase.[1–3)] During the compression phase of both S-CM and ACD-CM, the blood is thought to be propelled from the heart to the rest of the body by both cardiac compression and a rise in thoracic pressure.[5, 6)] Indeed, these combined mechanisms squeeze the blood out of the ventricles into the systemic and pulmonary circulations, force the mitral and tricuspid valves to close, and facilitate opening of the aortic and pulmonary valves.[5, 6)] However, previous studies using the transesophageal echocardiogram during cardiac massage have demonstrated that regurgitation flow through the mitral valve may occur during the chest compression phase of S-CM.[7, 8)] More importantly, it remains unknown how the addition of active decompression maneuver alters the state of the mitral and aortic valves. To estimate the state of mitral and aortic valves in a cardiac cycle during cardiac massage, we developed a mechanical model of cardiopulmonary circulation, enabling us to measure the pressure waves of aorta (AoP), left ventricle (LVP), and left atrium (LAP) during S-CM and ACD-CM as schematically shown in Fig. 1. Especially, we adopted a physical heart simulator (Dock-kun™, Sakai Sangyo Co., Ltd., Shizuoka, Japan) (Fig. 1), which was originally developed as a teaching device for a trainee to learn and master high-quality manual cardiac massage.[9)] Using the model, we compared hemodynamic variables associated with the performance of S-CM and ACD-CM, enabling us to better understand the underlying mechanisms of ACD-CM, which would also be of interest from its reproducibility and animal ethics point of view.

Methods

Experimental system

The physical heart simulator (Dock-kun™) consists of the inlet tube (=left atrium), inlet valve (=mitral valve), BB-type air pump (=left ventricle), outlet valve (=aortic valve), and outlet tube (=aorta) (Fig. 1A).[9)] The experimental system consisted of a custom-made, motor-driven, cardiac massage device; the physical heart simulator; two buckets as arterial and pulmonary vascular beds; pressure transducers to measure the aortic, left ventricular, and left atrial pressures; connecting tubes of 10 mm in diameter; and water as the blood (Fig. 1B). The base of physical heart simulator and the ends of inlet and outlet tubes were positioned at the same height. The tip of outlet tube was not dipped into water of the bucket to prevent a gradual increase of the afterload. The bottom face of air pump was fixed to the base metal panel using Velcro tape (Fig. 1C). No additional modification was performed for S-CM (Fig. 1C left), whereas top face of the pump was fixed to the compression panel with Velcro tape for ACD-CM (Fig. 1C right), enabling the piston to actively decompress the pump.

Experimental protocol

The physical heart simulator was driven by the cardiac massage device at a rate of 105 strokes/min and a stroke amplitude of 5 cm[10, 11)] for both S-CM and ACD-CM with the compression panel (Fig. 1C). The AoP, LVP, and LAP during S-CM or ACD-CM were simultaneously measured and recorded with polygraph system (RM-6000; Nihon Kohden Corporation, Tokyo, Japan) and analyzed with Power Lab and Lab Chart 7 (AD Instruments Ltd., Dunedin, New Zealand). The cardiac output was measured by the amount of water pumped out into the bucket. The experiment was performed for 1 min for S-CM or ACD-CM using five physical heart simulators (n = 5 for each).

Definition of phases in a cardiac cycle

The cardiac massage consists of two phases: compression and decompression. The compression phase was defined as the time period when the piston moved downward, whereas the decompression phase was done as that when the piston moved upward (Fig. 2 top). The ventricle recoiled up to the middle with S-CM (Fig. 1C left), whereas it expanded to as it was with ACD-CM (Fig. 1C right), which can reflect the difference in mechanics between S-CM and ACD-CM in the *in situ* heart. Thus, the magnitude of the decompression was equal to that of the expansion of the BB-type air pump with ACD-CM, but the compression panel was not elevated upward from the initial position in this study.

Statistical analysis

Data are presented as mean±SD (n = 5). Statistical differences in the measured values between S-CM and ACD-CM were assessed by paired *t*-test. A p-value <0.05 was considered to be significant.

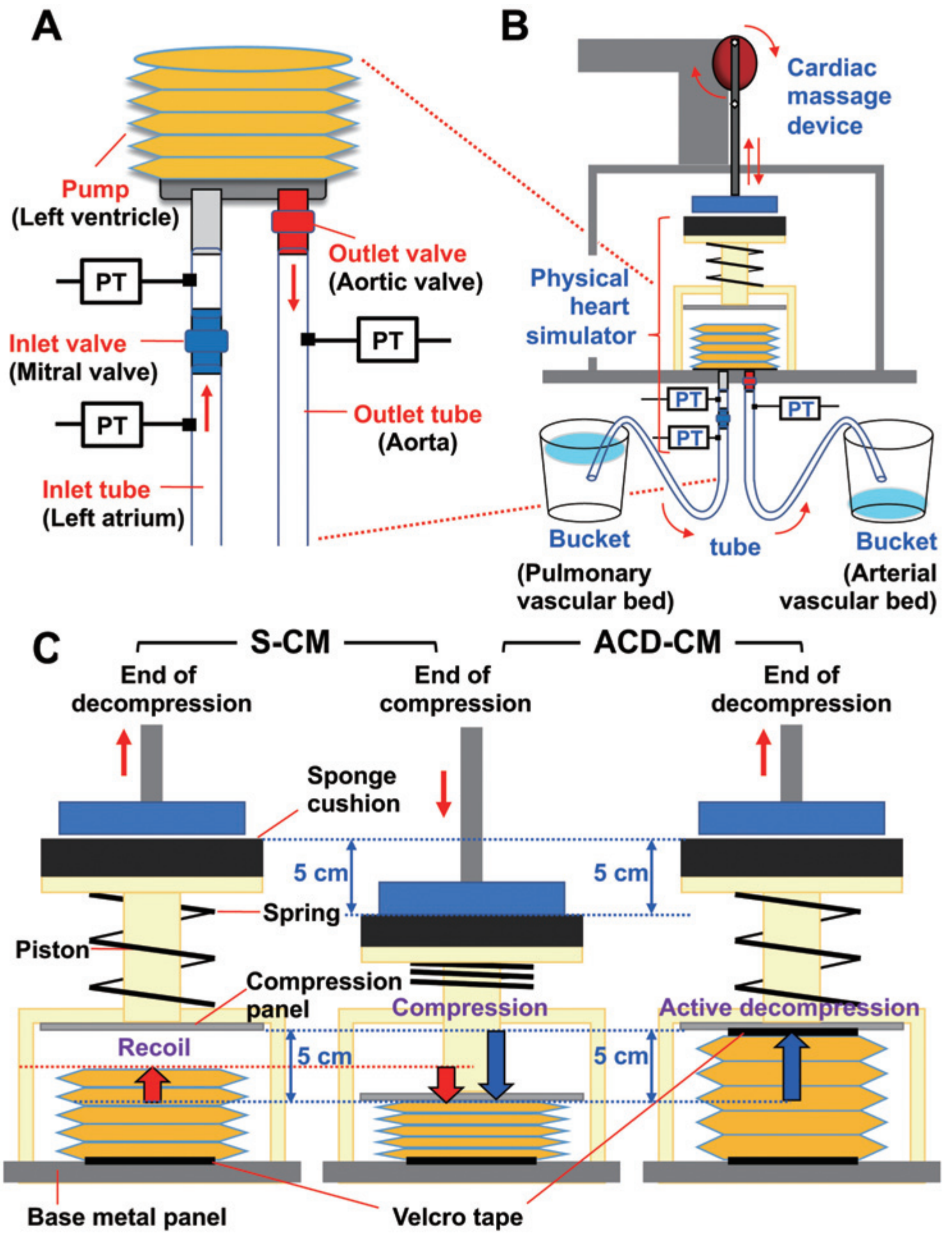

Fig. 1
Schematic representation of the experimental system. (A) Schema of the physical heart simulator, consisting of the inlet tube (left atrium), inlet valve (=mitral valve), BB-type air pump (=left ventricle), outlet valve (=aortic valve), outlet tube (aorta), and pressure transducer (PT) to measure the left atrial, left ventricular, and aortic pressures. A plastic, circular monoleaflet, check valve with a diameter of 14 mm was used for the mitral and aortic valves. (B) Schema of the whole experimental system, including motor-driven automatic cardiac massage device; the physical heart simulator and two buckets as arterial and pulmonary vascular beds. (C) Schema of standard cardiac massage (S-CM, left-center) and active compression-decompression cardiac massage (ACD-CM, center-right). Bottom face of the pump was fixed to the base metal panel with Velcro tape for both S-CM and ACD-CM, whereas top face of the pump was fixed to the compression panel with Velcro tape only for ACD-CM, enabling the piston to actively decompress the pump.

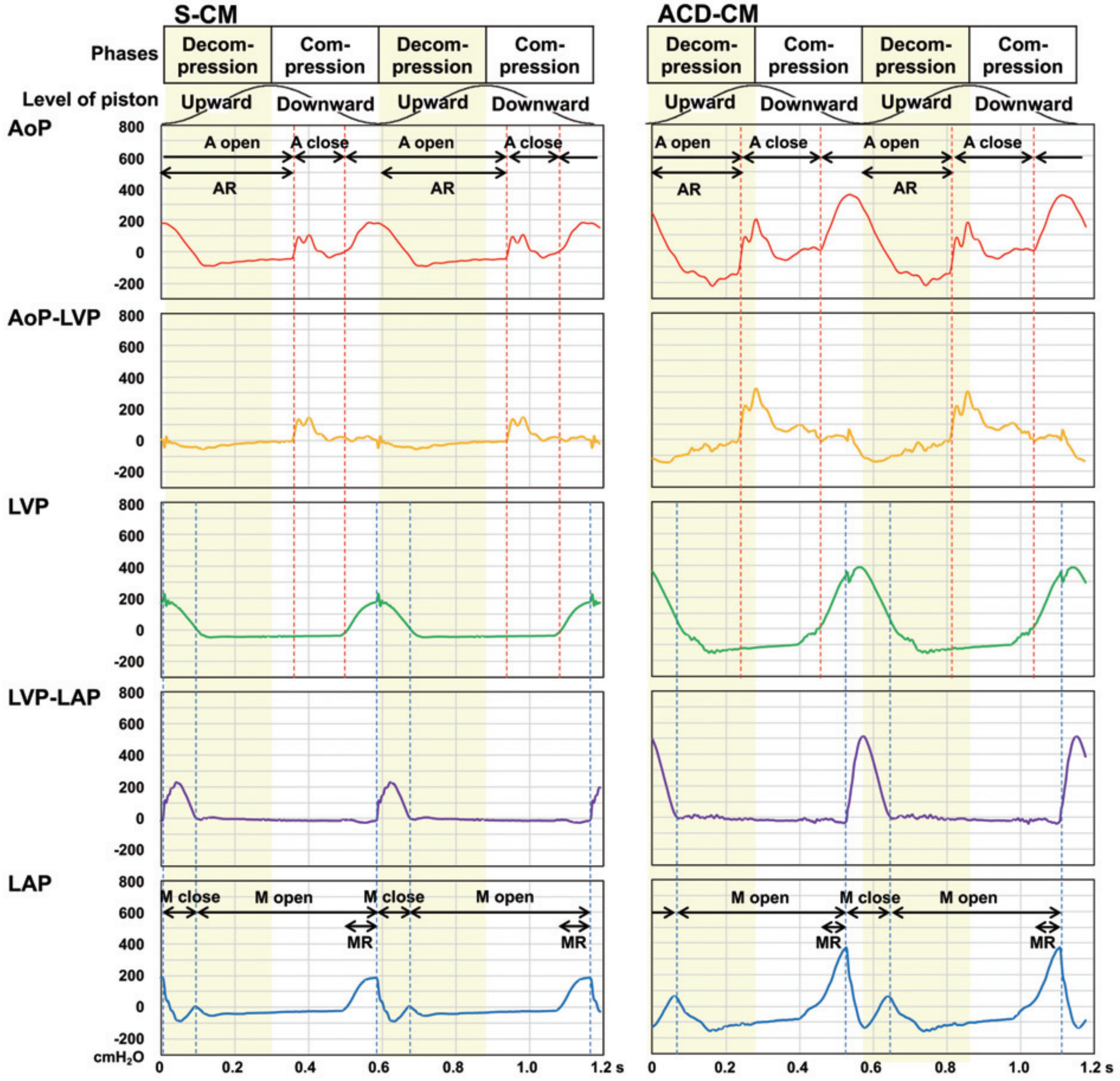

Fig. 2

Typical tracings of the aortic (AoP), left ventricular (LVP), and left atrial pressures (LAP) along with their pressure differences (AoP-LVP and LVP-LAP) during standard cardiac massage (S-CM, left) and active compression-decompression cardiac massage (ACD-CM, right). The cardiac massage consists of two phases: compression and decompression. The former was defined as the time period when the piston moves downward, whereas the latter was done as that when the piston moves upward. Note that in S-CM the aortic valve (A) closed at the early phase of compression before the compression panel started to press the pump and that the mitral valve (M) closed at the end of compression. Also, note that in ACD-CM the aortic valve closed at the late phase of decompression and that the mitral valve closed at the late phase of compression. In addition, AoP-LVP was below the zero-pressure line during AR during the decompression phase, which was greater in ACD-CM than in S-CM, reflecting the magnitude of suction. MR, mitral valve regurgitation; AR, aortic valve regurgitation.

Results

Effects on cardiac output and stroke volume

Table 1 summarizes cardiac output and stroke volume during S-CM and ACD-CM. The cardiac output as well as stroke volume were 1.72 times greater with ACD-CM than with S-CM, indicating that active decompression can significantly increase the net forward flow during cardiac massage.

Table 1 Cardiac output, stroke volume, and time windows for the state of mitral and aortic valves

			S-CM	ACD-CM
Cardiac output (L/min)			2.36 ± 0.13	4.05 ± 0.19[#]
Stroke volume (mL)			22.5 ± 1.3	38.6 ± 1.8[#]
Cardiac cycle		Total duration (ms)	580 ± 0	580 ± 0
	Mitral valve	Open duration (ms)	486 ± 5	458 ± 4[#]
		Closed duration (ms)	94 ± 4	122 ± 4[#]
	Aortic valve	Open duration (ms)	419 ± 11	363 ± 7[#]
		Closed duration (ms)	161 ± 11	218 ± 7[#]
Compression phase		Total duration (ms)	290 ± 0	290 ± 0
	Mitral valve	Open duration (ms)	286 ± 1	254 ± 3[#]
		Closed duration (ms)	4 ± 1	35 ± 3[#]
	Aortic valve	Open duration (ms)	129 ± 11	113 ± 7
		Closed duration (ms)	161 ± 11	176 ± 6
Decompression phase		Total duration (ms)	290 ± 0	290 ± 0
	Mitral valve	Open duration (ms)	200 ± 4	203 ± 3
		Closed duration (ms)	90 ± 4	87 ± 3
	Aortic valve	Open duration (ms)	290 ± 0	249 ± 5[#]
		Closed duration (ms)	0 ± 0	41 ± 5[#]

Data are presented as mean ± SD (n = 5). S-CM, standard cardiac massage; ACD-CM, active compression-decompression cardiac massage. [#]$p < 0.01$ vs. S-CM

Effects on the pressure waveform

Fig. 2 left shows typical tracings of the AoP, LVP, and LAP from the simulation model along with their pressure differences between AoP and LVP (AoP-LVP) and between LVP and LAP (LVP-LAP) during S-CM, whereas Fig. 2 right shows those during ACD-CM. Table 1 summarizes the time windows of the state of mitral and aortic valves, namely, the open and closed durations, during S-CM and ACD-CM.

The pressure differences between AoP and LVP as well as between LVP and LAP were calculated to assess the state of mitral and aortic valve function, respectively. When there was actually no difference in the pressure waveforms between the two consecutive chambers, the valve between the chambers was judged to be open. Meanwhile, when differences in the pressure were observed, the valve was considered to be closed. Using these criteria, the state of mitral and aortic valves was analyzed.

The closed durations of mitral and aortic valves in a cardiac cycle were 1.30 and 1.35 times greater with ACD-CM than with S-CM, respectively (Table 1). The opening duration of mitral valve in the compression phase and that of aortic valve in the decompression phase were 0.89 and 0.86 times shorter with ACD-CM than with S-CM, respectively (Table 1), indicating that active decompression can significantly shorten the duration of time window of regurgitation for mitral and aortic valves.

Discussion

We studied the effects of ACD-CM on the AoP, LVP, and LAP compared with those of S-CM using the physical heart simulator to explore unknown mechanisms of ACD-CM contributing to its effectiveness. We confirmed that an active decompression procedure can significantly increase the net forward flow during cardiac massage. Also, we found that S-CM as well as ACD-CM can induce sustained regurgitation through the mitral and aortic valves during compression and decompression phases, respectively, unlike those observed in the spontaneously beating *in situ* heart.

The regurgitation of mitral and aortic valves induced characteristic changes in the pressure waveforms in each chamber. As shown in Fig. 2, the mitral valve closed at the end of compression with S-CM and at the late phase of compression with ACD-CM, whereas the aortic valve shut at the early phase of compression with S-CM and at the late phase of decompression with ACD-CM. Namely, when the ventricle was started to be compressed, the mitral valve remained open, resulting in a lack of isovolumetric contraction phase for S-CM and ACD-CM (Fig. 2). On the other hand, when the ventricle was started to recoil with S-CM or expand with ACD-CM, the aortic valve was left open, resulting in a lack of isovolumetric relaxation phase for S-CM and ACD-CM (Fig. 2). Thus, the resulting regurgitant flow may limit the effectiveness of cardiac massage. A similar situation would be expected in the *in situ* heart during cardiac massage, since the heart with ventricular fibrillation is flaccid, lacks spontaneous contraction, and is just passively compressed and decompressed, making the valves unable to exert physiological function.

Although the increase of cardiac output during ACD-CM would largely depend on active decompression-associated increase of venous return and cardiac filling,[2, 3] which might be largely associated with the suction effect to increase the preload of the ventricle, the regurgitant flow occurring during decompression phase through the aortic valve may have attenuated its efficiency. To estimate the intracardiac blood flow, we focused on the kinetics of mitral and aortic valves as well as the developed pressure in each chamber during cardiac massage. As summarized in Table 1, the opening duration of mitral valve in the compression phase and that of aortic valve in the decompression phase were significantly shorter with ACD-CM than with S-CM, indicating that active decompression can shorten the time window of regurgitation for mitral and aortic valves. These effects may partly contribute to the increase in cardiac output with ACD-CM. However, since the valves could not fully exert their physiological function in this study as well as the *in situ* heart with ventricular fibrillation, the massage effect of ACD-CM would be more influenced by increased preload than by the contribution of reduced valve regurgitation time.

It would deserve a comment on why ACD-CM can shorten the time window of regurgitation for mitral and aortic valves. As shown in Fig. 2, ACD-CM exerted greater upstroke and downstroke velocities of the LVP than S-CM, suggesting that larger "dynamic pressure" can be developed with ACD-CM, which may help the mitral valve close in the compression phase and the aortic valve shut in decompression phase.

This study has several limitations. First, the results can be highly dependent on the mechanical properties of the valves of the heart simulator, which may not necessarily mimic the properties of biological ones. Second, tap water was used to simulate the blood.[9] As the water may not necessarily reflect the viscosity of the blood, it would produce less great "dynamic pressure" on the valves than the blood, which may partly explain longer opening time of the mitral valve during the compression phase and of the aortic valve during the decompression phase than those expected from our previous study of the *in situ* canine hearts.[12] In addition, the presence of mitral regurgitation during the compression phase may have made closure time of the aortic valve longer. Third, the preload as well as afterload to the ventricle can alter the ventricular motion kinetics during cardiac massage. We set the physical heart simulator and buckets about the same height, mimicking the supine position, in which cardiac massage is usually performed. Experiments are now ongoing to assess how the changes of preload and/or afterload may alter the effectiveness of cardiac massage by changing the height of respective buckets. Fourth, the increase in aortic pressure at the beginning of "aortic valve close" in Fig. 2 would be associated with a kind of dicrotic notch that appears when the aortic valve closes.[13] The notch was greater in amplitude and longer in duration than that observed in the physiological condition since the tube was too hard to simulate the compliance of aorta. In addition, the aortic pressure became negative during the latter phase of decompression, since the aortic valve was open, and the ventricle recoiled up to the middle with S-CM and expanded to as it was with ACD-CM, which may also partly contribute to the larger dicrotic notch. Fifth, the LAP markedly increased in the same way as the LVP and AoP at the late phase of compression, since the inlet valve developed mitral regurgitation and the tube was too hard to mimic the atrium. The tube needs to be improved to better reflect anatomical and physiological characteristics of the atrium.

In conclusion, mechanophysiological analyses using the physical heart simulator indicate that the active decompression procedure of cardiac massage can shorten the time window of mitral and aortic valve regurgitation, contributing to the effective augmentation of cardiac output.

Acknowledgement/Funding source: The authors thank Professors Takeshiro Fujii and Takaaki Kawaguchi for their productive advice, Dr. Yoko Amagase for final editing and correction, and Mrs. Yuri Ichikawa for her technical assistance. This study was supported in part by the Project Research Grant of Toho University School of Medicine Grant Number 21-10 (to A.G.).

Authors' contribution: A.G., R.K., and A.S. conceived and designed the research. A.G., R.K., K.C., and Y.N. conducted the experiments. A.G., R.K., and A.S. analyzed the data and wrote the manuscript. All authors read, discussed, and approved the manuscript.

Conflicts of interest: None declared.

References

1) Lindner KH, Pfenninger EG, Lurie KG, Schürmann W, Lindner IM, Ahnefeld FW. Effects of active compression-decompression resuscitation on myocardial and cerebral blood flow in pigs. Circulation. 1993; 88: 1254-63.

2) Lurie KG, Shultz JJ, Callaham ML, Schwab TM, Gisch T, Rector T, et al. Evaluation of active compression-decompression CPR in victims of out-of-hospital cardiac arrest. JAMA. 1994; 271: 1405-11.

3) Cohen TJ, Tucker KJ, Lurie KG, Redberg RF, Dutton JP, Dwyer KA, et al. Active compression-decompression. A new method of cardiopulmonary resuscitation. JAMA. 1992; 267: 2916-23.

4) Lafuente-Lafuente C, Melero-Bascones M. Active chest compression-decompression for cardiopulmonary resuscitation. Cochrane Database Syst Rev. 2013; CD002751.

5) Kühn C, Juchems R, Frese W. Evidence for the ‘cardiac pump theory’ in cardiopulmonary resuscitation in man by transesophageal echocardiography. Resuscitation. 1991; 22: 275-82.

6) Ewy GA. The mechanism of blood flow during chest compressions for cardiac arrest is probably influenced by the patient’s chest configuration. Acute Med Surg. 2018; 5: 236-40.

7) Redberg RF, Tucker KJ, Cohen TJ, Dutton JP, Callaham ML, Schiller NB. Physiology of blood flow during cardiopulmonary resuscitation. A transesophageal echocardiographic study. Circulation. 1993; 88: 534-42.

8) Ma MH, Hwang JJ, Lai LP, Wang SM, Huang GT, Shyu KG, et al. Transesophageal echocardiographic assessment of mitral valve position and pulmonary venous flow during cardiopulmonary resuscitation in humans. Circulation. 1995; 92: 854-61.

9) Nakamura Y, Miyamoto T, Yamada M, Goto A, Hagiwara-Nagasawa M, Izumi-Nakaseko H, et al. Development of an educational equipment “Dock-kun” to help school children master high-quality cardiac massage. Toho J Med. 2017; 3: 146-51.

10) Sugiyama A, Duval S, Nakamura Y, Yoshihara K, Yannopoulos D. Impedance threshold device combined with high-quality cardiopulmonary resuscitation improves survival with favorable neurological function after witnessed out-of-hospital cardiac arrest. Circ J. 2016; 80: 2124-32.

11) Duval S, Pepe PE, Aufderheide TP, Goodloe JM, Debaty G, Labarère J, et al. Optimal combination of compression rate and depth during cardiopulmonary resuscitation for functionally favorable survival. JAMA Cardiol. 2019; 4: 900-8.

12) Goto A, Kambayashi R, Izumi-Nakaseko H, Takei Y, Kawai S, Matsumoto A, et al. Magnitude of mitral valve closure plays a pivotal role in enhancing the forward blood flow during cardiac massage in dogs with ventricular fibrillation. J Vet Med Sci. 2022; 84: 877-80.

13) Politi MT, Ghigo A, Fernández JM, Khelifa I, Gaudric J, Fullana JM, et al. The dicrotic notch analyzed by a numerical model. Comput Biol Med. 2016; 72: 54-64.