Improving Neonatal Cardiopulmonary Resuscitation Hemodynamics:
Are Sustained Inflations During Compressions the Answer?

Running title: Wyckoff; Will sustained inflations improve neonatal CPR?

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Chest compressions are rarely needed in newly born infants with an estimated occurrence of 0.08% for near-term and term deliveries; however, the consequences of a hypoperfused state on the brain and other organ systems can be devastating. Newborns that require prolonged cardiac compressions with no signs of life beyond 10 minutes are at risk for exceptionally poor outcomes with up to 83% mortality and 77% severe disability noted in survivors. Although initiation of therapeutic hypothermia appears to have some advantage for cardiopulmonary resuscitation (CPR) recipients who survive the delivery room, optimization of the hemodynamics of neonatal cardiac compressions during CPR remains critical.

The infrequent use of cardiac compressions for newborns in the delivery room has impeded the design and completion of rigorous investigations to determine the most effective neonatal cardiac compression methodologies. The unique physiology of the newborn with initially fluid filled alveoli, the need to transition from fetal to newborn circulation, the open ductus arteriosus and the frequent presence of severe asphyxia as the cause of cardiovascular collapse add dimensions that cannot be accurately tested in adult or even pediatric patients or models. Adults most frequently receive CPR for cardiac etiologies such as ventricular fibrillation. Immediately following cardiac arrest, the aortic oxygen and carbon dioxide concentrations are close to the pre-arrest state. With initiation of CPR, the blood perfusing the coronaries has adequate oxygenation and pH. Thus, the problem in cardiac arrest is primarily the lack of blood flow rather than the content of the blood. For this reason, resuscitation algorithms for cardiac arrest focus predominantly on providing continuous, uninterrupted compressions with much less emphasis on ventilation. Adequate oxygenation and ventilation can continue without rescue breathing for several minutes because of chest compression-induced gas exchange and spontaneous gasping during compressions in victims of sudden cardiac arrest. This is quite
different from resuscitation following asphyxia-induced arrest. Animal models of asphyxia clearly demonstrate that a combination of chest compressions and ventilations generates better outcomes and survival than resuscitation with ventilations or compressions alone\textsuperscript{8,9} particularly during prolonged resuscitation\textsuperscript{10}. During asphyxia, blood continues to flow although with ever increasing hypoxemia, hypercarbia and acidemia until the heart runs out of energy substrate and becomes severely bradycardic and eventually asystolic. It is not surprising then that ventilation is essential to reversing asphyxial arrest.

The recent work by Schmolzer and colleagues\textsuperscript{11} appearing in this issue of Circulation uses a neonatal model of hypoxia/asphyxia to investigate new ways of enhancing blood flow during neonatal CPR while maintaining adequate ventilation. Although not a perfect model given the animals already had post-transitional physiology, the biochemical profiles closely replicates the severe mixed respiratory and metabolic acidemia seen in newborns who receive CPR in the delivery room\textsuperscript{1}. The use of randomization and \textit{a priori} power analysis add real strength to the study design. The use of sustained inflation to enhance intrathoracic pressure and thus antegrade blood flow while delivering compressions is novel in the neonatal resuscitation field. The fact that blood flow was substantially enhanced in critical vascular beds while simultaneously improving minute ventilation via the compressions themselves is especially intriguing and deserves further investigation. The use of the sustained inflation resulted in less supplemental oxygen exposure (a source of subsequently detrimental oxygen free radicals) and less epinephrine to achieve return of spontaneous circulation, both highly laudable goals. Future studies will need to determine if the improved hemodynamics and gas exchange hold true in the extreme of asphyxia-induced asystole and in a truly transitioning model with fluid filled alveoli and an open ductus arteriosus. Information regarding histologic lung injury and rates of
pneumothorax are lacking and deserve close attention. The lowest peak inspiratory pressure sustained inflation that maintains improved hemodynamics while minimizing lung injury should be determined.

Although more information is needed before this technique can be translated to the clinical arena, Schmolzer et al. provide critical information regarding a novel change in delivery of CPR that may provide an answer to that long vexing conundrum of how to improve perfusion without sacrificing essential ventilation during asphyxia-induced cardiovascular collapse of the newborn. Such an answer could be the key to improving the often devastating outcomes following neonatal CPR.

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References:


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