Traumatic Events:  
*Cautions about ETCO₂ Analysis in Trauma*

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No direct financial conflicts to disclose
End-tidal CO2 depends on 3 physiologic variables: ventilation, perfusion, and metabolism. As such, ETCO2 can be used to monitor these three variables. Perhaps the most important is ventilation.
ETCO2 provides a breath-by-breath confirmation of proper endotracheal tube placement (because of ventilation).
Perfusion status can be assessed with ETCO2.
Perfusion status in CPR is one example of ETCO2 usage. In the top image, falling ETCO2 shows either: fatiguing CPR provider and ineffective compressions or futile resuscitation.

The bottom image shows 3 episodes of significant rises in ETCO2 that correspond with ROSC. Bicarbonate administration can be a mimic of this.

(Images found through Google Image search—apologies for not being able to provide specific references or acknowledgements.)
ETCO2 is also affected by cellular metabolism; as such, acid-base abnormalities can be monitored with ETCO2.
Examples are DKA (high blood glucose + low ETCO2) and sepsis (infection + low ETCO2).

(Images found through Google Image search—apologies for not being able to provide specific references or acknowledgements.)
Finally, when perfusion, ventilation, and metabolism are all normal, ETCO2 reliably reflects paCO2, and can be used to guide ventilatory management in isolated traumatic brain injury.
The goal of TBI management is intracranial pressure control. Without herniation, guidelines recommend keeping ETCO2 between 35-40mmHg. For acute herniation control, the goal ETCO2 is 30-35mmHg. This 5-point delta results in a 20% reduction in cerebral blood flow. This decreases cerebral blood volume and can compensate for increased ICP; over-ventilating, or hyperventilating when herniation is not present, can cause significant harm.
Brain tissue oxygenation is very sensitive to respiratory rate—moving from 6 to 12 can have profound effects. This underscores the need to guide ventilations through an objective measure, such as ETCO2.
What must be remembered, though, is that perfusion, ventilation, and metabolism must be NORMAL; otherwise, the differences between paCO2 (in the blood) and ETCO2 become too great.

In this study of hemorrhage in rats, severe hemorrhage (decreased perfusion) causes significant discrepancies in values.
In this study of all comers (trauma, sepsis, neuro, etc.) the correlation in hypercapnea is not very good.
This paper by Copass et al shows further evidence that altered metabolism (base deficits in trauma patients are signs of acid-base imbalance due to poor perfusion) significantly affects the correlation.
A: no severe chest trauma, hypotension, or metabolic acidosis (100% concordance);
B: chest trauma, hypotension, or acidosis (60% concordance)

Finally, when chest trauma, hypotension, or altered metabolism are present, there is poor agreement. Please notice, though, that ETCO2 generally overstates paCO2.
What’s the bottom line?

• In polytrauma, high ETCO$_2$ likely represents high paCO$_2$
  – Can probably be used to titrate therapy

• Low ETCO$_2$ in polytrauma unreliable
  – May reflect hypoperfusion, not hyperventilation