

Non-invasive monitoring of trauma severity: Skin and End Tidal PCO₂ Responses to Hemorrhage and Resuscitation

L. Henderson, C. Buising, P. Wall.

Introduction

Assessing hemorrhage severity non-invasively is challenging. In the emergency department and peri-operative setting, associations have been shown between high skin PCO₂s and low end-tidal PCO₂s and decreased trauma survivorship.^{1,2} Both of these variables can be monitored non-invasively and might therefore be useful in assessing hemorrhage severity in the emergent setting and possibly also the response to resuscitative interventions. To explore this possibility, we investigated the responses of skin PCO₂ and end-tidal PCO₂ during pressure controlled severe hemorrhage and subsequent resuscitation.

Materials and Methods

Nine anesthetized (constant rate infusion of thiopental) dogs were mechanically ventilated to maintain their arterial PCO₂ values in the 35-40 mm Hg range. The dogs were hemorrhaged to and, by continued blood withdrawal as needed, maintained at a mean arterial pressure of 40-45 mm Hg for 50 minutes and then further hemorrhaged to a mean arterial pressure of 35-40 mm Hg until their mean arterial pressure dropped below 25 mm Hg for 1 minute (4 dogs) or 40 minutes elapsed (4 dogs). The dogs were then resuscitated with lactated Ringer's solution at 200 ml/kg/hr as needed to reach and maintain a mean arterial pressure of 75-80 mm Hg. Skin PCO₂ was monitored continuously with a 37°C, Severinghaus-type sensor and recorded every 10 minutes. End-tidal PCO₂ was monitored with a mainstream infrared sensor. Cardiac output was determined with a Swan-Ganz catheter by intermittent thermodilution technique (10 ml iced injections in triplicate) at baseline, at the end of the first hemorrhage period, at the end of the second hemorrhage period, during the start of resuscitation, 30 minutes into resuscitation, and 40 minutes into resuscitation. Arterial base deficit was measured at the start and end of hemorrhage and 30 minutes into resuscitation. Data were analyzed by one-way ANOVA with the Tukey-Kramer post hoc test and are shown ± SEM.

Results

One dog died 36 minutes into the first hemorrhage despite attempted resuscitation; the remaining dogs survived to euthanasia. In the dog that died, skin PCO₂ steadily increased during hemorrhage (57 to 104 mm Hg) while end-tidal PCO₂ steadily decreased (31 to 17 mm Hg); the dog's final base deficit was 21.7 mEq/l. During hemorrhage in the each of the remaining dogs, skin PCO₂ steadily increased (60 ± 7 to 115 ± 9 mm Hg, $p < 0.05$; Figure 1) as did base deficit (5.0 ± 0.5 to 16.4 ± 1.1 mEq/l, $p < 0.05$; Figure 1). The end-tidal PCO₂ at the end of hemorrhage in the other dogs was lower than at the start (34 ± 8 to 22 ± 3 mm Hg, $p < 0.05$; Figure 2), but the decline was only steady in one other dog. Cardiac output also decreased in response to hemorrhage (1.4 ± 0.2 to 0.6 ± 0.1 l/min, $p < 0.05$; Figure 2). Cardiac output and end-tidal PCO₂ increased rapidly along with blood pressure in response to lactated Ringer's solution administration. Both initially overshoot their baseline values. Skin PCO₂ values did not begin to decline until 10 minutes into resuscitation and, although steadily decreasing, did not reach baseline during 40 minutes of fluid administration. Base deficits also did not reach baseline. The Pearson correlation coefficient between skin PCO₂ and base deficit was 0.57 ($p < 0.0004$).

Figure 1. Skin PCO₂ and arterial base deficit (n=8).

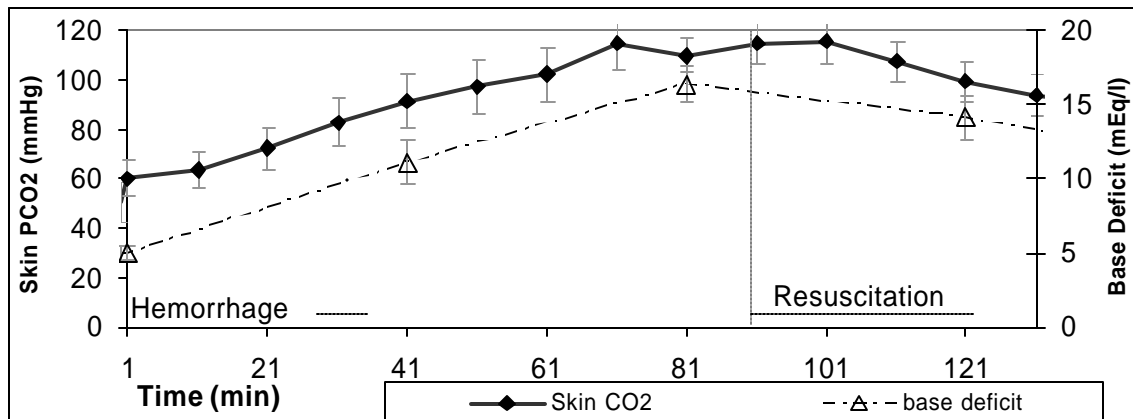
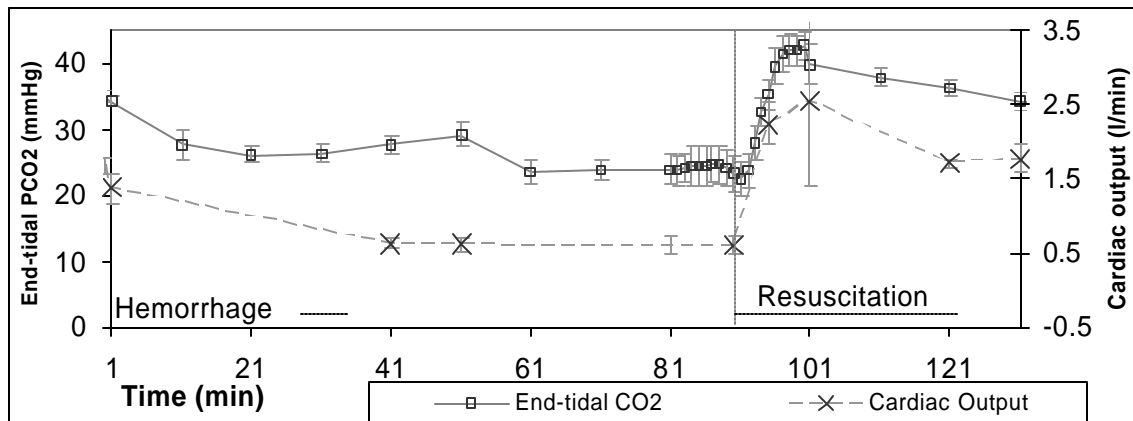


Figure 2. End-tidal PCO₂ and cardiac output (n=8).



Conclusions

This study shows that skin PCO₂ values rise progressively during hemorrhagic hypotension, and, while responsive to resuscitation, do not rapidly return to baseline. Declines in end-tidal PCO₂ during hemorrhage, on the other hand, occur but are frequently not progressive. At the start of cardiovascularly successful resuscitation following severe hemorrhagic hypotension, the response of end-tidal PCO₂ to fluid administration, like that of cardiac output, is rapid and considerable, generally resulting in end-tidal PCO₂ values transiently above baseline. Because they can both easily be monitored non-invasively and continuously and because of their differing responses to hemorrhage and resuscitation, both skin PCO₂ and end-tidal PCO₂ might prove to be useful variables to monitor in trauma patients starting in the pre-hospital setting. We would suggest skin PCO₂ may prove useful in a manner similar to base deficit – as an indicator of hemorrhage induced oxygen debt and its progressive accrual or repayment. Unlike base deficit, however, skin PCO₂ has the advantage for use in the emergent setting that it is determined non-invasively and can easily be monitored continuously. As for end-tidal PCO₂, we would suggest that while it may prove a useful adjunct when assessing patients with potentially declining or critically low cardiac output, it may prove more useful as a non-invasive indicator of the immediate cardiac output response to fluid administration.

References

1. Tatevossian RG, Wo CCJ, Velhamos GC, Demetriades D, Shoemaker WC: Transcutaneous oxygen and CO₂ as early warning of tissue hypoxia and hemodynamic shock in critically ill emergency patients. *Crit Care Med* 28:2248-2253, 2000.
2. Tyburski JG, Collinge JD, Wilson RF, Carlin AM, Albaran RG, Steffes CP: End-tidal CO₂-derived values during emergency trauma surgery correlated with outcome: a prospective study. *J Trauma* 53:738-743, 2002.