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Experimental paper

A tourniquet assisted cardiopulmonary resuscitation augments myocardial perfusion in a porcine model of cardiac arrest☆,☆☆

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ABSTRACT

Objective: During cardiopulmonary resuscitation (CPR), myocardial blood flow generated by chest compression rarely exceeds 35% of its normal level. Cardiac output generated by chest compression decreases gradually with the prolongation of cardiac arrest and resuscitation. Early studies have demonstrated that myocardial blood flow during CPR is largely dependent on peripheral vascular resistance. In this study, we investigated the effects of chest compression in combination with physical control of peripheral vascular resistance assisted by tourniquets on myocardial blood flow during CPR.

Methods: Ventricular fibrillation was induced and untreated for 7 min in ten male domestic pigs weighing between 33 and 37 kg. The animals were then randomized to receive CPR alone or a tourniquet assisted CPR (T-CPR). In the CPR alone group, chest compression was performed by a miniaturized mechanical chest compressor. In the T-CPR group, coincident with the start of resuscitation, the thin elastic tourniquets were wrapped around the four limbs from the distal end to the proximal part. After 2 min of CPR, epinephrine (20 μg/kg) was administered via the femoral vein. After 5 min of CPR, defibrillation was attempted by a single 150 J shock. If resuscitation was not successful, CPR was resumed for 2 min before the next defibrillation. The protocol was continued until successful resuscitation or for a total of 15 min. Five minutes after resuscitation, the elastic tourniquets were removed. The resuscitated animals were observed for 2 h.

Results: T-CPR generated significantly greater coronary perfusion pressure, end-tidal carbon dioxide and carotid blood flow. There was no difference in both intrathoracic positive and negative pressures between the two groups. All animals were successfully resuscitated with a single shock in both groups. There were no significant changes in hemodynamics observed in the animals treated in the T-CPR group before-and-after the release of tourniquets at post-resuscitation 5 min.

Conclusions: T-CPR improves myocardial and cerebral perfusion during CPR. It may provide a new and convenient method for augmenting myocardial and cerebral blood flow during CPR.

1. Introduction

The main goal of cardiopulmonary resuscitation (CPR) is to provide forward blood flow to the heart and brain until spontaneous circulation is restored.[1] Both experimental and clinical studies have consistently demonstrated that the success of resuscitation during CPR is largely dependent on the efficacy of cardiac output generated by chest compression.[2–5] However, cardiac output and myocardial perfusion generated by conventional CPR rarely exceeds 30% and 50% of normal levels, respectively.[6,7] In addition, cardiac output gradually decreases during prolonged CPR in spite

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of continuous precordial compression.[8] Therefore, global myocardial ischemia is persistent during the conventional resuscitation effort.[9,10] Coronary perfusion pressure (CPP), the most reliable predictor of the success of defibrillation and restoration of spontaneous circulation, is highly correlated with coronary blood flow and, therefore, myocardial perfusion.[11,12] Early investigations have demonstrated that the magnitude of myocardial perfusion during CPR is highly dependent on peripheral vascular resistance, which can be enhanced by vasopressor drugs such as epinephrine or the compression and binding of the abdomen.[13–20]

However, our previous study has demonstrated that epinephrine significantly increases the severity of post-resuscitation myocardial dysfunction. This is a result from its β-adrenergic effect during CPR.[21] Two clinical studies further demonstrated that administration of epinephrine during resuscitation compromises the outcomes of CPR.[22,23] In addition, several studies in the early 1980s have shown disappointing results of hemodynamics and survival on the effects of physical control of peripheral vascular resistance implemented by military anti-shock trousers (MAST) or abdominal binder (AB).[24,25] However, in their studies, the detrimental effects including impediment of ventilation, inadequate gas exchange, proportionate increase in diastolic right atrial pressure (RAP) and intrathoracic pressure (ITP) may be due to the increase in intra-abdominal pressure and the limited movement of diaphragm during utilization of MAST or AB.

An ideal method to increase peripheral vascular resistance and therefore improve the perfusion to vital organs during CPR should be simple, safe and noninvasive. An Esmarch tourniquet is a constricting or compressing device, specifically a bandage, used to control venous and arterial circulation to an extremity. It has been used routinely to limit or decrease blood flow in surgery. We developed a method to physically control the peripheral vascular resistance by tightening the elastic tourniquets from distal to proximal around the four limbs.

In the present study, we compared the effects of a tourniquet assisted CPR (T-CPR) on myocardial perfusion during CPR in a porcine model. We hypothesized that T-CPR would improve myocardial perfusion during CPR.

2. Materials and methods

All animals received humane care in compliance with the "Principles of Laboratory Animal Care" formulated by the National Society for Medical Research and the Guide for the Care and Use of Laboratory Animals prepared by the Institute of Laboratory Animal Resources and published by the National Institutes of Health (8th edition; Washington, DC, National Academic Press, 2011). The protocol was approved by the Institutional Animal Care and Use Committee of the Weil Institute of Critical Care Medicine.

2.1. Animal preparation

Ten male domestic pigs weighing 34 ± 2 kg were fasted overnight except for free access to water. Anesthesia was initiated by intramuscular injection of ketamine (20 mg/kg), followed by intravascular injection with sodium pentobarbital (30 mg/kg). When the animals awakened or showed signs of restlessness, an additional dose of sodium pentobarbital (8 mg/kg) was injected, or at intervals of approximately 1 h to maintain anesthesia, if necessary. A cuffed endotracheal tube was advanced into the trachea and the animals were mechanically ventilated with a volume controlled ventilator (Model MA-1, Puritan-Bennett, Carlsbad, CA) with a tidal volume of 15 ml/kg, peak flow of 40 L/min, and F_{iO2} of 0.21. End-tidal carbon dioxide ($ETCO_2$) was monitored with an infrared capnometer (NPB-70, Nellcor Puritan Bennett Inc., Pleasanton, CA).

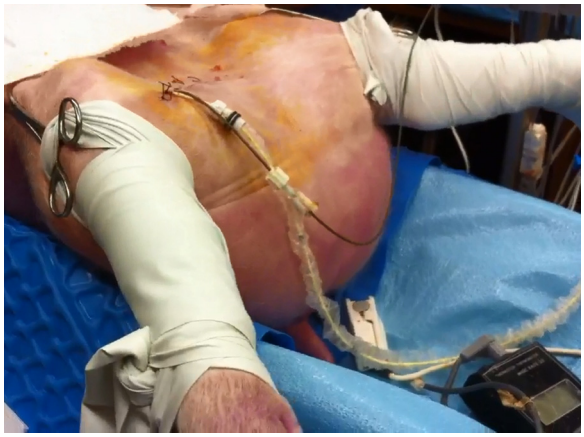

Fig. 1. A sketch of physical control of peripheral vascular resistance by tightening the elastic tourniquets from distal to proximal around the four limbs.

Respiratory frequency was adjusted to maintain $ETCO_2$ between 35 and 40 mm Hg before cardiac arrest. For recording electrocardiogram (ECG), three adhesive electrodes were applied to the shaved skin of the proximal right-and-left, upper-and-lower limbs. For the measurement of aortic pressure and the collection of the blood samples, a 5 F transducer-tipped Millar catheter (Model SPC-450S, Millar Instruments Inc., Houston, TX) was advanced from the right femoral artery into the thoracic aorta. For the measurements of RAP and core blood temperature, a 7 F catheter (Abbott Critical Care, Salt Lake City, UT) was advanced from the right femoral vein and directed into the right atrium. Both catheters were flushed intermittently with saline containing 5 IU bovine heparin per ml. For inducing ventricular fibrillation (VF), a 5 F pacing catheter (EP Technologies Inc., Mountain View, CA) was advanced from the right external jugular vein into the right ventricle. Carotid blood flow (CBF) was continuously measured with the aid of a flow probe (Ultrasonic Blood Flow Meter, T420, Transonic Systems Inc, Ithaca, NY) positioned around the right common carotid artery. The position of all catheters was confirmed by characteristic pressure morphology and with fluoroscopy. For the measurement of ITP, an additional 5 F Millar catheter was advanced from the incisor teeth into the esophagus for a distance of 35 cm. The piston of the compressor was positioned in the midline at the level of the fifth interspace. Body temperature was maintained at 37.5 ± 0.5 °C with the aid of a cooling/warm blanket (Blanket ROL, Cincinnati Sub-Zero Products, Cincinnati, OH) throughout the entire experiment.

2.2. Experimental procedures

Fifteen minutes prior to inducing cardiac arrest, baseline measurements were obtained. The animals were then randomized by the *Sealed Envelope Method* to receive CPR alone or T-CPR. Cardiac arrest, due to VF, was induced by 1 mA alternating current through a 5 F pacing catheter and into the right ventricular cavity. Mechanical ventilation was discontinued after the onset of VF. Prior to initiating the resuscitation procedure, the pacing catheter was withdrawn to avoid heart injury during chest compression. After 7 min of untreated VF, CPR was performed. The mechanical chest compressor (MCC) was programmed to provide 100 compressions-per-minute. The compression depth was adjusted to decrease the anterior–posterior diameter of the chest by 25%. For the T-CPR group, during fibrillation, thin elastic Esmarch tourniquets were simultaneously wrapped around the four limbs from the distal end to the proximal part as tight as possible (Fig. 1). Coincident with the start of precordial compression, all animals were mechanically ventilated with a tidal volume of 15 mL/kg and F_{iO2} of 1.0, with