o_two controlled™ ventilation

Review/Update

The Haemodynamic Effects of Inadvertent Hyperventilation and the Consequences for Survival from Cardiac Arrest

Despite intensive efforts to improve cardiac arrest survival over the last 3 decades, survival rates have seen little change. The unadjusted rates of survival to hospital discharge only increased from 5.7% in 2005/2006 to 9.8% in 2012[1] This is despite an exponential growth in the treatments and devices used to aid the rescuers in their resuscitative attempts.

Chest compression rate and depth has been a key focus of the resuscitation guidelines[2] development over the past 15 years and have spawned a number of products designed to replace the rescuer in the provision of chest compressions. However, none of these has shown any advantage over manual chest compression when it comes to patient survival[3].

Ventilation on the other hand has only attracted minimal attention. This is despite the fact that the effects of "Inadvertent Hyperventilation" (IH) have been shown to be a key factor in patient survival[4].

While chest compressions are key in circulating oxygenated blood to the vital organs during CPR, any action that inhibits venous return to the heart will of course reduce the volume of blood available to be circulated. This reduction in blood flow greatly impacts the patient's chance of survival.

IH has numerous effects on circulation as well creating other issues for the patient. In the mask ventilated patient, when ventilations are delivered at too fast a rate and too high a tidal volume, gastric insufflation and aspiration of stomach contents are a major (and well documented) concern[5,6]. In the protected airway, these force are applied directly to the lungs creating high peak airway pressures and risking barotrauma.

The mechanisms by which IH affects survival are purely mechanical and simple to understand. Over-inflation of the lungs or not allowing sufficient expiratory time for full lung emptying before delivery of the next breath ("breath stacking") has the effect of reducing intra-thoracic space as well as increasing peak airway pressure and creating inadvertent PEEP. In the mask ventilated patient, the creation of gastric insufflation pushes the diaphragm up into the thoracic cavity further reducing the intra-thoracic space and limiting lung expansion.

This reduction in intra-thoracic space decreases lung compliance (making it even more difficult to ventilate) and creates compression of the great vessels, decreasing venous return to the heart. This in turn reduces cardiac preload and subsequent forward blood flow.

With less blood to move forward the volume of blood that can be oxygenated, is decreased. While reducing circulatory flow affects all vital organs, deoxygenating heart muscle in particular makes it less susceptible to the effects of defibrillation. This of course greatly affects patient survivability from a cardiac arrest.

By the action of Inadvertent Hyperventilation we create a vicious circle of over ventilation, great vessel compression, reduced cardiac pre-load, reduced circulation, reduced oxygenation, lower coronary perfusion pressures, reduced CO_2 removal and a reduction in the effectiveness of defibrillation. All of which can be eliminated by providing high quality chest compressions along with the "controlled ventilation" that is key to increased patient survival.

Where small tidal volumes are delivered at a fast rate the issue of reduced cardiac pre-load due to compression of the great vessels may not be as serious a concern. However, the washout of CO_2 that will occur does create an additional insult to the patient.

Decreased CO_2 levels in the blood will create cerebral vasoconstriction, destroying brain cells and potentially risking an increase in the level of neurological deficit in cardiac arrest survivors. This effect is of course also very serious in Traumatic Brain Injury (TBI) patients where cerebral ischaemia may well be exacerbated by CO_2 washout caused by inadvertent hyperventilation, increasing neurological damage[7].

Conclusion

When ventilating cardiac arrest patients, or indeed, when ventilating any patient, regardless of their illness or injury, care should be taken to provide controlled ventilation and to avoid inadvertent hyperventilation at all costs if patient survival is to improve.

References

1. Sasson C, et al; Predictors of survival from out-of-hospital cardiac arrest: a systematic review and meta-analysis. Circ Cardiovasc Qual Outcomes. 2010;3:63–81

2,3, 2015 American Heart Association (AHA) Guidelines update for Cardiopulmonary Resuscitation and Emergency Cardiovacular Care: Circulation, October 2015

4. Hyperventilation-Induced Hypotension During CPR Aufderheide, et al: Circulation. 2004;109:1960-1965.

5. Influence of tidal volume on the distribution of gas between the lungs and stomach in the un-intubated patient receiving positive-pressure ventilation. Wenzel et al., Crit Care Med 1998;26:364-369

6. Aspiration of stomach contents is common during cardiac arrest Felegi WB, et al; Gastric trauma and pulmonary aspiration at autopsy after CPR (Abstract). Acad Emerg Med 1996;3:441 6.

7. Guidelines for the management of Severe Brain Injury; brain Trauma Foundation 1995