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Reducing ventilation frequency combined with an inspiratory impedance device improves CPR efficiency in swine model of cardiac arrest

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Abstract

Background: The basic premise that frequent ventilations during cardiopulmonary resuscitation (CPR) are a necessity for tissue oxygenation has recently been challenged. An inspiratory impedance threshold device (ITD) recently has also been shown to increase CPR efficiency, principally by augmenting circulation with little impact on ventilation. The optimal compression to ventilation (C/V) is not known for this new device. The purpose of this study was to compare the currently recommended C/V ratio of 5:1 with a 10:1 ratio, ± the ITD, to optimize circulation and oxygenation during CPR. *Methods:* Thirty-two adult pigs weighing 26–31 kg were randomized to CPR with varying C/V ratios ± the ITD as follows: $A = 5{:}1$, $B = 5{:}1 + \text{ITD}$, $C = 10{:}1$, $D = 10{:}1 + \text{ITD}$. After 6 min of untreated ventricular fibrillation (VF), closed-chest standard CPR was performed with an automatic piston device that does not impede passive chest wall recoil, at a continuous compression rate of 100 min^{-1}. Synchronous breaths were given every 5 or 10 compressions during the decompression phase depending on the group. CPR was performed for 6 min and physiological variables were measured throughout the experimental protocol. *Results:* A reduction in the frequency of ventilation from 5:1 to 10:1 resulted in significantly improved arterial and coronary perfusion pressure in a pig model of cardiac arrest. Addition of an ITD resulted in further increases in arterial and coronary perfusion pressures with both 5:1 and 10:1 C/V ratios, without compromising oxygenation. *Conclusion:* CPR efficiency can be optimized by changing the compression: ventilation ratio from 5:1 to 10:1 and with concurrent use of the inspiratory threshold device.

Keywords: Cardiopulmonary resuscitation (CPR); Coronary perfusion pressure; Ventilation; Cardiac arrest

Resumo

Contexto: O conceito de que durante a reanimação cardio-pulmonar (CPR), são necessárias ventilações frequentes para a assegurar a oxigenação tecidular tem sido desafiada recentemente. Um aparelho de limiar de impedância inspiratória (ITD) também mostrou recentemente aumentar a eficiência da CPR, principalmente aumentando a circulação e com pouco impacto na ventilação. A razão compressão para ventilação (C/V) optimizada para este aparelho não é conhecida. O objectivo deste estudo foi comparar as recomendações actuais da relação C/V de 5:1 com 10:1, ± o ITD, para optimizar a oxigenação e a circulação durante a CPR. *Método:* Foram aleatorizados 32 porcos adultos com peso 26–31 kg para CPR com C/V variáveis ± ITD da seguinte forma: $A = 5{:}1$, $B = 5{:}1 + \text{ITD}$; $C = 10{:}1$, $D = 10{:}1 + \text{ITD}$. AO fim de 6 minutos de fibrilhação ventricular (VF) não tratada era efectuada CPR externa com um aparelho de piston automático que não impede a recolha elástica da parede torácica, a uma frequência de compressões constante de 100 min^{-1}. Efectuaram-se insuflações síncronas a cada 5 ou 10 compressões durante a fase de descompressão, dependendo do grupo. A CPR foi efectuada durante 6 minutos e os parâmetros fisiológicos foram medidos durante o protocolo experimental. *Resultados:* A redução da frequência de ventilação de 5:1 para 10:1 resultou numa pressão de perfusão arterial e coronária significativamente melhoradas num modelo de paragem cardíaca em porcos. A adição do ITD resultou em melhoria adicional nas pressões de perfusão arterial e coronária em ambas as relações de C/V, 5:1 ou 10:1, sem comprometer a oxigenação. *Conclusão:* A eficiência da CPR pode ser optimizada pela alteração da relação compressão: ventilação de 5:1 para 10:1 e com o uso concomitante do aparelho de limiar inspiratório.

Palavras chave: Reanimação cardio-pulmonar (CPR); Pressão de perfusão coronária; Ventilação; Paragem cardíaca

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Resumen

Antecedentes: La premisa básica que dice que ventilaciones frecuentes durante la reanimación cardiopulmonar (CPR) son una necesidad para la oxigenación tisular ha sido recientemente desafiada. Recientemente se ha mostrado que un dispositivo de umbral impedancia inspiratoria (ITD)mejora la eficiencia de la CPR, principalmente aumentando la circulación con muy poco impacto en la ventilación. No se conoce aun la relación ventilación compresión (C/V) óptima para este dispositivo. El propósito de este estudio fue comparar la relación C/V de 5 :1 actualmente recomendada con una relación 10:1, ± el ITD, para optimizar la oxigenación y circulación durante la CPR. *Métodos*: Se usaron 32 cerdos adultos que pesaban 26–31 kg, randomizados a CPR variando la relación C/V ± el ITD de la siguiente manera: $A = 5{:}1$, $B = 5{:}1 + \text{ITD}$, $C = 10{:}1$, $D = 10{:}1 + \text{ITD}$. Después de 6 minutos de fibrilación ventricular (VF) sin tratamiento, se realizó CPR estándar a tórax cerrado con un dispositivo de pistón automático que no impide la recuperación pasiva del tórax, a una frecuencia de compresión continua de 100 por minuto. Se dieron ventilaciones sincronizadas cada 5 o 10 compresiones durante la fase de descompresión dependiendo del grupo. Se realizó CPR por 6 minutos y se midieron los parámetros fisiológicos a lo largo del protocolo del experimento. *Resultados*: Una reducción en la frecuencia de ventilación de 5:1 a 10:1 resultó en presiones arteriales y de perfusión coronaria significativamente aumentadas en un modelo porcino de paro cardíaco. La adición del ITD resulto en ulteriores aumentos en presiones arteriales y de perfusión coronaria con ambas relaciones 5:1 y 10:1, sin comprometer la oxigenación. *Conclusión*: La eficiencia de la CPR puede ser optimizada cambiando la relación ventilación compresión de 5:1 a 10:1 y con el uso del dispositivo de umbral de impedancia inspiratoria.

Palabras clave: Reanimación cardiopulmonar (RCP); Presión de perfusión coronaria; Ventilación; Paro cardíaco

1. Introduction

The basic premise that frequent ventilations are a necessity to maintain tissue oxygenation during cardiopulmonary resuscitation (CPR) has been challenged by recent animal and human studies [1–5,12,13]. These new investigations have shown that adequate oxygenation can be maintained with chest compressions alone without ventilation for some limited period of time and that there is no haemodynamic compromise with that approach. When chest compressions are stopped to deliver a breath, coronary perfusion is interrupted and consequently falls until the next series of compressions [17]. An additional cause and explanation for the negative hemodynamic consequences of ventilation is that frequent positive pressure ventilations may result in higher intrathoracic pressures and thereby impede venous return to the heart during the decompression phase of CPR. As such, the frequency of ventilation preventing venous return of blood to the heart during the chest wall decompression phase directly may alter coronary perfusion pressure (CPP) and cardiac output during CPR [8].

The importance of changes in intrathoracic pressure during CPR recently has been highlighted by studies of a new device called an inspiratory impedance threshold device (ITD). This helps to pump more blood back to the heart during cardiac arrest by enhancing negative intrathoracic pressure during the decompression phase of CPR, thereby enhancing blood return [6,9–11,14,15]. In view of the increased efficiency of CPR with the ITD, we hypothesized that the cardiopulmonary interactions associated with blood flow and ventilation may be improved further by reducing the frequency of ventilations when using the ITD. A reduction in the ventilation frequency would result in less overall positive intrathoracic pressure, enable more time for venous blood flow back to the heart and provide more time per minute for the ITD to increase circulation [6,7,10].

To test the hypothesis we investigated the effects of two different compression: ventilation ratios, with and without an ITD, on the coronary perfusion pressure, mean arterial pressure (MAP), and oxygenation. Compressions were performed continuously at a rate of 100 min^{-1}. The delivery of each breath was initiated during the decompression phase of CPR. To ensure that the chest wall was allowed to recoil fully during the decompression phase, an automatic compression-release device was used. The results support the hypothesis that fewer ventilations per minute improved hemodynamics during CPR. The physiological benefits of fewer ventilations per minute can be further enhanced using the ITD, without compromising oxygen delivery.

2. Materials and methods

The study was approved by the Committee of Animal Experimentation at the University of Minnesota. The animals received care in compliance with the 1996 Guide for the Care and Use of Laboratory Animals by the National Research Council in a facility that was accredited by the American Association for Accreditation of Laboratory Animal Care. Anaesthesia was used in all surgical interventions to avoid all unnecessary suffering. Experiments were performed by a qualified team. The study was performed on female farm pigs (26–31 kg).

2.1. Preparatory phase

The preparatory aspects of this study have been previously described [10]. Briefly, each animal received 7 ml (100 mg/ml) of intramuscular ketamine HCl (Ketaset®, Fort Dodge Animal Health, Fort Dodge, IA) for initial sedation. Propofol (PropoFlo®, Abbott Laboratories, North Chicago, IL) (2.3 mg/kg), was delivered initially as an intravenous

(IV) bolus via the lateral ear vein. While breathing spontaneously breathing, but sedated, the pigs were intubated with a 7.0 French tracheal tube. Additional propofol (1 mg/kg) was administered, followed by a propofol infusion of 160 μg/kg min.

Animals were positioned supine and unilateral femoral artery cannulation was performed under aseptic conditions. Central aortic blood pressures were recorded continuously, with a micromanometer-tipped catheter (Mikro-Tip® Transducer, Millar Instruments, Inc., Houston, TX). A central venous catheter was placed in the right internal jugular vein. All animals were treated with heparin, (100 units/kg IV) bolus, once catheters were in place. During the preparatory phase, animals were ventilated with room air using a positive pressure cycle ventilator, (Harvard Apparatus Co., Dover, MA), with a tidal volume of 450 cc and rate adjusted to maintain an arterial CO_2 at 40 mmHg and PaO_2 of >60 mmHg (oxygen saturation >90%), based upon analysis of arterial blood gases (IL Synthesis, Instrumentation Laboratory, Lexington, MA). Electrocardiographic monitoring was recorded continuously. Intrathoracic pressures (intratracheal) were measured continuously using a micromanometer-tipped catheter positioned 2 cm below the tip of the tracheal tube. Data were digitized by a digital recording system (Superscope II vl.295, GW Instruments, Somerville, MA) and a Power Macintosh G3® computer (Apple Computer, Inc., Cupertino, CA). End tidal CO_2 ($ETCO_2$) was recorded with a CO_2SMO Plus®, (Novametrix Medical Systems, Wallingford, CT). Intrathoracic pressure area was calculated by (1) extracting data from Superscope II over a 20 s during each min of CPR (2) transferring the numerical data to Microsoft Excel program and (3) using Rim and Sums method. The calculated area (mmHg × s) was multiplied by 3 to give the total minute intrathoracic pressure area. By summing only the negative values over time we calculated the total negative intrathoracic pressure on a per minute basis. Thus, the total negative intrathoracic pressure area is an analogue of the mean negative intrathoracic pressure over time. Coronary perfusion pressure was calculated as the difference between the diastolic (decompression phase) aortic pressure and right atrial pressure. Three measurements of diastolic aortic and right atrial pressure were made at each minute during CPR. These values were then averaged to generate the mean CPP value for the animal at each time point.

2.2. Experimental protocol

Once the surgical preparations were completed, oxygen saturation was >90% and $ETCO_2$ stable between 35 and 42 mmHg for 5 min, ventricular fibrillation (VF) was induced by delivering direct electrical current via a temporary pacing wire (Daig Division, St Jude Medical, Minnetonka, MN) positioned in the right ventricle. At that time a computer generated randomization list was used to determine the compression to ventilation ratio and whether or not the ITD would be used. Once VF was induced, the ventilator was disconnected from the ET tube and the dose of propofol was reduced to 100 mcg/kg min. Animals were randomized prospectively to receive CPR with a compression: ventilation ratio of either 5:1 or 10:1; with or without the ITD. After 6 min of untreated ventricular fibrillation, closed-chest standard CPR was performed with a pneumatically driven automatic piston device (Pneumatic Compression Controller, AMBU International, Glostrup, Denmark), as previously described (10). The compression rate was 100 min^{-1}, uninterrupted, with a 50% duty cycle, and a depth of 25% of the anterior-posterior diameter of the chest wall. The chest wall is allowed to recoil passively but completely with this device as the compression piston is actively pulled upwards off the chest after each compression. Pressure-controlled ventilation was performed with a semiautomatic ventilator (Demand valve model L063-05R, Life Support Products Inc., Irvine, CA, USA) using 100% oxygen at a constant flow rate (160 l/min). Ventilations were initiated synchronously with the decompression phase of CPR: approximately 400 cc was delivered with each breath. In addition, an ITD (CPRx LLC, Minneapolis, MN, USA) was attached to the ET tube in some animals based on the randomization schedule. The cracking pressure of the ITD was −15 cm of H_2O at a flow rate of 10 l/min. CPR was performed continuously for 6 min. Aortic pressure, right atrial pressure, intrathoracic pressure, $ETCO_2$ and O_2 saturation were measured continuously and sampled every minute. At the end of 6 min of CPR, animals were defibrillated with a monophasic defibrillator starting at 200 J × 3. If VF persisted, adrenaline (epinephrine) was administered at a dose of 45 mcg/kg and then three more dc shocks (200 J) were delivered. If VF still persisted all resuscitation efforts were terminated. When resuscitation was successful, animals were again ventilated with a positive pressure cycle Harvard ventilator at a rate of 12 breaths per minute with a tidal volume of 450 cc. No further interventions were performed after restoration of spontaneous circulation. Arterial and venous blood gases were collected before ventricular fibrillation was induced, after 4.5 min of CPR and 5, 20, 40, 60 min after resuscitation. Hemodynamic variables, blood gases, were monitored for an hour. At the end of the experiment, the animals were sacrificed using an intravenous bolus of propofol 60 mg and then 10 M KCl.

2.3. Statistical analysis

Values are expressed as mean ± S.E.M. The primary endpoint was coronary perfusion pressure. Additional measurements included mean arterial pressures, intrathoracic pressure, arterial oxygen saturation and arterial partial pressure of oxygen. The comparability of weight and baseline data was tested with the *t*-test for continuous variables. A two-factor ANOVA with repeated measures on one factor was used to determine statistical significance between groups. Regression analysis was performed between coro-

nary perfusion pressure and the negative minute intrathoracic area.

3. Results

A total of 32 pigs randomized to four equal groups: group A received CPR with a compression:ventilation (C/V) ratio of 5:1, group B with a C/V ratio of 5:1 plus the ITD, group C with a C/V ratio of 10:1 and group D with a C/V ratio of 10:1 plus the ITD. Before induction of cardiac arrest, there were no statistically significant differences in weight, temperature, hemodynamic variables and arterial blood gases between the groups (Table 1).

Intrathoracic pressures during the decompression phase were significantly lower and the magnitude of pressure change during the compression-decompression cycle was potentiated markedly in groups B and D animals treated with the ITD compared with animals in groups A and C (Fig. 1, Table 2). Animals in groups C and D had a greater amount of negative intrathoracic pressure for over time.

As shown in Fig. 2, the coronary perfusion pressure, calculated as the diastolic difference between aortic and right atrial pressures, in group A was significantly lower than coronary perfusion pressures observed in groups B–D. In addition, the coronary perfusion pressure measured in group D was significantly higher than those measured in groups A, B, and C.

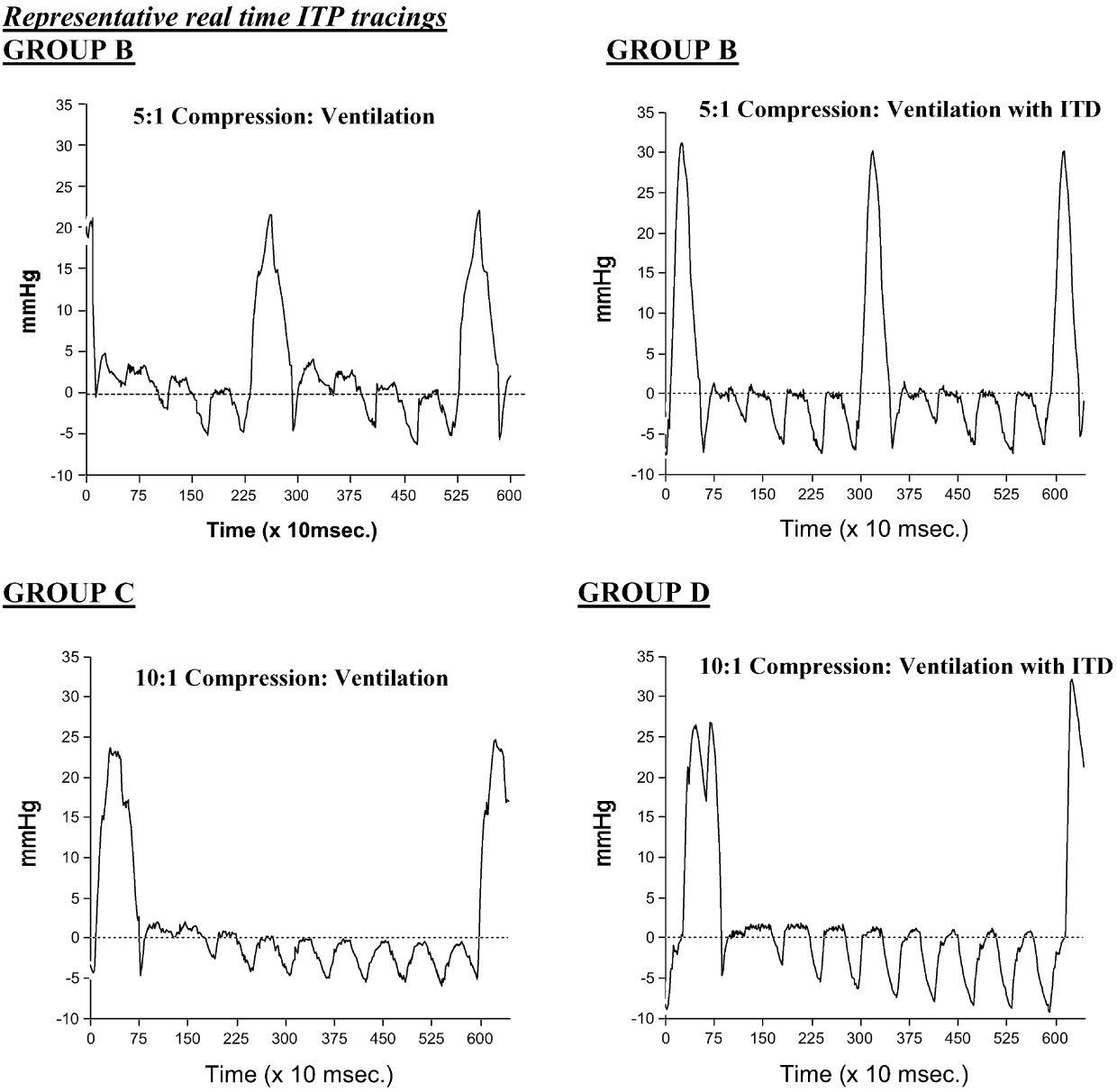

Fig. 1. Four representative real time tracings of intrathoracic pressure (one from each group) during minute 4 of CPR. The total amount of time that the intrathoracic pressure is less than zero during the chest wall recoil phase is greater with less frequent ventilations and with use of the ITD.

Table 1
There were no statistical significant differences in baseline physiological values of weight, temperature (temp), systolic blood pressure (SBP), diastolic arterial pressure (DAP), right atrial pressure (RAP), coronary perfusion pressure (CPP)

Group	Weight	Temp	SBP	DBP	RAP	CPP
A (5:1)	28.3 ± 1	37.7 ± 0.4	87 ± 4	67 ± 3	2 ± 0.4	65 ± 3
B (5:1 + ITD)	28.6 ± 1.7	37.1 ± 1.1	94 ± 8	76 ± 9	3 ± 1.4	73 ± 8
C (10:1)	29.1 ± 1.2	37.8 ± 0.7	88 ± 4	67 ± 3	1 ± 0.7	66 ± 4
D (10:1 + ITD)	29.4 ± 0.7	37.5 ± 0.6	96 ± 7	76 ± 8	2 ± 0.7	74 ± 8

$P > 0.4$.

Table 2
Hemodynamic values measured after 4 min of CPR, expressed as mean ± S.E.M.

Group	Negative ITP area	MAP	SBP	DBP	RAP	CPP
A (5:1)	60 ± 8.5*	27 ± 1.5*†	44 ± 3.4*†‡	16.4 ± 1	4 ± 1	14 ± 1*
B (5:1 + ITD)	69 ± 9†	37.5 ± 3.6†	68 ± 7†	21 ± 2.5	3 ± 1	19 ± 3
C (10:1)	82 ± 11.6	33 ± 1.8	55 ± 8.2‡	18 ± 1	1.8 ± 0.5	17.8 ± 1.2
D (10:1 + ITD)	102 ± 12*†	37 ± 2.9*	65 ± 6.2*	23 ± 1.8	1.8 ± 1.2	23.5 ± 3*

Right atrial pressure (RAP), systolic blood pressure (SBP), diastolic blood pressure (DBP), mean arterial blood pressure (MAP), coronary perfusion pressure (CPP), negative intra thoracic pressure min area (Neg ITP). Area expressed in mmHg × s and pressures in mmHg. (*, †, ‡) Mean statistical significance between groups ($0.03 > P > 0.002$).

The mean arterial pressure (MAP), calculated as 2/3 diastolic blood pressure plus 1/3 systolic pressure, was significantly higher in group B–D versus group A (Fig. 3). There were no differences in arterial blood gas values between groups at baseline, before induction of ventricular fibrillation. Arterial pH, measured after 4.5 min of CPR (min 4.5) was significantly lower ($P < 0.01$) in groups B–D when compared with group A (Table 3). $PaCO_2$ during CPR was correspondingly significantly higher ($P < 0.01$) in groups B–D when compared with group A. There were no differences in base deficit between groups: during the 4th minute of CPR the mean values were −10.8, −10.3, −10.2, −10.5 for groups A–D respectively. In addition, $ETCO_2$ values were higher in groups B–D compared with group A ($P < 0.01$). Oxygenation was adequate in all groups and there were no statistically significant differences between groups ($P > 0.3$) (Table 3).

Fig. 4 demonstrates the relationship between negative intrathoracic pressure and coronary perfusion pressure. There was a linear relation ($R^2 = 0.73$) between negative intrathoracic pressures, calculated on a per minute basis, and the coronary perfusion pressures.

Return to spontaneous circulation was achieved in all animals treated with the ITD. There were 4 deaths in 16 animals in the non ITD groups. Adrenaline requirements needed for return of spontaneous circulation were significantly higher for the group A versus groups B–D (Table 4) ($P < 0.01$).

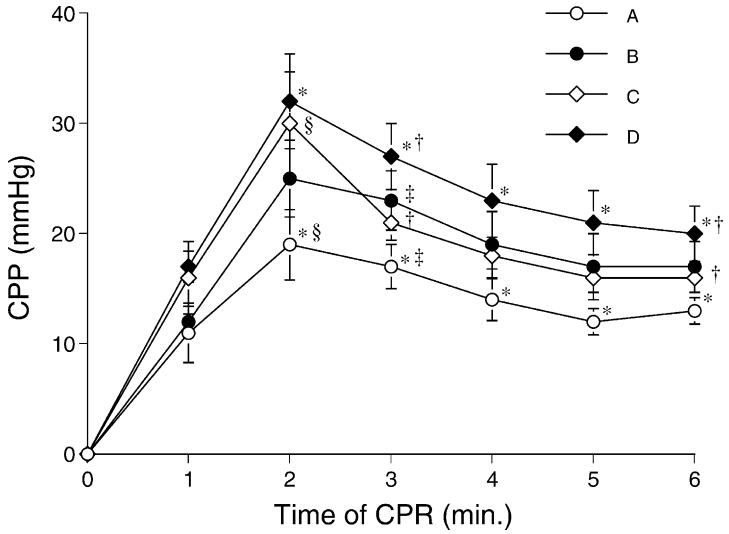

Fig. 2. Coronary perfusion pressure (CPP) during CPR. Mean ± S.E.M. coronary perfusion pressure during CPR in groups A–D. Asterisk (*) signifies a significant difference between groups A and D ($0.03 > P > 0.001$, (§) between groups A and C ($P < 0.05$), (†) between groups C and D ($P < 0.05$), (‡) between A and B ($P < 0.05$).

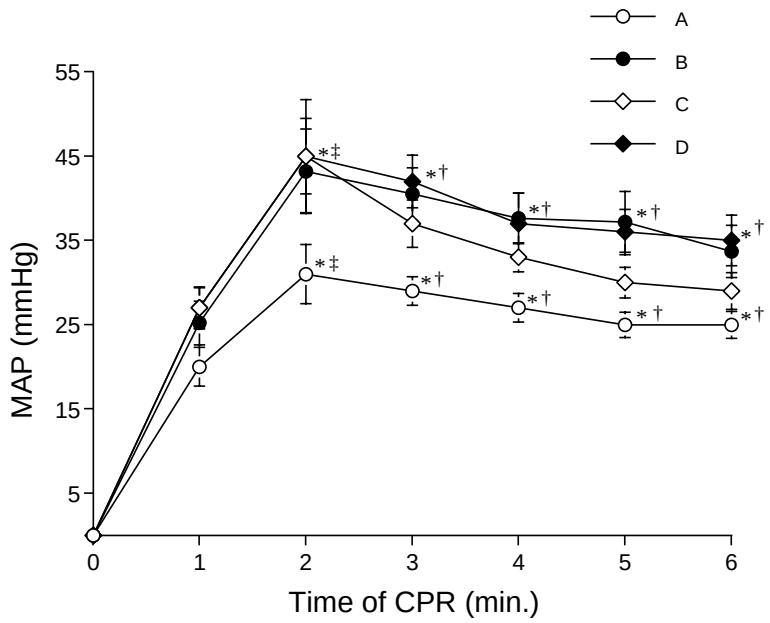

Fig. 3. Mean arterial pressure (MAP) during CPR. Mean ± S.E.M. mean arterial pressure during CPR in groups A–D. Asterisk (*) signifies a significant difference between groups A and D ($P < 0.03$), (†) between groups A and B ($P < 0.05$), (‡) between groups A and C ($P < 0.05$).

Table 3
Arterial blood gases and end tidal CO_2 ($ETCO_2$) values during CPR after 4.5 min of CPR

Group	Arterial Blood at 4.5 min of CPR			$ETCO_2$
	pH	pCO_2	pO_2	
A	7.4 ± 0.03*†§	21.3 ± 2.07*†§	259.4 ± 43.3	20 ± 2.7*
B	7.27 ± 0.03*‡	35.9 ± 4.5*	181.1 ± 47.7	32.6 ± 5.1†
C	7.25 ± 0.02†	34.9 ± 1.9†	257.6 ± 41.8	30.4 ± 1.6§
D	7.19 ± 0.02§‡	41.6 ± 3.4§	242 ± 27.1	46.1 ± 6.9*†§

There were significant differences between groups A and B–D. After return of spontaneous circulation the blood gases of the survivors normalized over 20 min and there were no differences up to an hour. Mean pH, $PaCO_2$, PaO_2, and base deficit values at 60 min post ROSC were: ($A = 7.41, 32.9, 67.9, -3$), ($B = 7.43, 30, 77, -3.2$), ($C = 7.44, 31, 77, -2.4$), ($D = 7.43, 31.8, 73, -2.4$), respectively. (*, †, §, ‡) Statistical significance between groups with a P-value <0.05.

Table 4
Survival and adrenaline requirements between groups

Group	Number of pigs	Successful ROSC		Animals that received adrenaline		Animals that required adrenaline for successful ROSC	
		Yes	No	Yes	No	ROSC + EPI	ROSC, no adrenaline
A	8	6	2	6*	2	4*	2
B	8	8	0	3	5	3	5
C	8	6	2	3	5	1	5
D	8	8	0	3	5	3	5

Asterisk (*) signifies statistical significance between groups A and B–D for the adrenaline requirements ($P = 0.0025$). ROSC: return of spontaneous circulation and perfusing rhythm.

4. Discussion

Cardiopulmonary interactions during CPR are influenced by the ratio of chest compressions to ventilations and the degree of negative intrathoracic pressure achieved during chest wall recoil. The results of this study demonstrate that both a reduction in the frequency of ventilation and use of an ITD enhance the decompression phase vacuum created by the chest wall recoil. This results in a significant improvement in the efficiency of CPR and in survival rates [10,14].

At present, the international guidelines recommends a C/V ratio of 5:1 for patients undergoing two-rescuers CPR in whom the airway has been secured by tracheal intubation or a similar means. Group A animals received this type of CPR. However, when CPR was delivered with C/V ratio of

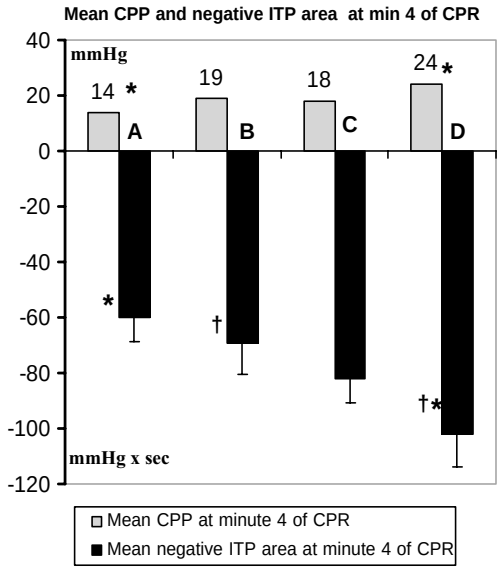

Fig. 4. Relationship between coronary perfusion pressure (mmHg) and negative minute intrathoracic pressure (ITP) (mmHg × s). Coronary perfusion is influenced by the amount of time that the pressure within the thorax remains negative and on the magnitude of ΔP during each compression-decompression cycle. The linear relationship between coronary perfusion pressure and minute negative ITP has a correlation coefficient of 0.73 (R^2). (*, †) Mean statistical significance between groups with $P < 0.01$.

10:1, there was a significantly improvement in coronary perfusion pressure and mean arterial pressure. This was associated with an increase in the negative intrathoracic pressure generated within the thorax by the recoil of the chest over each minute of time. Moreover, use of the ITD resulted in a further increase in coronary perfusion pressure and mean arterial pressure, without any compromise in oxygenation. In addition, these data demonstrate that there are no observable adverse physiological consequences of reducing the ventilation rate and using the ITD concurrently in this animal model of cardiac arrest. It remains unknown, at present, whether a further reduction in the ventilation rate, with or without the ITD, will further enhance the efficiency of CPR.

These data are consistent with those of others demonstrating that less frequent ventilations result in higher vital organ perfusion pressures [3,4,7,12,13] In the current study we observed that the generation of negative intrathoracic pressure, measured on a per minute basis, resulted in higher coronary perfusion pressures and mean arterial pressures.

The greater the negative intrathoracic pressure, on a per minute basis, the greater opportunity for venous return over each compression-compression cycle. These data suggest that efforts to improve CPR efficiency should focus on ways to optimize the frequency of ventilation as well as to reduce tidal volume and peak airway pressures in order to optimize coronary perfusion pressure, mean arterial pressures and tissue oxygenation during CPR.

In our study the arterial blood pH in group A was significantly higher when compared to the other groups. This is best explained by a pseudo-respiratory alkalosis (as the low $PaCO_2$ suggests) as a result of the low arterial blood flow state and higher ventilation rate. When cardiac output, arterial pressures and tissue perfusion improved with the interventions in groups B–D the arterial blood became severely acidotic, and the arterial pH was reduced being more reflective of the actual tissue acid–base state.

This study was not designed to be powered statistically to study survival rates or the need for adrenaline. Nonetheless, there was statistical significance between animals treated with the currently recommendation 5:1 C/V ratio in group A and all the other groups in terms of the need for adrenaline to achieve return of spontaneous circulation.

The higher need for adrenaline in group A animals support the hypothesis that animals treated with the international 5:1 C/V ratio also were more severely damaged due to less efficient CPR. There are several other limitations of this study as well. We did not measure tissue perfusion or blood flow directly. Surrogates were used for these endpoints. However, based on previous work, that showed that a 30% (5 mmHg) increase in coronary perfusion pressure resulted in a 100% increase of left ventricular blood flow, we can only assume that the increase of 10–15 mmHg of coronary perfusion pressure achieved with a ratio of 10:1 and use of an ITD during CPR significantly enhanced myocardial flow and the potential for survival [10,16]. Finally, only two different C/V ratios were studied. Further work is needed to explore the impact of even lower ventilation rates.

5. Conclusions

This study supports the hypothesis that the efficiency of cardiopulmonary resuscitative measures can be improved by optimizing the cardiopulmonary interactions that regulate vital organ blood flow during CPR. A reduction in the frequency of ventilations resulted in a lower intrathoracic pressure during CPR and this contributed to the increase in arterial and coronary perfusion pressures. Addition of an inspiratory impedance device resulted in a further decrease in intrathoracic pressures during chest wall recoil and further augmented arterial and coronary pressures without compromising oxygenation. Based upon this animal study, clinical studies should be performed to determine if the efficiency of CPR in patients can be improved with these straight forward changes in CPR protocols. The higher coronary perfusion pressure, mean arterial pressure and end-tidal CO_2 levels observed with the lower ventilation rates and use of the ITD all support the hypothesis that cardio-pulmonary manoeuvres that increase the total amount of negative intrathoracic pressure on a per minute basis and augment the changes (ΔP) of intrathoracic pressures during compression–decompression cycle, improve vital organ perfusion during CPR by optimizing the chest wall/heart pump mechanics. Finally, based upon this study, the importance of maintaining a low ventilation frequency needs to be reemphasized during the teaching

of CPR to optimize venous return to the heart after each chest wall compression, with or without concurrent use of the ITD.

Disclosure

Keith G. Lurie is a co-inventor of the inspiratory impedance threshold device and active compression decompression CPR technology and founded a company, CPRx LLC, to develop this device. There are no other conflicts of interest.

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