Commentary

Capnography during cardiac resuscitation: a clue on mechanisms and a guide to interventions

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Abstract

Measurement of the end-tidal partial pressure of carbon dioxide ($P_{ET}CO_2$) during cardiac arrest has been shown to reflect the blood flow being generated by external means and to prognosticate outcome. In the present issue of *Critical Care*, Grmec and colleagues compared the initial and subsequent $P_{ET}CO_2$ in patients who had cardiac arrest precipitated by either asphyxia or ventricular arrhythmia. A much higher $P_{ET}CO_2$ was found immediately after intubation in instances of asphyxial arrest. Yet, after 1 min of closed-chest resuscitation, both groups had essentially the same $P_{ET}CO_2$, with higher levels in patients who eventually regained spontaneous circulation. The Grmec and colleagues' study serves to remind us that capnography can be used during cardiac resuscitation to assess the mechanism of arrest and to help optimize the forward blood flow generated by external means.

Keywords arrhythmias, asphyxia, capnography, cardiac arrest, prognosis, resuscitation

Twenty-five years ago, Professor Zden Kalenda from Utrecht University Hospital, The Netherlands proposed the use of capnography (display of the airway partial pressure of carbon dioxide waveform) as a means to assess pulmonary, and thus systemic, blood flow during cardiac resuscitation [1]. In this pioneer work, which included observations in three cardiac arrest victims, the end-tidal partial pressure of carbon dioxide ($P_{ET}CO_2$) closely mirrored the haemodynamic effects of 'cardiac massage' and served to promptly identify the return of spontaneous circulation. Subsequent work in animal models [2–4] and in human victims [5–7] of cardiac arrest corroborated these findings and defined, with greater precision, the pathophysiologic mechanisms underlying the changes in $P_{ET}CO_2$ during cardiac resuscitation and the potential clinical applicability of capnography.

In the present issue of *Critical Care*, Grmec and colleagues report changes in $P_{ET}CO_2$ in relation to the mechanisms of cardiac arrest and the efficacy of closed-chest resuscitation [8]. They specifically studied two groups of cardiac arrest victims in whom cardiac arrest was precipitated by either asphyxia ($n=44$) or ventricular dysrhythmia (ventricular fibrillation or pulseless ventricular tachycardia, $n=141$). The $P_{ET}CO_2$ measured immediately after endotracheal intubation (preceded by only two positive pressure breaths delivered using a valve-bag device) was substantially higher in instances of asphyxial arrests than in dysrhythmic arrests (66 ± 17 mmHg versus 17 ± 9 mmHg). This difference rapidly disappeared, however, and after 1 min of closed-chest resuscitation both groups had a similar $P_{ET}CO_2$ (29 ± 5 mmHg versus 24 ± 5 mmHg). As previously reported, patients who eventually regained spontaneous circulation had significantly higher $P_{ET}CO_2$ during cardiopulmonary resuscitation (36 ± 9 mmHg versus 19 ± 9 mmHg in the asphyxia group, and 30 ± 8 mmHg versus 14 ± 5 mmHg in the dysrhythmic group).

These findings are remarkably similar to those previously reported by Berg and colleagues in animal models of asphyxial and dysrhythmic cardiac arrest [4]. The studies by Grmec and colleagues thus corroborate earlier findings and

$P_{ET}CO_2$ = end-tidal partial pressure of carbon dioxide; V/Q = ventilation/perfusion ratio.

validate animal studies suggesting that the initial $P_{ET}CO_2$ may help identify the mechanism of cardiac arrest.

$P_{ET}CO_2$ during cardiac arrest and resuscitation

The $P_{ET}CO_2$ provides an estimate of the alveolar CO_2 tension and reflects the combined effects of CO_2 production, CO_2 transport (to the lungs), and CO_2 elimination modulated by the anatomical and physiological dead space. Most of the $P_{ET}CO_2$ changes herein reported can be readily explained by examining the pathophysiologic abnormalities that occur during cardiac arrest and resuscitation.

During cardiac arrest, CO_2 continues to be produced in part because of aerobic metabolism (flow generated by closed-chest resuscitation) and in part because of buffering of anaerobically produced hydrogen ions by tissue-bound bicarbonate (leading to the generation of carbonic acid and its dissociation products CO_2 and H_2O) [9]. However, CO_2 transport to the lungs is severely curtailed because conventional closed-chest resuscitation typically fails to generate more than 25% of the normal cardiac output. As a result, CO_2 accumulates in the tissues, with exceedingly high levels in metabolically active organs (i.e. ≈ 350 Torr in the fibrillating myocardium [10]), and in venous blood [11]. Diminished CO_2 transport means that less CO_2 becomes available to the alveolar space for elimination through ventilation. Thus, if ventilation is kept at normal levels, a state of an increased global ventilation/perfusion ratio (V/Q) ensues, causing the alveolar partial pressure of CO_2 (and the resulting $P_{ET}CO_2$) to decline.

In experimental models in which ventilation is kept normal throughout cardiac arrest and resuscitation [3], the $P_{ET}CO_2$ decays exponentially after cessation of blood flow and reaches zero within a few minutes, as CO_2 is washed out from the lungs. Generation of blood flow by chest compression (or other means) re-establishes CO_2 transport and the measurement of the $P_{ET}CO_2$ at levels that are proportional to the amount of flow being generated [12]. Many clinical studies have now established that the $P_{ET}CO_2$ can predict the outcome of the resuscitation effort. For example, failure of closed-chest resuscitation to increase the $P_{ET}CO_2$ above 10 mmHg has been reported to predict an extremely low likelihood of restoring spontaneous circulation [6,7]. Conversely, higher $P_{ET}CO_2$ levels are associated with increased likelihood of successful resuscitation. In one study, successfully resuscitated victims all had a $P_{ET}CO_2$ level of at least 18 mmHg before the return of spontaneous circulation [7]. This concept was well illustrated in the study by Grmec and colleagues and was shown to be independent of the mechanism of arrest [8].

Clues on the mechanism of arrest

The more novel findings of the study by Grmec and colleagues, however, relate to the effects of ventilation. Patients with asphyxial arrest had nearly double the normal $P_{ET}CO_2$ at the time of intubation. This is certainly consistent with asphyxia in which impaired gas exchange precedes cessation of cardiac activity, allowing CO_2 to travel to and accumulate in the lungs before the onset of cardiac arrest (decreased V/Q). In contrast, patients with dysrhythmic arrest had nearly one-half of the normal $P_{ET}CO_2$, suggesting that some form of ventilation developed after the onset of cardiac arrest (increased V/Q).

One possible mechanism of ventilation during cardiac arrest is agonal breathing, which has been reported to occur in approximately 40% of cardiac arrest victims [13]. An intriguing observation, however, was that patients with dysrhythmic arrests who were eventually resuscitated had a significantly higher initial $P_{ET}CO_2$ (20 ± 6 mmHg versus 8 ± 4 mmHg). If the $P_{ET}CO_2$ reflects the global V/Q, one could speculate that less agonal breathing (ventilation) occurred. However, this would not be consistent with a better outcome; agonal breathing has been shown to promote not only ventilation but also forward blood flow [14] and to increase resuscitability [13]. Thus, if agonal breathing occurred, perhaps it was more vigorous with a larger effect on flow (perfusion) yielding a lower V/Q ratio and thus a higher $P_{ET}CO_2$. This, however, remains to be proven.

Clinical relevance

Beyond the specific findings, the work of Grmec and colleagues serves to remind us of the value of capnography for guiding the resuscitation process [8]. Practical and more affordable infrared technology for CO_2 measurement is now available in the form of hand-held portable devices or is embedded in portable defibrillators. Current resuscitation approaches emphasize algorithms that lack objective and real-time measurements of efficacy.

The principles underlying capnography are scientifically robust and supported by good laboratory and clinical research. The incorporation of capnography as a routine measurement during cardiac resuscitation is long overdue. Capnography can help identify proper placement of an endotracheal tube, can discern the mechanism of cardiac arrest, and can guide the technique of closed-chest resuscitation such as to maximize the blood flow generated by external means, with the expectation that such an approach could enhance the likelihood of a successful resuscitation.

Competing interests

None declared.

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