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Analysis of transthoracic impedance during real cardiac arrest defibrillation attempts in older children and adolescents: Are stacked-shocks appropriate?☆

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Abstract

Background—In 2005, the AHA changed the treatment recommendation for shockable rhythms from 3 transthoracic stacked-shocks to a single shock followed by immediate chest compressions. The stacked-shock recommendation was based on low first-shock efficacy of monophasic waveforms and the theoretical decrease in transthoracic impedance (TTI) following each shock. The objective of this study was to characterize TTI following biphasic defibrillation attempts in children ≥8 yrs during cardiac arrest to assess whether a stacked-shock approach may be appropriate to improve defibrillation success.

Methods—TTI (Ohms (Ω)) was collected via standard anterior-apical defibrillator electrode pads during consecutive in-hospital cardiac arrest biphasic defibrillation attempts in children ≥8 yrs. Analytic data points for TTI were: 0.1 s pre-shock (baseline); post-shock at 0.1, 0.5, 1.0, 1.5, and 2.0 s. TTI variables analyzed with descriptive summaries/paired *t*-test. p values < 0.05 considered statistically significant after correction for multiple comparisons.

Results—Analysis yielded 13 evaluable shock events during 5 cardiac arrests (mean age 14.3 ± 5 yrs, weight 47.4 ± 7.3 kg) between September 2006 and May 2009. Compared to 0.1 s pre-shock baseline values (56.8 ± 23.4 Ω), TTI was significantly lower immediately 0.1 s post-shock (55.2 ± 22.2 Ω, $p = 0.003$). Post-shock mean difference from baseline was 1.6 Ω at 0.1 s ($p = 0.015$), 1.4 Ω

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Conflict of interest statement

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at 0.5 s ($p = 0.019$) 1.4 Ω at 1.0 s ($p = 0.023$), 1.1 Ω at 1.5 s ($p = 0.028$), and 0.95 Ω at 2.0 s ($p = 0.096$). Time to recharge our clinical defibrillators to standard biphasic shock dose was 2.80 ± 0.05 s.

Conclusions—During cardiac arrests in children ≥8 yrs, TTI decreased after biphasic shocks, but the limited magnitude and duration of TTI changes suggest that stacked-shocks would not improve defibrillation success.

Keywords

Transthoracic impedance; Resuscitation; Pediatric; Adolescent; Defibrillation

1. Introduction

Of hospitalized pediatric patients, 2–6% will suffer a cardiac arrest.[1–3] The incidence of a shockable rhythm (both initial and subsequent ventricular fibrillation and pulseless ventricular tachycardia) is approximately 27% during pediatric in-hospital cardiac arrests.[1] Recent pediatric data indicate that only 50–60% of initial shocks successfully terminate fibrillation compared with approximately 90% success rates in adults.[4–10] In addition, interruptions in chest compressions (i.e., blood flow) to recharge the defibrillator during stacked-shocks is associated with worse shock success and worse survival outcome in adults.[9–13]

Successful defibrillation is dependent upon the delivery of adequate electrical current to the myocardium. One of the major determinants of current flow is transthoracic impedance. Early studies found that repeated direct current (DC) shocks resulted in a progressive decrease in transthoracic apparent impedance that was dependent upon the time interval between DC shocks.[14] Since lowered impedance results in higher delivered current for the same energy setting on a defibrillator, this observation seemed to explain the apparent enhanced effectiveness of repeated monophasic shocks in defibrillation. As a result of these preliminary studies, stacked-shocks were recommended because of low first-shock efficacy of monophasic waveform shocks and theoretical decrease in transthoracic impedance (TTI) following each shock.[15]

TTI varies with many factors, including body weight, body surface area, paddle/pad size, pad location, pad–skin contact, body fat, number of prior shocks and hypothermia.[16–25] There is conflicting animal and adult evidence whether TTI significantly decreases after shocks[7,16,26–28] and whether this makes a clinical difference in termination of fibrillation outcomes.[29–31] In 2005, the AHA changed recommendations to treat ventricular fibrillation from three transthoracic stacked-shocks to one shock followed by immediate chest compressions because biphasic shocks were approximately 90% effective at terminating fibrillation and therefore providing uninterrupted perfusion post-shock is more important than further shocks.

We evaluated TTI in children after biphasic defibrillation shocks during cardiac arrest. Our hypothesis was that TTI may decrease immediately after a shock in children ≥8 yrs during cardiac arrest, but the duration of decreased TTI would be too short for a stacked-shock approach to feasibly improve defibrillation success.

2. Methods

2.1. Study design

This was a retrospective observational study approved by the Institutional Review Board at the Children's Hospital of Philadelphia. Data collection procedures were completed in compliance with the guidelines of the Health Insurance Portability and Accountability Act to ensure subject confidentiality. Written informed consent was waived since all data collected were de-identified. Consecutive children ≥8 yrs with cardiac arrest who received biphasic shocks using standard clinical anterior-apical self-adhesive electrode defibrillation pads were eligible for inclusion.

2.2. Data collection

Demographic data including age, gender, and weight were obtained for all subjects. Data on TTI was obtained with commercially available self-adhesive defibrillator pads (HeartStart Pad, M3718A—Adult Radiotransparent Multifunction Electrode Pads, Philips Medical Systems, Seattle, WA) and continuously recorded and measured via the Philips HeartStart MRx/Q-CPR biphasic monitor/defibrillator (Philips Medical Systems, Andover, MA). Adult pads were selected for those patients >10 kg and pads were placed in standard anterior-apical position. To determine time required to charge and discharge the Philips HeartStart MRx/Q-CPR defibrillator, investigators used a fully charged, unplugged HeartStart MRx monitor/defibrillator, selected a standard dose of 150 joules (J), and manually charged and manually discharged the defibrillator in a rapid manner 5 consecutive times and recorded the results.

2.3. Data analysis

TTI (Ohms) was continuously measured by the defibrillator pads and specifically documented at baseline (0.1 s pre-shock) and then at 0.1, 0.5, 1.0, 1.5 and 2.0 s post-shock. Descriptive statistics are presented as mean ± SD. Paired *t*-test was used for univariate analysis to evaluate a change in TTI before and after defibrillation. Standard number comparison ($k = 5$) was used for univariate analysis. A hierarchical mixed effect linear model was developed to estimate the change of TTI. This allowed a model of the initial drop and subsequent return of TTI in each event with piecewise method while controlling for clustering of events by subject, using a random intercept model by subject and a random intercept, random slope model by event nested within a subject. *p* values less than 0.05 is considered statistically significant. The ‘xtmixed’ command in Stata version 11 (College Station, TX) was used for hierarchical analysis. Time to charge and discharge the defibrillator is reported with standard descriptive summaries (mean ± SD).

3. Results

From September 2006 to May 2009 there were 37 cardiac arrests in children ≥8 years old. Of those 37, 10 patients had a shockable rhythm and we attempted to retrieve TTI data from each. The 5 patients with the most stable impedance signal pre- and post-shock were included for analysis. Analysis yielded 13 evaluable shock events during 5 cardiac arrests. Table 1 displays demographic data and actual transthoracic impedance at baseline (0.1 s pre-shock) and after each defibrillation attempt. Mean age of patients was 14.3 ± 5 yrs and their mean weight was 47.4 ± 7.3 kg. Compared to 0.1 s pre-shock baseline values (56.8 ± 23.4 Ω), TTI was significantly lower immediately 0.1 s post-shock (55.2 ± 22.2 Ω, $p = 0.003$). Post-shock mean difference from baseline was 1.6 Ω at 0.1 s ($p = 0.015$), 1.4 Ω at 0.5 s ($p = 0.019$), 1.4 Ω at 1.0 s ($p = 0.023$), 1.1 Ω at 1.5 s ($p = 0.028$), and 0.95 Ω at 2.0 s ($p = 0.096$). These *p* values were adjusted for multiple comparisons. Based on a hierarchical mixed effect analysis, the initial shock dropped the TTI by 1.6 ± 0.4 Ω then gradually increased by 0.4 ± 0.2 Ω/s, after adjusting for clustering by subject and variance among the events. The model predicts that the TTI will return to baseline at 4 s.

Average time to charge the Philips Heartstart MRx biphasic defibrillator to 150 J with fully charged battery was 2.80 ± 0.05 s. Time to charge and discharge 150 J with fully charged battery was 2.87 ± 0.04 s (Philips specifies charge time of less than 5 s to 200 J with a new, fully charged lithium ion battery at 25 °C and a Shock-to-Shock cycle time of less than 20 s.[32]).

4. Discussion

These data establish that TTI can decrease after a biphasic shock for children ≥8 yrs in ventricular fibrillation. However, the magnitude of decrease in TTI is <3% at 0.1 s post-shock. Importantly, the hierarchical mixed effect model indicates that the initial biphasic shock dropped the TTI by 1.6 Ω from a mean TTI of 56.8 ± 23.4 Ω, and then the TTI gradually increased at 0.4 Ω/s, thereby returning to baseline by 4 s post-shock. Because of the small decrease in TTI and because the minimal time required for the defibrillator to recharge and discharge was 2.87 ± 0.04 s, these data support our hypothesis that a stacked-shock approach for children is unlikely to improve defibrillation success.

Current, not energy, is the agent of defibrillation. Successful defibrillation is dependent upon the delivery of adequate electrical current to the myocardium. One of the major determinants of current flow is transthoracic impedance. TTI is dependent on multiple patient and care delivery factors: body weight, body surface area, chest size, fat, hypothermia, variations in patient fluid status (pulmonary edema, heart failure, edema, etc.), paddle/pad size, paddle force and skin contact, pad orientation and number of prior shocks given.[16–26] Defibrillation success is affected mainly by TTI and energy dose delivered, though various other patient and equipment factors also affect the response to defibrillation attempts.

Prior recommendations of stacked-shock approach for defibrillation was based on the low efficacy of monophasic waveform defibrillation and the theoretical decrease in transthoracic impedance after shocks.[15] Prior protocols with escalating energy stacked-shocks were based on the belief that a failed defibrillation attempt indicated the presence of high transthoracic impedance and that increasing to a higher energy setting for the next defibrillation attempt would therefore improve success. The rationale of providing the first two shocks at the same energy level was that the decrease in TTI following the first shock would increase trans-myocardial current during the second shock. In theory, one would eventually reach an efficacious current while avoiding excessive current. However, this issue is less important in the era of adult biphasic defibrillation because approximately 90% of first shocks now terminate VF.[4–10]

Limited animal and human data indicate that repeat shocks can decrease the TTI moderately, presumably by cutaneous post-shock hyperemia and edema.[14,16,17] Nevertheless, other animal and adult human data have shown that TTI may not decrease after a defibrillation attempt.[7,26,28] The largest adult study evaluating TTI after 863 defibrillator shocks demonstrated that the TTI is nearly as likely to increase as decrease after a first shock for out-of-hospital cardiac arrests.[7] The authors noted that the lack of consistent decreases in TTI post-shock may have been partly due to the prolonged duration of cardiac arrest in the out-of-hospital, because prolonged poor cutaneous blood flow may have precluded the post-shock hyperemia purported to decrease the TTI.

What is the clinical significance of the small decreases in TTI noted in this series of children ≥8 yrs? Although we are not aware of any studies that support an absolute or relative drop in TTI which would make subsequent defibrillation attempts more successful, Walker and colleagues asserted that the 1% decrease in TTI and resultant 1% increase in current that they documented after shocks for adult out-of-hospital cardiac arrests were not "meaningful".[7] We agree with these authors. In addition, our model also shows that the TTI will return to baseline by 4 s. Because charging and discharging the defibrillator requires more than 2 s, it is likely that the TTI is essentially unchanged by the time a shock is provided.

5. Limitations

The most important limitation of this study is the small sample size. Nevertheless, our data are quite similar to recent published data in adults.[7,26] None of the children were younger than 8 years old, so this data may not be generalizable to younger children. Many patients were necessarily excluded due to poor pad–chest interface resulting in inadequate TTI data. TTI was examined in isolation, and the source of the TTI (e.g. thoracic volume, body fat, time interval from initial VF/VT to first shock, orientation and size of defibrillation pads, presence of pneumothorax, pleural effusion, pulmonary edema, respiratory cycle, transient changes in patient electrode contact, or whether the defibrillator impedance signal filters are stabilizing post-shock) was not examined. Importantly, since this data is not generalizable to the smaller, younger patient, the approach to defibrillation, correct dose, and effects on TTI relative to heart size in smaller children and infants continues to be unknown and should be fully investigated.

6. Conclusion

During in-hospital defibrillation of children ≥8 yrs, TTI decreased by <3% at 0.5 s post-shock. This small TTI reduction quickly dissipated and returned to baseline within 4 s. Because of the small decrease in TTI and because the minimal time required for the defibrillator to recharge and discharge was 2.87 ± 0.04 s, these data support our hypothesis that a stacked-shock approach for children is unlikely to improve defibrillation success.

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Abbreviations

CPR	cardiopulmonary resuscitation
TTI	transthoracic impedance
ΔTI	change in thoracic impedance
J	joules
Ω	Ohms
VF	ventricular fibrillation

References

1. Samson RA, Nadkarni VM, Meaney PA, et al. American Heart Association National Registry of CPR Investigators. Outcomes of in-hospital ventricular fibrillation in children. N Engl J Med. 2006; 354:2328–39. [PubMed: 16738269]
2. Reis AG, Nadkarni V, Perondi MB, Grisi S, Berg RA. A prospective investigation into the epidemiology of in-hospital pediatric cardiopulmonary resuscitation using the international Utstein reporting style. Pediatrics. 2002; 109:200–9. [PubMed: 11826196]

3. Suominen P, Olkkola KT, Voipio V, Korpela R, Palo R, Räsänen J. Utstein style reporting of in-hospital paediatric cardiopulmonary resuscitation. Resuscitation. 2000; 45:17–25. [PubMed: 10838235]
4. Berg MD, Samson RA, Meyer RJ, Clark LL, Valenzuela TD, Berg RA. Pediatric defibrillation doses often fail to terminate prolonged out-of-hospital ventricular fibrillation in children. Resuscitation. 2005; 67:63–7. [PubMed: 16199288]
5. Kudenchuk PJ, Cobb LA, Copass MK, Olsufka M, Maynard C, Nichol G. Transthoracic incremental monophasic versus biphasic defibrillation by emergency responders (TIMBER): a randomized comparison of monophasic with biphasic waveform ascending energy defibrillation for the resuscitation of out-of-hospital cardiac arrest due to ventricular fibrillation. Circulation. 2006; 114:2010–8. [PubMed: 17060379]
6. van Alem AP, Chapman FW, Lank P, et al. A prospective, and randomised comparison of first shock success of monophasic and biphasic waveforms in out-of-hospital cardiac arrest. Resuscitation. 2005; 58:17–24. [PubMed: 12867305]
7. Walker RG, Koster RW, Sun C, et al. Defibrillation probability and impedance change between shocks during resuscitation from out-of-hospital cardiac arrest. Resuscitation. 2009; 80:773–7. [PubMed: 19423211]
8. Schneider T, Martens PR, Paschen H, et al. Multicenter, randomized, controlled trial of 150-J biphasic shocks compared with 200- to 360-J monophasic shocks in the resuscitation of out of hospital cardiac arrest victims. Circulation. 2000; 102:1780–6. [PubMed: 11023932]
9. Meaney PA, Nadkarni VM, Kern KB, Indik JH, Halperin HR, Berg RA. Rhythms and outcomes of adult in-hospital cardiac arrest. American Heart Association National Registry for Cardiopulmonary Resuscitation Investigators. Crit Care Med. 2010; 38:101–8. [PubMed: 19770741]
10. American Heart Association. Guidelines 2005 on cardiopulmonary resuscitation and emergency cardiovascular care. Circulation. 2005; 112:1–203.
11. 2005 International consensus on cardiopulmonary resuscitation and emergency cardiovascular care science with treatment recommendations. Circulation. 2005; 112:1–136.
12. Edelson DP, Abella BS, Kramer-Johansen J, et al. Effects of compression depth and pre-shock pauses predict defibrillation failure during cardiac arrest. Resuscitation. 200610.1016/j.resuscitation.2006.04.008
13. Abella BS, Alvarado JP, Myklebust H, et al. Quality of cardiopulmonary resuscitation during in-hospital cardiac arrest. JAMA. 2005; 293:363–5. [PubMed: 15657330]
14. Dahl CF, Ewy GA, Ewy MD, Thomas ED. Transthoracic impedance to direct current discharge: effect of repeated countershocks. Med Instrum. 1976; 10:151–4. [PubMed: 1272089]
15. American Heart Association. Guidelines 2000 for cardiopulmonary resuscitation and emergency cardiovascular care: international consensus on science. Circulation. 2000; 102:I142–57. [PubMed: 11001639]
16. Sirna SJ, Kieso RA, Fox-Eastham KJ, Seabold J, Charbonnier F, Kerber RE. Mechanisms responsible for decline in transthoracic impedance after DC shocks. Am J Physiol. 1989; 257:H1180–3. [PubMed: 2801977]
17. Kerber RE, Grayzel J, Hoyt R, Marcus M, Kennedy J. Transthoracic resistance in human defibrillation. Influence of body weight, chest size, serial shocks, paddle size and paddle contact pressure. Circulation. 1981; 63:676–82. [PubMed: 7460251]
18. Krasteva V, Matveev M, Mudrov N, Prokopova R. Transthoracic impedance study with large self-adhesive electrodes in two conventional positions for defibrillation. Physiol Meas. 2006; 27:1009–22. [PubMed: 16951460]
19. Zhang Y, Clark CB, Davies LR, Karlsson G, Zimmerman MB, Kerber RE. Body weight is a predictor of biphasic shock success for low energy transthoracic defibrillation. Resuscitation. 2002; 54:281–7. [PubMed: 12204462]
20. Deakin CD, Sado DM, Petley GW, Clewlow F. Is the orientation of the apical defibrillation paddle of importance during manual external defibrillation? Resuscitation. 2003; 56:15–8. [PubMed: 12505733]
21. Garcia LA, Kerber RE. Transthoracic defibrillation: does electrode adhesive pad position alter transthoracic impedance? Resuscitation. 1998; 37:139–43. [PubMed: 9715772]

22. Deakin CD, McLaren RM, Petley GW, Clewlow F, Dalrymple-Hay MJ. A comparison of transthoracic impedance using standard defibrillation paddles and self-adhesive defibrillation pads. Resuscitation. 1998; 39:43–6. [PubMed: 9918446]
23. Deakin CD, Bennetts SH, Petley GW, Clewlow F. What is the optimal paddle force during paediatric external defibrillation? Resuscitation. 2003; 59:83–8. [PubMed: 14580737]
24. Deakin CD, Sado DM, Petley GW, Clewlow F. Differential contribution of skin impedance and thoracic volume to transthoracic impedance during external defibrillation. Resuscitation. 2004; 60:171–4. [PubMed: 15036735]
25. Atkins DL, Kerber RE. Pediatric defibrillation: current flow is improved by using "adult" electrode paddles. Pediatrics. 1994; 94:90–3. [PubMed: 8008545]
26. Deakin CD, Ambler JJ, Shaw S. Changes in transthoracic impedance during sequential biphasic defibrillation. Resuscitation. 2008; 78:141–5. [PubMed: 18486297]
27. Fumagalli S, Tarantini F, Caldi F, et al. Multiple shocks affect thoracic electrical impedance during external cardioversion of atrial fibrillation. Pacing Clin Electrophysiol. 2009; 32:371–7. [PubMed: 19272068]
28. Niemann JT, Garner D, Lewis RJ. Transthoracic impedance does not decrease with rapidly repeated countershocks in a swine cardiac arrest model. Resuscitation. 2003; 56:91–5. [PubMed: 12505744]
29. Dalzell GW, Adgey AA. Determinants of successful transthoracic defibrillation and outcome in ventricular fibrillation. Br Heart J. 1991; 65:311–6. [PubMed: 2054239]
30. White RD, Blackwell TH, Russell JK, Snyder DE, Jorgenson DB. Transthoracic impedance does not affect defibrillation, resuscitation or survival in patients with out-of-hospital cardiac arrest treated with a non-escalating biphasic waveform defibrillator. Resuscitation. 2005; 64:63–9. [PubMed: 15629557]
31. Dalzell GW, Cunningham SR, Anderson J, Adgey AA. Electrode pad size, transthoracic impedance and success of external ventricular defibrillation. Am J Cardiol. 1989; 64:741–4. [PubMed: 2801525]
32. Philips website: HeartStart MRx ALS Monitor/Defibrillator; specifications: http://www.healthcare.philips.com/main/products/resuscitation/products/MRx/mrx_als.wpd#&&/wEXAQUOY3VycmVudFRhYlBhdGgFFkRldGFpbHM6U3BlY2lmaWNhdGlvbnOMsm5HSTlfUhiZBla88BAYqeUicw==.

Table 1

Demographic data and transthoracic impedance (Ohms).

Event	Age (yrs)	Gender	Body mass (kg)	Chest depth (cm)	Shock#	Time since last shock (s)	Energy selected (J)	0.1 s pre-shock (baseline)	0.1 s post-shock	0.5 s post-shock	1.0 s post-shock	1.5 s post-shock	2.0 s post-shock
1	16.9	M	55	20.3	1	–	150	78.6	74.9	75.2	75.3	76.2	76.1
					2	146	200	77.5	73.4	74.1	74.5	76.7	77.7
2	10.4	F	40	15	1	–	50	69.9	68.4	69.1	*a*	*a*	*a*
					2	232	100	46.7	46.2	46.5	46.6	46.6	46.9
					3	103	100	48.2	46	46.3	46.4	46.4	46.8
					4	93	100	48.2	47	46.9	46.8	46.7	47
					5	336	150	45.8	45.3	45.4	45.3	45.2	45.5
3	8.1	F	40	15	1	–	50	92.6	88.9	89	89	89	89
					2	189	50	94.9	93.1	93.2	93.2	93.2	93.5
4	14.9	F	48	21	1	–	100	27.7	28.3	28.2	28.1	28.1	28
					2	113	100	29	29.6	29.5	29.4	29.3	29.2
					3	152	200	29.5	29.9	29.9	29.8	29.8	29.8
5	21	M	54	20	1	–	150	48.3	46.6	46.8.	47.1	47.4	47.4
							Mean	56.8	55.2	56.1	54.3	54.6	54.7
							SD	23.4	22.2	23.1	22.9	23.2	23.3

[a]No data due to ventilation or chest compression artifact.