Original Study

Journal of Veterinary Emergency and Critical Care 22(4) 2012, pp 435–440
doi: 10.1111/j.1476-4431.2012.00782.x

The effect of using an impedance threshold device on hemodynamic parameters during cardiopulmonary resuscitation in dogs

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Abstract

Objective – To investigate the hemodynamic effects following the use of an impedance threshold device (ITD) in a canine model of cardiopulmonary arrest.
Design – Experimental, randomized crossover study.
Setting – Cardiovascular research laboratory at a university veterinary center.
Animals – Eight purpose bred beagle dogs.
Interventions – Dogs were anesthetized and instrumented for the measurement of right atrial pressure , systolic blood pressure, mean blood pressure, and diastolic arterial blood pressure, end-tidal CO_2, and carotid blood flow (CBF). CBF was determined via ultrasonic flow probe placed around the carotid artery. Animals were taking part in an unassociated terminal study and following subsequent euthanasia with pentobarbital, standardized cardiopulmonary resuscitation (CPR) was performed with an impedance threshold device attached (ITD-CPR group) and without (S-CPR group). Order of treatment was randomized.
Measurements and Main Results – ITD group had increased CBF, coronary perfusion pressure, and a decrease in right atrial diastolic pressure. No differences in end-tidal CO_2, diastolic arterial blood pressure, mean blood pressure, or systolic blood pressure were seen. Return of spontaneous circulation was not observed in any of the animals
Conclusions – Use of the ITD resulted in favorable changes in hemodynamic parameters in dogs undergoing CPR. The ITD may be a useful adjunct during CPR in dogs and warrants clinical evaluation.

(J Vet Emerg Crit Care 2012; 22(4): 435–440) doi: 10.1111/j.1476-4431.2012.00782.x

Keywords: carotid blood flow, CPR, hemodynamic monitoring, intrathoracic pressures, ITD

Introduction

Cardiopulmonary resuscitation (CPR) in veterinary medicine historically had a limited success rate.[1–3] In a prospective study, CPR was able to achieve return of spontaneous circulation (ROSC) in 35% of dogs and 44% of cats,[1] and several studies have reported poor survival rates to discharge of between 4% and 7%.[1–5] Due to a limited number of veterinary studies, CPR in veterinary medicine has been largely adapted from human guidelines. The 2010 American Heart Association (AHA) guidelines for CPR recommended immediate chest

Abbreviations

CBF	carotid blood flow
CPP	coronary perfusion pressure
CPR	cardiopulmonary resuscitation
DAP	diastolic arterial pressure
$ETCO_2$	end-tidal CO_2
ICP	intracranial pressure
ITD	impedance threshold device
ITTP	intrathoracic pressure
MAP	mean arterial pressure
RAP	right atrial pressure
ROSC	return of spontaneous circulation
SAP	systolic arterial pressure

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The authors declare no conflicts of interest.

Presented in part at the American College Veterinary Internal Medicine (ACVIM) Forum, Denver, CO, USA, June 2011.

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Submitted May 17, 2011; Accepted June 5, 2012.

compression at a fast rate (>100/min) allowing for full chest recoil, with minimal interruption.[6] The chest wall needs to fully recoil after each compression in order to decrease intrathoracic pressure (ITTP) and thereby maximize venous return.[6] Standard manual chest compressions, however, produce coronary and cerebral perfusion, that is, 20%–30% of normal.[7] Several adjunctive CPR techniques and devices may improve hemodynamics by manipulation of ITTP and thus provide additional blood flow.

The aim of CPR is to provide maximal blood flow to the heart and to the brain, ultimately facilitating restoration of spontaneous circulation.[2] During cardiac arrest and subsequent resuscitation, the coronary perfusion pressure (CPP), and hence the blood supply to the myocardium, is determined by the difference between aortic diastolic pressure and right atrial pressure (RAP); hence, interventions should be aimed at increasing aortic pressure and minimizing increases in RAP to maximize perfusion. The cerebral perfusion pressure is determined as the difference between mean arterial pressure (MAP) and intracranial pressure (ICP). So interventions to improve cerebral perfusion are targeted at increasing MAP and at reducing ICP. During conventional CPR with positive pressure ventilation, both chest compression and ventilation contribute to an abnormally high ITTP, restricting venous return, and increasing RAP due to transmural transmission of ITTP to the atrium. Lowering ITTP can both decrease RAP and ICP as well as increasing venous return and cardiac output improving both cerebral and CPP.[8–10] The mechanism of how lowering ITTP can cause an immediate drop in ICP is not fully understood. Pressure is transferred through the thoracic vertebral column to the cerebrospinal fluid and the nonvalvular vein around the spinal cord.[10]

The impedance threshold device (ITD) can be attached between the endotracheal tube and the breathing circuit.[11,12] The device prevents air inflow via the endotracheal tube during chest recoil until a certain "cracking pressure" is reached, usually –12 cm H_2O for the devices used during CPR. In practice, this means that a more negative pressure is generated in the chest during chest wall recoil, as the ITD will not allow airflow into the endotracheal tube during the first part of chest wall recoil. In porcine CPR models, the use of an ITD has been associated with improved hemodynamics.[13–15] Several randomized controlled trials in people have demonstrated improved clinical outcomes in patients who are resuscitated using the ITD and a recent meta-analysis concluded that the ITD improves short term outcome in cardiopulmonary arrest.[16–20] The AHA guidelines for cardiopulmonary resuscitation include use of an ITD during cardiopulmonary resuscitation in adults (class IIb).[21] Use of the low cracking pressure ITD (–7 cm H_2O) has been reported in spontaneous breathing dogs to improve cardiac output, hemodynamic parameters during hemorrhagic shock, and anesthesia-induced hypotension.[12,22] The hemodynamic effects of ITD use during CPR in dogs, however, has not been investigated. The aim of the present study was to investigate the short-term cardiovascular effects of application of an ITD in dogs during CPR. The hypothesis of this study was that the use of ITD during CPR would improve carotid blood flow (CBF), CPP, and arterial blood pressure when compared to standard CPR (S-CPR).

Materials and Methods

Animal preparation

The study design was approved by the Institutional Animal Care and Use Committee of the University of Florida. Eight healthy, purpose bred, 1–5 year old beagle dogs that were part of a separate terminal study were used. All dogs were deemed healthy based on physical exam and serum biochemistry profile. Animals were fasted for 12 hours prior to the experiment but had free access to water.

An 18-G over the needle intravenous catheter[a] was aseptically placed in a cephalic vein; anesthesia was induced by injection of propofol[b] 5–10 mg/kg intravenously. An endotracheal tube was placed and the cuff inflated. Anesthesia was maintained with isoflurane[c] vaporized in oxygen and delivered via a circle breathing system.[d] The dogs were mechanically ventilated using a rate and volume controlled ventilator[e] at a tidal volume of 15 mL/kg, the respiratory rate was adjusted to maintain an end-tidal CO_2 ($ETCO_2$) of between 35 and 45 mm Hg. Normothermia was maintained using a circulating warm water blanket and warm forced air blanket. A 20-G 1″ dorsal pedal arterial catheter was placed percutaneously. Using a standard cut-down technique the right carotid artery and right internal jugular vein were exposed. A 15-cm 7-Fr right atrial catheter was placed via the right jugular vein, and correct placement was confirmed by monitoring of the pressure waveform as the catheter was advanced into the heart. A 3-mm ultrasonic flow probe[f] was placed around the left common carotid artery to allow measurement of blood flow through the vessel. Using an integrated hemodynamic monitor and bioamplifier[g] the $ETCO_2$, end-tidal isoflurane, direct systolic arterial pressure, mean arterial pressure, and diastolic arterial pressure (SAP, MAP, and DAP, respectively), RAP, heart rate, and CBF was collected. All data were transferred to a computer with data recording program[h] at a sampling rate of 100 data points per second. The CPP was calculated from the difference between the peripheral arterial diastolic pressure and the right atrial diastolic pressure.

 doi: 10.1111/j.1476-4431.2012.00782.x

Figure 1: Graphic representation of right atrial pressure (RAP), invasive arterial blood pressure (IBP), carotid blood flow (CBF), and ECG during: (A) baseline (general anesthesia); (B) during CPR with the impedance threshold device (CPR-ITD); and (C) during standard cardiopulmonary resuscitation (S-CPR), after pentobarbital-induced cardiac arrest.

Experimental protocol

The protocol for the previous study utilizing the dogs dictated placement of a high-epidural catheter (T1-T2) and the administration of methylene blue followed later by euthanasia via intravenous injection of pentobarbital.[i] Baseline hemodynamic values, and CBF were recorded over 2 minutes immediately before euthanasia, with the animal still anesthetized, end-tidal isoflurane was kept constant at 1.6 ± 0.1%. Following euthanasia, ventilation and administration of isoflurane was stopped.

CPR was initiated 2 minutes after euthanasia, basic life support was provided with manual chest compressions given by a single rescuer at 100–120/min. Mechanical ventilation was reinstituted at 8/min, and a tidal volume of 15 mL/kg with 100% oxygen.

The dogs were randomized in a crossover design to receive S-CPR and CPR with the ITD device[g] (ITD-CPR). Two minutes of CPR were performed followed by a 1-minute break and a further 2 minutes of CPR. One of the periods of CPR was performed with the ITD[j] attached to the breathing circuit and one without the ITD (Figure 1). The order of application of the ITD was not determined for each dog in advance using a computer generated randomization table. The rescuer was blinded to the application of the ITD and the data monitoring by placing a blanket over the head of the dog, ventilator, breathing system, and data monitoring equipment. The same rescuer (F.G.) was used for all animals and all treatments.

Statistical methods

The average across the entire 2 minutes continuous beat-to-beat hemodynamic data for baseline, S-CPR, and ITD-CPR was used for analysis. Data are reported as mean and standard deviation and were analyzed using commercial statistical software.[k] Normality and equality of data variances were assessed using the Kolmogorov–Smirnov and Leven test, respectively. For each group, a one-way ANOVA was used. When appropriate a pairwise multiple comparison procedure (Fisher's least significance difference [LSD]

Table 1: Mean ± standard deviation of carotid blood flow (CBF) and percentage of baseline CBF (%CBF), right atrial diastolic pressure (RAD), right atrial systolic pressure (RAS), systolic blood pressure, mean blood pressure, and diastolic arterial blood pressure (SAP, MAP, and DAP), coronary perfusion pressure (CPP), and end-tidal CO_2 ($ETCO_2$) during general anesthesia (baseline), during standard cardiopulmonary resuscitation (S-CPR) and during CPR with the impedance threshold device (CPR-ITD) after pentobarbital-induced cardiac arrest. RAS data expressed as median (first quartile, third quartile). *P*-value reflects statistics difference between S-CPR and ITD-CPR groups $^*P < 0.05$. CPP was calculated as DAP-RAD.

Level	Baseline	S-CPR	ITD-CPR	*P*-value
CBF (mL/min)*	165 ± 19.1	30.8 ± 18.5	53.6 ± 23.7	0.03
%CBF (%)*	100 ± 0	18.5 ± 10.1	30±15	0.028
RAD (mm Hg)*	−1.6 ± 2.7	1.8 ± 4.6	−5.1±6.6	0.026
RAS (mm Hg)	0.5 (−1, 1.5)	34.6 (27, 48)	47 (21, 60)	0.68
SAP (mm Hg)	70 ± 15	29 ± 7.3	30 ± 9.5	0.94
MAP (mm Hg)	52 ± 12.9	19 ± 6.7	20.8 ± 9.3	0.74
DAP (mm Hg)	43 ± 10.7	10.5 ± 5.8	16.6 ± 7.0	0.15
CPP (mm Hg)*	44.6 ± 10	8.9 ± 6.9	21 ± 7.5	0.033
ET_{CO2} (mm Hg)	37 ± 3	6.6 ± 6.5	6.1 ± 4	0.94

method) was used to assess the differences between baseline and CPR groups. For all analyses a *P*-value < 0.05 was considered statistically significant.

Results

There was no difference within the group in any of the hemodynamic data at baseline. All values passed normality with the exception of right atrial systolic pressure (data expressed as median, interquartile range). All animals developed asystole following the administration of euthanasia solution and no animals had signs of ROSC following or during CPR. SAP, MAP, and DAP, CPP, and CBF values were higher at baseline when compared to any of the CPR methods (Table 1). The RAP during the decompression phase (diastolic RAP) was significantly more negative during ITD-CPR as compared to S-CPR. There was no difference in peak RAP during the compression phase (systolic RA pressure) comparing techniques (Figure 1). The CPP and CBF was significantly higher in the ITD-CPR group as compared to S-CPR group as shown in Table 1. There were no significant differences in $ETCO_2$, MAP, SAP, or DAP.

Discussion

Chest compressions combined with rescue ventilation are the method of choice for CPR delivered by medical professionals.[23] Unfortunately, the combination of positive pressure ventilation and thoracic compressions causes significant increases in ITTP as well as RAP, which in turn limits venous return to the heart. The action of the ITD at least partially attenuates these negative consequences of CPR.[13,14,16–20] The use of the ITD during CPR leads to improved chest decompression as evidenced by the negative diastolic RAP. The reduction in right atrial diastolic pressure seen in ITD-CPR versus S-CPR also led to improved CPP and therefore likely better myocardial perfusion. This is consistent with what has been previously reported in porcine models of CPR.[14,24,25] As arterial pressure is collected more distantly from the heart, there are changes in the arterial waveforms due to elastance of the vessels, reflective waves, and other factors. Usually MAP remains the same but systolic and diastolic pressure change.[26] The CPP was calculated from the DAP and the right atrial diastolic pressure. Though, ideally a catheter placed in the aorta would have given a more accurate indication of coronary perfusion pressure.

The investigators elected to wait a full 2 minutes after euthanasia before the start of CPR to ensure organ hypoxia and to simulate the time it takes for patient in-hospital suffering from cardiac arrest to start receive appropriate resuscitation care.

This study demonstrated that CBF, and so likely cerebral blood flow, was significantly higher in dogs with ITD-CPR than with S-CPR. Previous porcine models have produced mixed results with some investigators[15,25] demonstrating an improvement in cerebral blood flow using ITD-CPR versus S-CPR but other investigators[14] finding no difference. It is important to point out that CBF is an indirect method to evaluate cerebral flow. This study did not directly measure myocardial or cerebral perfusion. This would have been beneficial to assess whether gross changes in major vessel blood flow translated into improved vital tissue perfusion. Alternative techniques would include surgically implanted tissue probes, administration of radio-labeled microspheres during CPR or use of orthogonal polarized spectral imaging techniques. None of those methods were available to the investigators at the time of this study.

The study does have several important limitations; instrumentation and protocols were limited by the fact that the dogs were euthanized as a part of an unrelated study. The previous study dictated euthanasia by barbiturate overdose. Even though other relevant studies evaluating the efficacy of CPR in veterinary medicine have used barbiturate overdose,[27] most animal CPR models have used electrical ventricular fibrillation or asphyxia (hypoxia) to induce cardiac arrest.[13–15] The other disadvantage is the combination of general anesthesia and global ischemia may have caused massive vasodilation and severe decreases in systemic vascular resistance.[28] Barbiturates by themselves can cause severe vasodilation and drop in systemic vascular resistance.[28] This can lead to a decrease

  doi: 10.1111/j.1476-4431.2012.00782.x

in forward flow and reduction of blood pressure during resuscitation efforts regardless of treatment used. Indeed, this study found no significant changes for SAP, DAP, and MAP with the use of the ITD, despite an improvement of CBF.

The number of animals was relatively small ($n = 8$) and because a blinded crossover method was used, temporal bias could have occurred. The order of having ITD or not having ITD was randomized and should have decreased the bias. Another important point is that the current Advanced Cardiac Life Support (ACLS) guidelines recommend minimizing the time that the patient is not receiving chest compressions to a minimum.[6,21] This allows for maximum cardiac priming and ensures continuous forward flow. In the current study, we set a long wait period (1 minute) between the 2 treatments. This allowed for us to place the ITD, initiate hemodynamic monitoring and, more importantly to avoid rescuer fatigue causing further bias in the results as rescuer fatigue plays a significant role in CPR quality.[29–31] Despite the fact that we did not detect a difference between first round of compression from the second, a type II statistical error cannot be ruled out.

The present study examined hemodynamic changes over a short period of time and was not designed to evaluate ROSC and long-term survival. Our results may also not fully represent the hemodynamic scenario of the entire duration of normally performed CPR. Further investigation in veterinary medicine is needed to assess whether ITD use leads to improvements in ROSC, survival, and neurological outcome in the clinical setting.

Conclusion

Application of the ITD during CPR in dogs leads to significant improvements in various hemodynamic parameters. Clinical studies using this device are required to assess whether these favorable hemodynamic changes translate into better long-term outcome in dogs undergoing CPR.

Footnotes

[a] Surflo intravenous catheters, Terumo, Somerset, NJ.
[b] Propoflo 20 mL vial, Abbott Animal Health, IL.
[c] Isoflo, Abbott Animal Health, IL
[d] Surgivet Vaporistic Anesthesia Machine, Smiths Medical, Norwell, MA.
[e] Hallowell EMC 2000 time cycled ventilator.
[f] Animal Blood Flow Meter T206, Transonic Systems Inc, Ithaca, NY.
[g] ADInstruments Power Lab Systems, Castle Hill, NSW, Australia.
[h] Chart pro V7.3 by ADInstruments Power Lab Systems.
[i] Beuthanasia solution, Schering Plough Animal Health Corps, Union, NJ.
[j] ResQPod, Advanced Circulation Systems, Inc, Roseville, MN.
[k] SIGMA STATS, Aspire Software Int. Ashburn, VA.

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