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Experimental paper

Transthoracic impedance for the monitoring of quality of manual chest compression during cardiopulmonary resuscitation☆

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ABSTRACT

Objective: The quality of cardiopulmonary resuscitation (CPR), especially adequate compression depth, is associated with return of spontaneous circulation (ROSC) and is therefore recommended to be measured routinely. In the current study, we investigated the relationship between changes of transthoracic impedance (TTI) measured through the defibrillation electrodes, chest compression depth and coronary perfusion pressure (CPP) in a porcine model of cardiac arrest.
Methods: In 14 male pigs weighing between 28 and 34 kg, ventricular fibrillation (VF) was electrically induced and untreated for 6 min. Animals were randomized to either optimal or suboptimal chest compression group. Optimal depth of manual compression in 7 pigs was defined as a decrease of 25% (50 mm) in anterior posterior diameter of the chest, while suboptimal compression was defined as 70% of the optimal depth (35 mm). After 2 min of chest compression, defibrillation was attempted with a 120-J rectilinear biphasic shock.
Results: There were no differences in baseline measurements between groups. All animals had ROSC after optimal compressions; this contrasted with suboptimal compressions, after which only 2 of the animals had ROSC (100% vs. 28.57%, $p = 0.021$). The correlation coefficient was 0.89 between TTI amplitude and compression depth ($p < 0.001$), 0.83 between TTI amplitude and CPP ($p < 0.001$).
Conclusion: Amplitude change of TTI was correlated with compression depth and CPP in this porcine model of cardiac arrest. The TTI measured from defibrillator electrodes, therefore has the potential to serve as an indicator to monitor the quality of chest compression and estimate CPP during CPR.

1. Introduction

Out-of-hospital cardiac arrest (OHCA) is a major public health problem all over the world. Each year, an estimated 330,000 victims in US, 350,000 in Europe and 544,000 in China experience OHCA.[1–3] Although survival rate varies considerably, the overall functional survival is less than 10% among patients successfully resuscitated.[4,5] Successful cardiopulmonary resuscitation (CPR) from cardiac arrest requires the delivery of high quality chest compression, including adequate depth, rate and full chest recoil between compressions.[6–9] The 2010 guidelines, therefore, emphasized the importance of performing optimal chest compression.[10–12] The role of chest compression is to restore, although minimally, the flow of oxygenated blood to vital organs, and thereby minimise ischemic injury, especially in the brain and heart.

However, the quality of manual chest compression is often not optimal for both trained emergency medical professionals and lay rescuers resuscitation attempts.[13–15] In a recent multicenter clinical study, the median compression depth performed by emergency medical services was around 37 mm and nearly half of cases had a mean value less than 38 mm during OHCA resuscitation.[6] Rescuer's fatigue, which is a main reason for the decreased quality of CPR, can lead to inadequate compression depth.[16] Although rescuers may not be aware, the fatigue is present and the compression depth decay became evident after only 90 s of chest compression, even though the compression rate does not change.[17,18]

The use of mechanical chest compression devices can potentially overcome some of the limitations of manual compression

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and deliver consistent chest compression with adequate depth and rate. However, there is still no high-quality evidence that these devices may improve clinical outcome.[19] When the use of a mechanical device is not possible, it might be important to have a real-time CPR feedback that prompts the rescuer to perform high quality CPR.[20–24] Quality variables such as the rate and depth of chest compression measured through force transducer and acceleration sensors have been employed to monitor the quality of chest compression and demonstrated their utility in improving the effectiveness of CPR.[25–27] Yet, these techniques for monitoring the quality of chest compression require additional sensors and devices. Impedance cardiography, which had been initially developed to monitor stroke volume and cardiac output noninvasively, has been used to evaluate the performance of chest compression by analyzing the transthoracic impedance (TTI) waveforms recorded through the two defibrillation electrodes.[28–30] With the increasingly greater availability of automated external defibrillators (AEDs), more comprehensive analysis of the TTI waveforms is feasible since current generation of defibrillators can record ECG together with TTI waveform synchronously. However, the relationships between TTI, compression depth and CPP have not been established yet. This prompted the present effort to monitor the quality of chest compressions by utilizing TTI signals recorded during CPR. We hypothesized that the amplitude change of TTI was correlated with compression depth and CPP, and therefore had the potential to serve as an alternative indicator for the monitoring of the quality of chest compression and estimate CPP.

2. Materials and methods

2.1. Study design

This prospective, randomized, single center controlled experiment was designed to investigate the relationship between TTI, compression depth and CPP. Experiments were performed in an established swine model of electrically induced cardiac arrest. All animals received humane care and the experiments were conducted after approval of the Animal Ethics Committee, Sun Yat-sen University (Guangzhou, China). The protocol was performed according to institutional guidelines.

2.2. Animal preparation

Fourteen male domestic pigs weighing between 28 and 34 kg were fasted overnight except for free access to water. Anesthesia was initiated by intramuscular injection of ketamine (20 mg/kg), completed by ear vein injection of sodium pentobarbital with a dose of 30 mg/kg and maintained by additional doses of 8 mg/kg. A cuffed endotracheal tube was advanced into the trachea and animals were mechanically ventilated with a volume-controlled ventilator (model Avs III, T Bird, CA, USA), with a tidal volume of 15 mL/kg, and fraction of inspired oxygen (F_{IO2}) of 0.21.

For the measurement of aortic pressure, a 6F fluid-filled angiographic catheter (model 070, Cordis Corporation, Miami Lakes, FL, USA) was advanced from the surgically exposed right femoral artery into the thoracic aorta. For measurements of right atrial pressure and pulmonary arterial pressure, a 7-Fr, pentalumen, thermodilution-tipped catheter (model 131HF7, Swan-Ganz TD, Edwards Lifesciences, CA, USA) was advanced from the surgically exposed right femoral vein and flow-directed into the pulmonary artery. For inducing VF, a 5-Fr pacing catheter (Cordis Corporation, Miami Lakes, FL, USA) was advanced from the right jugular vein into the right ventricle until an endocardial electrocardiogram confirmed endocardial contact via a multi parameter monitor (78352C, HP Corporation, Palo Alto, CA, USA). The hard gel type of adult defibrillation/pacing pads (stat-padz, Zoll Medical Corporation, Chelmsford, MA, USA) were applied with an anterior to lateral placement. TTI waveform was recorded through a user designed circuit which was parallelly connected with the defibrillator using a sinusoid-wave excitation current of 2 mA and 30 kHz across the defibrillation pads. An accelerometer-based handheld CPR device (CPR-D-padz, Zoll Medical Corporation, Chelmsford, MA, USA) was placed on the surface of the animal's chest just above the heart and underneath the rescuer's hands during chest compression. Cardiac output was measured by the thermo-dilution technique with the aid of a cardiac output computer (Baxter COM-2TM, Edwards Division, Santa Ana, CA, USA) after a bolus injection into the right atrium of 5 mL cold saline solution, which had been maintained at a temperature between 0 °C and 2 °C. Aortic blood gases were measured with the aid of a handheld blood analyzer (model CG4 + Cartridge, Abbott i-STAT System, Princeton, NJ, USA). Respiratory frequency was adjusted to maintain $Petco_2$ between 35 mmHg and 40 mmHg before inducing cardiac arrest and when mechanical ventilation was resumed after resuscitation.

2.3. Experimental procedures

After collection of baseline data, cardiac arrest was induced with a 2 mA alternating current delivered to the endocardium of the right ventricle. After VF had been successfully induced, mechanical ventilation was discontinued and cardiac arrest was untreated for a total of 6 min.

Animals were then randomized to one of the following two groups: optimal CPR, where manual chest compression was performed by an emergency medical doctor at a rate of 100 per min and a depth comparable to 25% of the anterior posterior diameter of the chest, which represented approximately 50 mm; suboptimal CPR, where chest compression was operated by another emergency medical doctor at the same rate but the chest was compressed to 70% of the depth of optimal group, which was equivalent to approximately 17% of the anterior posterior diameter of 35 mm.[9,16] The suboptimal depth represented a value corresponding to the average suboptimal depth of compression recorded during out-of-hospital CPR.[6,9,31] During chest compression, the rescuer was blinded from the monitored compression depth and TTI values but with acknowledgment of whether his compressions were below or above 38 mm. The animal's chest wall was allowed to completely recoil in both groups. The animals were manually ventilated with a bag-valve device during CPR. Chest compression was synchronized to provide a compression/ventilation ratio of 30:2 with equal compression–relaxation intervals. No epinephrine or other vasopressor agents were administered.

After 2 min of compression in each group, a defibrillation was attempted with a single 120-J rectilinear biphasic shock (M-Series, Zoll medical corporation, Chelmsford, MA, USA). Chest compression was immediately resumed followed by ECG rhythm analysis within 5 s until confirmation of spontaneous circulation. The defibrillation attempt was regarded as successful when the electrical shock converted VF to an organized rhythm with a mean aortic pressure of $\geq$60 mmHg for an interval $\geq$10 s.[31] If spontaneous circulation was not restored, chest compressions were continued for another 2 min, after which defibrillation was attempted with another single 120 J shock. This sequence was repeated for a maximum of 5 cycles. Catheters were removed after 1 h of post resuscitation monitoring, and the animals were euthanized by injection of 150 mg/kg intravenous pentobarbital.

2.4. Measurements

Baseline measurements were obtained, including ECG, the aortic pressure, right atrial pressure, TTI, cardiac output and blood

Table 1
Baseline measurements.

Measurements	Optimal compression group ($n=7$)	Suboptimal compression group ($n=7$)	p Value
Body weight (kg)	31.71 ± 2.25	32.00 ± 2.29	0.82
Thoracic anterior posterior diameter (cm)	22.27 ± 0.56	22.07 ± 0.73	0.58
Thoracic transverse diameters (cm)	17.63 ± 0.57	17.90 ± 0.38	0.32
Heart rates (beats/min)	112.00 ± 12.70	114.29 ± 13.94	0.75
Mean aortic pressure (mmHg)	103.86 ± 21.67	105.14 ± 14.01	0.90
Respiratory rate (respiration/min)	14.86 ± 2.79	15.71 ± 2.14	0.53
Core temperature (°C)	37.91 ± 0.41	37.96 ± 0.50	0.86
Cardiac output (L/min)	4.63 ± 0.56	4.49 ± 1.11	0.67
$PaCo_2$ (mmHg)	36.61 ± 1.85	36.36 ± 1.26	0.77
PaO_2 (mmHg)	81.29 ± 8.54	83.00 ± 9.49	0.73
Arterial lactate (mmol/L)	1.76 ± 0.30	1.86 ± 0.34	0.57

gas analysis. The ECG, pressure measurements, acceleration signals and TTI waveform were continuously measured and recorded through a data acquisition system supported by Windaq hardware/software (Dataq Instruments Inc., Akron, OH, USA) at a sample rate of 300 Hz. The CPP was digitally computed from the differences in time-coincident diastolic aortic and right atrial pressures. The compression rate and depth was calculated from the double integration of acceleration signals recorded from accelerometer by matlab7.0 (The MathWorks, Inc., Natick, MA, USA). The TTI amplitude change during each compression was defined as the difference between the wave crest and trough of the impedance waveform measured through the data acquisition system. Compression depth, amplitude change of TTI and CPP were obtained from each identifiable compression and reported within a 5 s average window.

2.5. Statistical analyses

Data are presented as mean ± standard deviation (SD). Differences in TTI, compression depth and CPP between the two groups were analyzed by two-tailed Student's t-test for independent samples test. A two-tailed Fisher's exact test was performed for rate comparison. The relationship between TTI, compression depth and CPP were tested with Pearson correlation coefficients and analyzed with linear regression. A $p < 0.05$ was regarded as statistically significant.

3. Results

There were no differences in baseline measurements between the two groups before inducing cardiac arrest (Table 1). Fig. 1 illustrates the ECG, TTI, acceleration signal and the reconstructed compression depth signal recorded during the experiment.

No difference in compression rate (97.43 ± 6.41 vs. 93.93 ± 9.77 compressions per minute, $p = 0.45$) and ventilation rate (5.71 ± 0.45 vs. 5.86 ± 0.35 ventilations per minute, $p = 0.55$) were obtained between groups. The measured compression depth was ranged from 18.6 to 38.5 mm in the suboptimal group and between 35.2 to 57.0 mm in the optimal group. As shown in Fig. 2, the compression depth was significantly higher in the optimal group during the first 2 min of chest compression (44.16 ± 4.61 vs. 29.06 ± 4.90 mm, $p < 0.001$). As anticipated, CPP was significantly higher in the optimal group compared with suboptimal group (26.53 ± 7.58 vs. 13.09 ± 2.41 mmHg, $p < 0.001$). Amplitude change of TTI also demonstrated a great difference between groups (1.45 ± 0.37 vs. 0.47 ± 0.12 ohm, $p < 0.001$). The Pearson correlation coefficient was 0.89 between TTI amplitude and compression depth ($p < 0.001$), 0.83 between TTI amplitude and CPP ($p < 0.001$), 0.73 between compression depth and CPP ($p < 0.001$). The linear regression results are shown in Fig. 3.

The defibrillation success rate for the first shock was higher in the optimal group than in the suboptimal group, but a statistical significance was not achieved (85.71% vs. 28.57%, $p = 0.103$). All of the 7 animals had ROSC after optimal compressions, while only 2

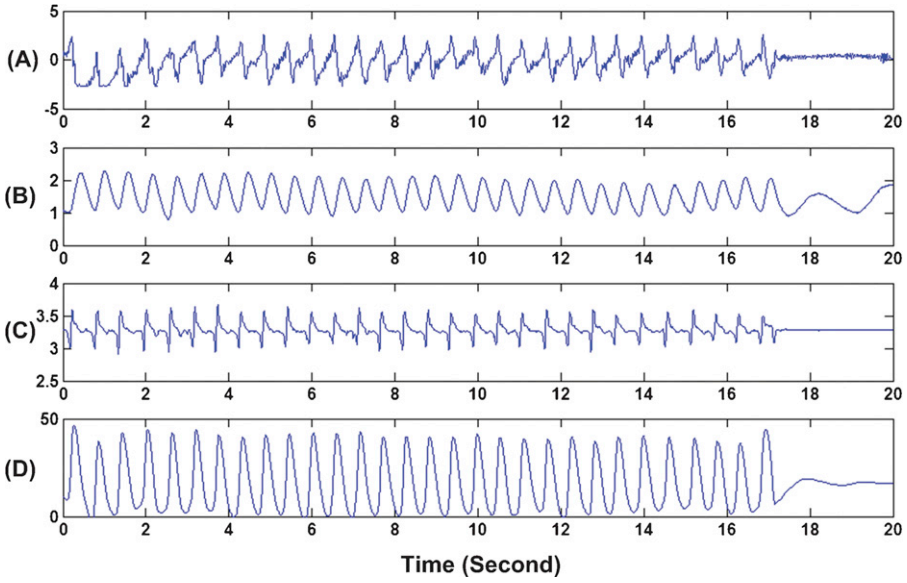

Fig. 1. ECG (A), transthoracic impedance (TTI) (B), acceleration signal (C) and the reconstructed compression depth signal (D) in the experiment.

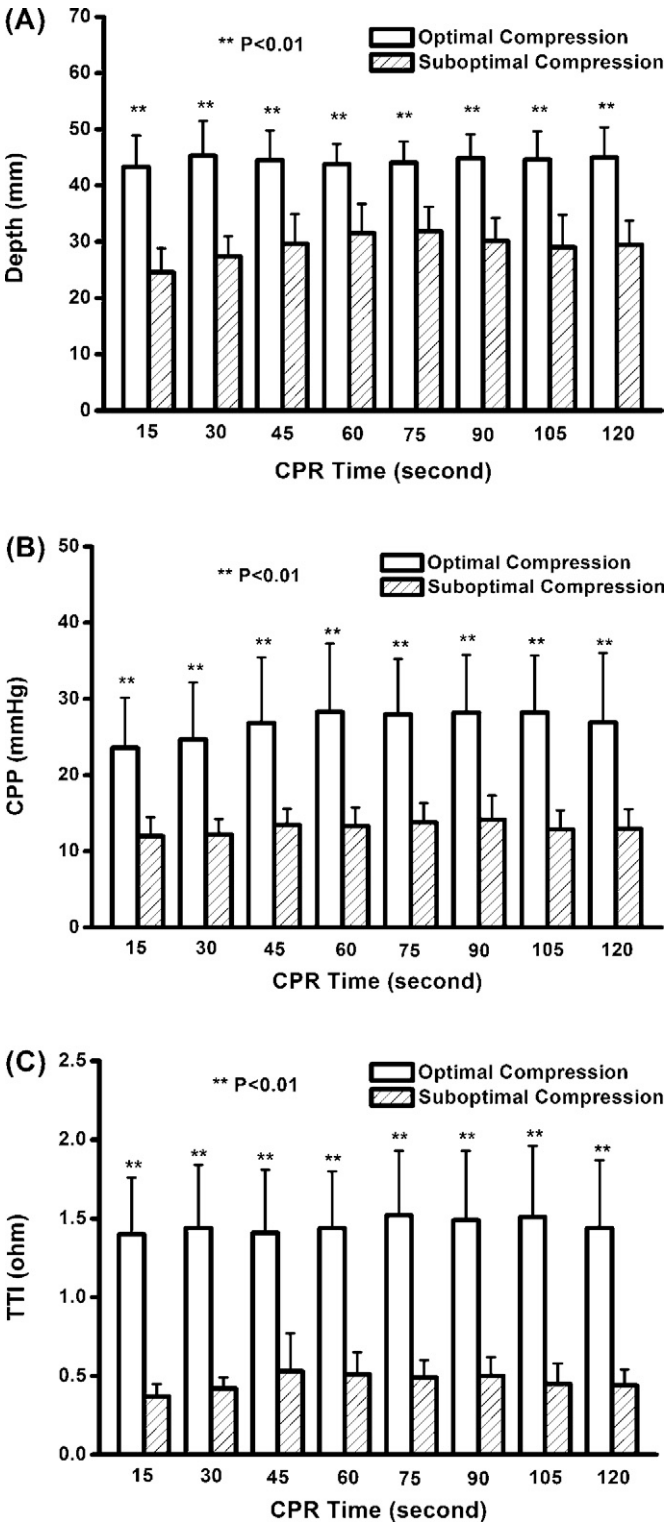

Fig. 2. Compression depth (A), Coronary perfusion pressure (CPP) (B) and transthoracic impedance (TTI) (C) during first 2 min of cardiopulmonary resuscitation (CPR).

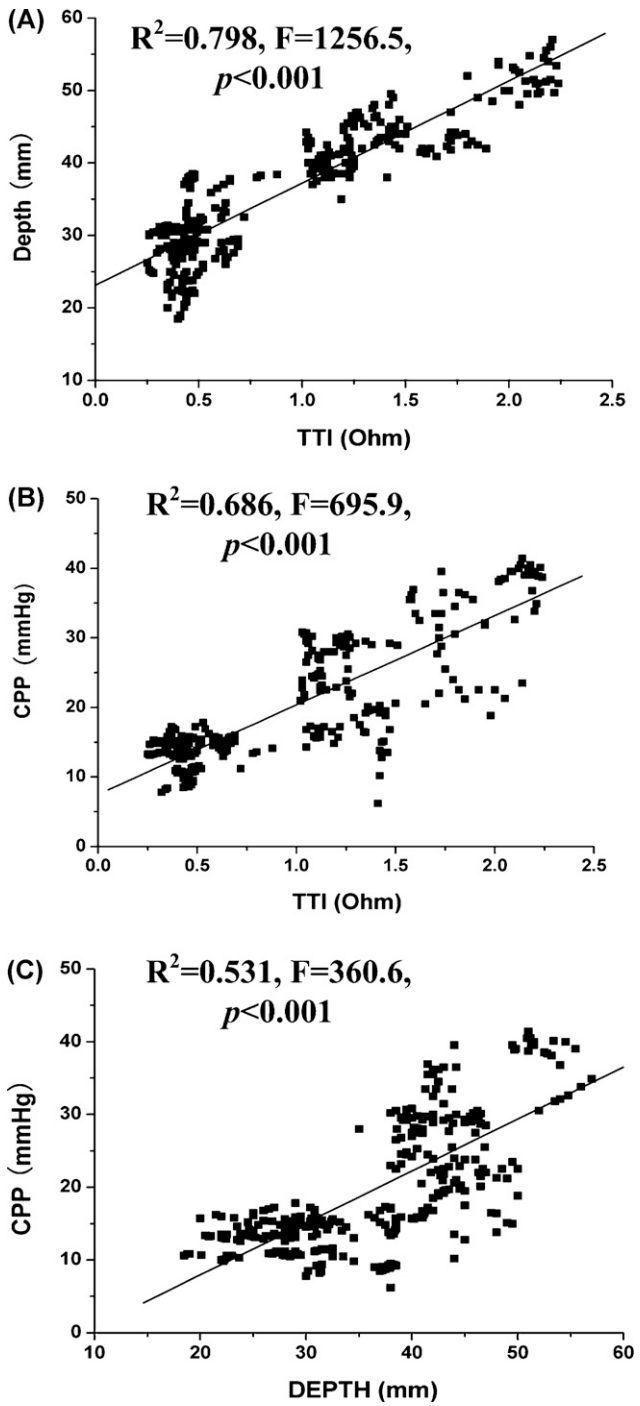

Fig. 3. Linear regression results between transthoracic impedance (TTI) and compression depth (A), TTI and coronary perfusion pressure (CPP) (B), compression depth with CPP (C).

of the animals had ROSC with suboptimal compressions (100% vs. 28.57%, $p = 0.021$). No rib fractures were observed in both groups.

4. Discussion

The present study has demonstrated that change of TTI has the capability to monitor the quality of chest compression in a porcine model of cardiac arrest. The main finding was that TTI measured from the two defibrillation pads was strongly correlated with the depth of chest compression ($r = 0.89$), and with CPP ($r = 0.83$).

Because more than half of the patients receive less than the recommended compression depth of at least 50 mm and because the quality of CPR prior to defibrillation directly affects resuscitation outcome, many efforts have been tried to monitor the quality of chest compression during CPR.[6,11,32] Relevant chest compression parameters, including compression depth and rate, could be monitored and recorded in real-time during CPR by using different transducers and sensors. For example, Baubin[25]

measured the compression and decompression forces with two transducers to determine chest displacement and deriving the depth of compressions. Aase[26] estimated compression depth with the use of accelerometers. The feedback based audiovisual CPR techniques have demonstrated their utility in improving the quality of chest compressions provided by hospital care professionals.[22,23] However, the compression force and mechanical displacement measurement required additional sensors and devices and might not always correlate with the actual physiological changes produced by CPR.

Another available method, i.e. the TTI measurement can also be used in the field of resuscitation. Earlier investigations based on TTI have been studied this approach as a non-invasive technique for estimating stroke volume and cardiac output.[28,29] Losert et al.[33] reported that TTI changes measured via defibrillation pads correlated with ventilation rate and addressed the potential of using impedance measurements for quantifying tidal volume. TTI was also used to distinguish correct tracheal intubation from erroneous esophageal tube placement.[34] In a recent study, Roberts et al.[35] demonstrated that changes of TTI obtained via defibrillation pads can accurately detect and guide ventilations in stable, mechanically ventilated children, corresponding to rescue ventilations recommended during CPR.

Johnston et al.[36] first proved that impedance recorded through two ECG/defibrillation pads placed in an anterior-apical position was a potential haemodynamic sensor for AEDs. Pellis et al.[37] also demonstrated the utility of TTI for the classification of respiratory arrest and cardiac arrest by detecting pulse pressure during cardiorespiratory arrest. Furthermore, useful information was extracted from TTI signals about the circulatory state of cardiac arrest patient and could serve as an aid to give circulation-related feedback to rescuers and thereby improve CPR quality.[38] In order to evaluate the performance of CPR during cardiac arrest, Stecher et al.[30] measured the changes of TTI produced by chest compressions and ventilations in 122 events of OHCA and concluded that the core CPR values can be measured from TTI signals assessed by a conventional defibrillator. Aramendi et al.[39] demonstrated that the instantaneous compression rate can be accurately estimated from TTI and the estimated rate can be used to suppress CPR artefact. However, these studies only evaluated the quality of chest compression in terms of compression rate and compression fraction.

In this study, we intended to monitor the quality of chest compression by assessing compression depth and found a strong correlation between changes of TTI, compression depth and CPP. Bivariate correlations demonstrated that TTI, compression depth and CPP were highly correlated with each other, but TTI and compression depth had the highest Pearson correlation coefficient of 0.89. On the other hand, the correlation coefficient between TTI and CPP ($r = 0.83$) was greater than the coefficient between compression depth and CPP ($r = 0.73$). Linear regression analysis showed that F statistic was 1257 and 696, R square was 0.798 and 0.686 when TTI served as dependent variable for the prediction of compression depth and CPP. This compared to an F statistic of 361 and R square of 0.531 when compression depth was used to predict CPP. The positive correlation between compression depth and TTI can be explained by the deformation of the thorax during the external compressions since TTI is measured through the electrical resistance changes in the thorax. The correlation between TTI and CPP may be explained by the intra-thoracic pressure changes caused by the deflection of compression and the consequent movement of blood from the ventricle to the vascular system. The strong correlations between TTI, compression depth and CPP indicate the TTI measurement not only reflect the quality of chest compression, but also associated with physiological changes produced by chest compressions. Additionally, this method does not require any additional sensor and equipment, so that it can be readily usable in current AEDs and in professional ECG monitoring/defibrillation devices.

We recognize limitations in the present report. First, the TTI was measured between the two defilation electrodes, so the effects of electrode size, location, skin contact, and conduction together with variable chest configurations, may produce vectoral changes in the impedance. Secondly, there were no rib fractures observed in the current study; so the effect of rib fractures on chest compliance and TTI measurement was not evaluated. Furthermore, even though the TTI was promising for the monitoring of the quality of chest compression in this animal study, whether TTI can provide a self-referenced measurement of depth that is subject-independent is still need to be investigated.

5. Conclusion

Amplitude change of TTI was strongly correlated with chest compression depth and CPP in this porcine model of cardiac arrest. The TTI measured from the two defibrillator pads, therefore has the potential to serve as an alternative indicator for monitoring the quality of chest compression and estimate CPP during CPR.

Conflicts of interest statement

None.

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References

1. Nichol G, Thomas E, Callaway CW, et al. Regional variation in out-of-hospital cardiac arrest incidence and outcome. JAMA 2008;300:1423–31.
2. Atwood C, Eisenberg MS, Herlitz J, Rea TD. Incidence of EMS-treated out-of-hospital cardiac arrest in Europe. Resuscitation 2005;67:75–80.
3. Hua W, Zhang LF, Wu YF, et al. Incidence of sudden cardiac death in China: analysis of 4 regional populations. J Am Coll Cardiol 2009;54:1110–8.
4. Bobrow BJ, Spaite DW, Berg RA, et al. Chest compression–only CPR by lay rescuers and survival from out-of-hospital cardiac arrest. JAMA 2010;304:1447–54.
5. Sasson C, Rogers MA, Dahl J, Kellermann AL. Predictors of survival from out-of-hospital cardiac arrest: a systematic review and meta-analysis. Circ Cardiovasc Qual Outcomes 2010;3:63–81.
6. Stiell IG, Brown SP, Christenson J, et al. What is the role of chest compression depth during out-of-hospital cardiac arrest resuscitation? Crit Care Med 2012;40:1–7.
7. Fried DA, Leary M, Smith DA, et al. The prevalence of chest compression leaning during in-hospital cardiopulmonary resuscitation. Resuscitation 2011;82:1019–24.
8. Brown TB, Dias JA, Saini D, et al. Relationship between knowledge of cardiopulmonary resuscitation guidelines and performance. Resuscitation 2006;69:253–61.
9. Wik L, Kramer-Johansen J, Myklebust H, et al. Quality of cardiopulmonary resuscitation during out-of-hospital cardiac arrest. JAMA 2005;293:299–304.
10. Berg RA, Hemphill R, Abella BS, et al. Part 5: adult basic life support: 2010 American heart association guidelines for cardiopulmonary resuscitation and emergency cardiovascular care. Circulation 2010;122:S685–705.
11. Soar J, Mancini ME, Bhanji F, et al. Part 12: Education, implementation, and teams: 2010 international consensus on cardiopulmonary resuscitation and emergency cardiovascular care science with treatment recommendations. Resuscitation 2010;81:e288–330.
12. Soar J, Monsieurs KG, Ballance JH, et al. European resuscitation council guidelines for resuscitation 2010. Section 9. Principles of education in resuscitation. Resuscitation 2010;81:1434–44.
13. Van Hoeyweghen RJ, Bossaert LL, Mullie A, et al. Quality and efficiency of bystander CPR. Belgian Cerebral Resuscitation Study Group. Resuscitation 1993;26:47–52.

14. Wik L, Steen PA, Bircher NG. Quality of bystander cardiopulmonary resuscitation influences outcome after prehospital cardiac arrest. Resuscitation 1994;28:195–203.
15. Gallagher EJ, Lombardi G, Gennis P. Effectiveness of bystander cardiopulmonary resuscitation and survival following out-of-hospital cardiac arrest. JAMA 1995;274:1922–5.
16. Hightower D, Thomas SH, Stone CK, Dunn K, March JA. Decay in quality of closed-chest compressions over time. Ann Emerg Med 1995;26:300–3.
17. Heidenreich JW, Berg RA, Higdon TA, Ewy GA, Kern KB, Sanders AB. Rescuer fatigue: standard versus continuous chest-compression cardiopulmonary resuscitation. Acad Emerg Med 2006;13:1020–6.
18. Sugerman NT, Edelson DP, Leary M, et al. Rescuer fatigue during actual in-hospital cardiopulmonary resuscitation with audiovisual feedback: a prospective multicenter study. Resuscitation 2009;80:981–4.
19. Perkins GD, Woollard M, Cooke MW, et al. Prehospital randomised assessment of a mechanical compression device in cardiac arrest (PaRAMeDIC) trial protocol. Scand J Trauma Resusc Emerg Med 2010;18:58.
20. Kramer-Johansen J, Myklebust H, Wik L, et al. Quality of out-of-hospital cardiopulmonary resuscitation with real time automated feedback: a prospective interventional study. Resuscitation 2006;71:283–92.
21. Peberdy MA, Silver A, Ornato JP. Effect of caregiver gender, age, and feedback prompts on chest compression rate and depth. Resuscitation 2009;80:1169–74.
22. Pozner CN, Almozlino A, Elmer J, Poole S, McNamara D, Barash D. Cardiopulmonary resuscitation feedback improves the quality of chest compression provided by hospital health care professionals. Am J Emerg Med 2011;29(6):618–25.
23. Hostler D, Everson-Stewart S, Rea TD, et al. Effect of real-time feedback during cardiopulmonary resuscitation outside hospital: prospective, cluster-randomised trial. BMJ 2011;4:d512.
24. Yeung J, Meeks R, Edelson D, Gao F, Soar J, Perkins GD. The use of CPR feedback/prompt devices during training and CPR performance: a systematic review. Resuscitation 2009;80:743–51.
25. Baubin M, Haid C, Hamm P, Gilly H. Measuring forces and frequency during active compression decompression cardiopulmonary resuscitation: a device for training, research, and real CPR. Resuscitation 1999;43:17–24.
26. Aase SO, Myklebust H. Compression depth estimation for CPR quality assessment using DSP on accelerometer signals. IEEE Trans Biomed Eng 2002;49:263–8.
27. Krasteva V, Jekova I, Didon JP. An audiovisual feedback device for compression depth, rate and complete chest recoil can improve the CPR performance of lay persons during self-training on a manikin. Physiol Meas 2011;32:687–99.
28. Kinnen E, Duff C. Cardiac output from transthoracic impedance records using discriminant analysis. J Assoc Adv Med Instrum 1970;4:73–8.
29. Ito H, Yamakoshi KI, Togawa T. Transthoracic admittance plethysmograph for measuring cardiac output. J Appl Physiol 1976;40:451–4.
30. Stecher FS, Olsen JA, Stickney RE, Wik L. Transthoracic impedance used to evaluate performance of CPR during cardiac arrest. Resuscitation 2008;79:432–7.
31. Li Y, Ristagno G, Bisera J, Tang W, Deng Q, Weil MH. Electrocardiogram waveforms for monitoring effectiveness of chest compression during cardiopulmonary resuscitation. Crit Care Med 2008;36:211–5.
32. Edelson DP, Abella BS, Kramer-Johansen J, et al. Effects of compression depth and pre-shock pauses predict defibrillation failure during cardiac arrest. Resuscitation 2006;71:137–45.
33. Losert H, Risdal M, Sterz F, et al. Thoracic impedance changes measured via defibrillator pads can monitor ventilation in critically ill patients and during cardiopulmonary resuscitation. Crit Care Med 2006;34:2399–405.
34. Kramer-Johansen J, Eilevstjønn J, Olasveengen TM, Tomlinson AE, Dorph E, Steen PA. Transthoracic impedance changes as a tool to detect malpositioned tracheal tubes. Resuscitation 2008;76:11–63.
35. Roberts K, Srinivasan V, Niles DE, et al. Does change in thoracic impedance measured via defibrillator electrode pads accurately detect ventilation breaths in children? Resuscitation 2010;81:1544–9.
36. Johnston PW, Imam Z, Dempsey G, Anderson J, Adgey AA. The transthoracic impedance cardiogram is a potential haemodynamic sensor for an automated external defibrillator. European Heart Journal 1998;19:1879–88.
37. Pellis T, Bisera J, Tang W, Weil MH. Expanding automatic external defibrillators to include automated detection of cardiac, respiratory, and cardiorespiratory arrest. Crit Care Med 2002;30:S176–8.
38. Losert H, Risdal M, Sterz F, et al. Thoracic-impedance changes measured via defibrillator pads can monitor signs of circulation. Resuscitation 2007;73:221–8.
39. Aramendi E, Ayala U, Irusta U, Alonso E, Eftestøl T, Kramer-Johansen J. Suppression of the cardiopulmonary resuscitation artefacts using the instantaneous chest compression rate extracted from the thoracic impedance. Resuscitation 2011, http://dx.doi.org/10.1016/j.bbr.2011.03.031.