Review of resuscitation physiology in children

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Abstract

More than one quarter of children survive to hospital discharge after in-hospital cardiac arrests, and 5—10% of children survive to hospital discharge after out-of-hospital cardiac arrests. Cardio-pulmonary resuscitation (CPR) differs in children from adults. Following the Airway, Breathing, Circulation format, this article reviews the physiology of paediatric cardio-pulmonary resuscitation. It addresses the appropriate interventions during CPR, mechanisms of action of commonly used drugs and special resuscitation circumstances: premature and newly born infants, traumatic cardiac arrest, and ECMO (Extracorporeal Membrane Oxygenation). New exciting discoveries in resuscitation science postulate that the key factor in improving outcomes of paediatric cardiac arrest is improving the quality of interventions. A thorough understanding of the physiology underpinning CPR is helpful in ensuring optimal delivery of CPR in children and improving clinical outcomes.

Keywords cardio-pulmonary resuscitation; child; heart arrest; paediatric advanced life support; physiology

Paediatric cardiac arrest

Cardio-pulmonary resuscitation (CPR) differs in children from adults owing to differences in the aetiology of cardiac arrest. In adults, cardiac arrest is commonly caused by sudden ventricular fibrillation (VF) on a background of coronary artery disease with myocardial ischaemia. Paediatric cardiac arrests are commonly due to respiratory failure and/or circulatory shock, arising from progressive tissue hypoxia and acidosis of the underlying illness. These pathologies may intrinsically be more reversible than established diseases of adults. Moreover, CPR in children results in better myocardial and cerebral blood flows owing to their compliant and less circular chest walls.

Physiology of CPR

Airway

Airway obstruction can be partial or complete and may be multifactorial (i.e. central nervous system depression coupled with blood or vomitus in the upper airway). The immediate clinical priority is to ensure that the airway remains patent, if necessary securing the airway with a cuffed endotracheal tube.

Breathing

Cardiac arrest patients are frequently unintentionally hyperventilated during CPR. During cardiac arrest, cardiac output and pulmonary blood flow are reduced to 10—25% of normal. Therefore much less ventilation to achieve adequate carbon dioxide clearance during CPR.

Hyperventilation leads to hypocarbia which in turn decreases coronary and cerebral perfusion pressures during resuscitation efforts and may worsen survival. Moreover, positive pressure ventilation either via an endotracheal tube or mask may prohibit the development of negative intra-thoracic pressure during chest wall recoil, inhibiting venous blood return to the right heart and thereby decreasing the haemodynamic effectiveness of CPR.

Of course, there are important differences between adults and children. In sudden VF cardiac arrest, which occurs more commonly in adults, aortic concentrations of oxygen and carbon dioxide remain almost equal to pre-arrest levels. This is because there is minimal blood flow before commencement of CPR, and therefore, aortic oxygen consumption is minimal. During CPR, the adult lungs act as reservoir for oxygen, and hence, adequate ventilation and oxygenation can continue without the need for rescue breaths. In cardiac arrests caused by asphyxia/ischaemia, which are the most common aetiology of paediatric cardiac arrests, blood continues to flow to the tissues. The underlying disease process causes oxygen saturations to decrease and this is accompanied with rise in circulatory levels of carbon dioxide and lactate, which in turn leads to severe hypoxaemia and acidaemia prior to CPR. Rescue breaths are paramount in successful resuscitation of cardiac arrests caused by asphyxia/ischaemia.

To counter the hypoxaemia high flow oxygen should be administered during the initial resuscitation and reviewed with return of spontaneous circulation (ROSC) to minimise reperfusion and hyperoxaemia injury.

Current Resuscitation Council UK guidelines (2010) recommend a compression/ventilation ratio of 15:2. However, the ideal ratio of ventilations to compressions in paediatric CPR is not established. The ratio for chest compressions and ventilations relies on several factors, including tidal volume, compression rate, volume of blood flow generated by compressions, and the duration of time that compressions were interrupted to perform ventilations. Recent studies on mannequin-simulated paediatric CPR showed equal minute ventilation in chest compression/ventilation ratio of 15:2 and in compression/ventilation ratio of 5:1. However there was an increase of 48% in the number of chest compressions delivered with the 15:2 ratio.

Continuous chest compressions maintain adequate coronary perfusion pressure during CPR, and interrupting these compressions will cause the coronary perfusion pressure to drop rapidly. Increasing the ratio of compressions to ventilations minimizes interruptions and consequently, minimizes drops in coronary perfusion pressure during CPR. This was demonstrated in a recent study, which evaluated a protocol change from the recommended compression/ventilation ratio of 15:2 to 30:2 during CPR. However, another study hypothesized that an increased compression/ventilation ratio will result in poor compression

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depth and pressure and increased rescuer fatigue during adolescent, child and infant manikin CPR.

Circulation

Normally, the myocardium receives its blood supply during diastole from the coronary arteries, which originate from the root of the aorta. During cardiac arrest, closed-chest manual cardiac massage relies on the natural elasticity of the chest to generate a transient period of negative intra-thoracic pressure immediately after maximal compression, improving venous return. This is called the 'thoracic pump theory'. It is believed that the effects of direct compression of the heart, causing blood to be pushed out and pulled in during recoil, is less important overall but rather that the heart acts merely as a conduit during CPR in cardiac arrest.

The pressure gradient between the right atrium and the aorta, which is created during the relaxation phase of CPR is the coronary perfusion pressure, which is correlated positively with ROSC.

The three factors that affect overall cardiac output during cardiac massage, as a result of altering intra-thoracic pressure, are: the rate, the depth and chest recoil. There have been a number of human and animal studies investigating these factors, which have resulted in the phrase "Push hard and fast and allow for full chest recoil". A fourth factor, which obstructs good cardiac output in CPR, already mentioned above, is hyperventilation. All of these, when optimised generate the most effective intra-thoracic pressure gradient to allow for good cardiac output.

Therefore, in children and infants, the chest wall must be compressed up to one third of the depth and at a rate of 100/minute (no more than 120/minute) with full recoil in between each one. It is vital that the rescuer gets help to avoid fatigue and suboptimal closed-chest cardiac massage.

New technologies may help to improve the efficiency of CPR. The Impedance Threshold Device (ITD) is a relatively new device, which enhances the changes in intra-thoracic pressures during cardio-pulmonary resuscitation (CPR). This effect is achieved by preventing the passive inflow of air into the chest during chest recoil between chest compressions without impeding active ventilation.

During CPR, the ITD works effectively with either an endotracheal tube or facemask. The ITD is equipped with timing lights that flash 10 times per minute, prompting rescuers to ventilate at the proper rate and avoid haemodynamically detrimental and potentially harmful hyperventilation.

Studies on ITD have shown that negative intra-thoracic pressure is enhanced and this augmentation of negative intra-thoracic pressure during CPR improved vital organ perfusion and myocardial blood flow. ITD doubles both blood flow to the heart and blood pressures, and improves circulation to the brain.

The significance of intra-thoracic pressure in CPR haemodynamics has been highlighted by recent studies of active compression–decompression devices. Several studies found that short-term survival improved significantly with active compression–decompression (ACD) CPR as compared with standard CPR. With this method, the use of a hand-held suction device during active chest-wall decompression actively decreases intra-thoracic pressure, thereby enhancing venous blood return. Then, during compression, more blood is propelled out of the thorax. Consequently, active compression–decompression CPR provides greater perfusion of vital organs than does standard CPR. Combined use of ACD and ITD is superior in improving coronary perfusion pressure in cardiac arrest, than with either device alone. This combined usage causes a more rapid decrease in negative intra-thoracic pressure.

Although ITDs were recommended in the American Heart Association's (AHA) guidelines in 2005 as a CPR device to improve haemodynamics, subsequent reports indicated improvement to ROSC and short term survival but insufficient data showing any impact on survival to discharge. It has therefore been removed from the AHA and ERC resuscitation guidelines. There remains little research into the use of ITDs in paediatric CPR.

Medications used during CPR

Adrenaline

During CPR, adrenaline enhances cerebral and myocardial blood flows, by increasing peripheral vasoconstriction, and thus elevating the perfusion pressure for these organs. This α-adrenergic effect causes backpressure from the rest of the systemic arterial circulation on the cerebral and coronary arteries i.e. increased after-load pressure. Additionally, adrenaline has a less important β-adrenergic effect, which increases myocardial contractility and heart rate, and relaxes smooth muscles in the skeletal muscle bed and in the bronchi.

Studies showed that high-dose of adrenaline does not improve survival, and in one study, it was associated with unfavourable neurologic outcome. Hence 1:10,000 ml/kg of adrenaline is given in cardiac arrest, and not higher doses. If needed, doses are simply repeated every 3–5minutes.

Vasopressin

By acting on V1 receptors, vasopressin causes peripheral vasoconstriction, and by acting on the V2 receptors it leads to reabsorption of water in the renal tubule. Administration of vasopressin causes increased cerebral blood flow and oxygenation, and raises blood flow to other vital organs. In both a meta-analysis and systemic review of randomized controlled trials (RCT) including in-hospital and out-of-hospital arrests in adults, vasopressin had comparable efficacy to adrenaline. However, vasopressin may have shown improved long-term outcome in asystole patients. Further research is necessary to assess the efficacy of vasopressin use in conjunction with adrenaline in paediatric cardiac arrest.

Fluids

Fluids used during resuscitation: intravenous fluids are used during resuscitation to improve haemodynamics, by restoring the intra-vascular volume relative to the vascular space and optimizing ventricular preload. Fluid resuscitation is essential in the therapy of shock, even cardiogenic shock, although in this situation a more cautious approach with smaller boluses and a paediatric volume of total fluid is indicated.

i) Crystalloids: crystalloid solutions do not contain large protein molecules that are found in the plasma, i.e. colloid composed of proteins found in the blood. Therefore, when isotonic crystalloid solutions are given, the maximal amount of fluid that remains in the intra-vascular space is 30% of the initial volume given, with