Commentary

The optimal endpoint of resuscitation in trauma patients

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Abstract

Although it has never been prospectively validated, the base excess (BE) is regarded as the standard end-point of resuscitation in trauma patients. In a rat hemorrhage model, in this edition of *Critical Care*, Totapally and colleagues demonstrate that the BE is an insensitive and slowly responsive indicator of changes in intravascular volume. This contrasts with changes in the esophageal-arterial carbon dioxide gap which more closely followed changes in blood volume. Esophageal or sublingual capnometry may prove to be a useful tool for monitoring the adequacy of resuscitation in trauma victims.

Keywords base excess, carbon dioxide, esophageal capnometry, hemorrhage, resuscitation, sublingual capnometry, tissue hypoxia, trauma

The assessment of intravascular volume and the adequacy of volume resuscitation are among the most difficult clinical challenges. Systolic blood pressure, heart rate and urine output change minimally in early hemorrhagic shock. Hypotension, tachycardia, cold extremities, decreased urine output and poor capillary refill are only present in patients who have lost in excess of 30% of their blood volume (class III hemorrhage) [1]. Furthermore, both the central venous pressure and the changes in the central venous pressure in response to volume loading are poor indicators of intravascular volume and recruitable cardiac index [2]. While flow to the brain and the myocardium is preserved in patients with 'compensated shock', splanchnic and renal perfusion may be seriously compromised [3]. Splanchnic hypoperfusion leads to both functional and structural changes in the gut mucosa, with increased permeability and translocation of bacteria and bacterial products [4]. Increased mucosal permeability has been strongly associated with the development of the multiorgan dysfunction syndrome [4,5].

The expedient detection and correction of tissue hypoperfusion associated with 'compensated shock' may limit organ dysfunction, may reduce complications and may improve patient outcome. It is probable that the earlier tissue hypoperfusion is detected and corrected, the greater the likelihood that outcome will be improved [6]. Indeed, Rivers and colleagues reported a 32% relative reduction in the 28 day, all cause mortality of patients with severe sepsis who received early aggressive volume resuscitation in the emergency department [7]. Rivers *et al*. used the central venous oxygen saturation as the endpoint of resuscitation in the intervention group, while treatment in the control group was guided by standard clinical endpoints including the central venous pressure. While their study clearly demonstrates the value of early aggressive volume resuscitation, the use of central venous oxygen saturation to guide early resuscitation is not practical and has important limitations [8].

The base excess (BE) has become the standard endpoint of resuscitation in trauma patients. Remarkably, while the BE has been demonstrated to be of prognostic value, it has never been assessed prospectively in trauma patients [9–15]. The use of the BE is based on the principle that tissue hypoxia associated with poor perfusion will result in the generation of hydrogen ions and a metabolic acidosis. However, it is probable that tissue hypoperfusion may occur in the absence of a significant change in the BE. Furthermore, as significant time is required for the liver and kidney to regenerate bicarbonate [16], it can be expected that there will be a long lag phase between the correction of intravascular volume and normalization of the BE.

BE = base excess.

Both of these assumptions are elegantly demonstrated in the study by Totapally and colleagues reported in the present issue of *Critical Care* [17]. In a rat hemorrhage model these authors demonstrated that the BE responded slowly to changes in intravascular volume and that there was a significant increase in the BE only when the mean arterial blood pressure fell by greater than 50%. However, Totapally *et al*. demonstrated that changes in the esophageal carbon dioxide gap closely mirrored changes in the intravascular volume. Similar findings have been reported by other investigators. In patients with penetrating trauma, Baron and colleagues demonstrated that sublingual carbon dioxide measurements correlated well with the degree of blood loss [18]. Both Ivatury and colleagues and Kirton and coworkers have demonstrated that gastric intramucosal pH correlates well with the degree of injury and that optimizing the gastric intramucosal pH in the first 24 hours following trauma is associated with a reduction in the incidence of organ failure and death [19–21].

The study by Totapally and colleagues suggests that the BE is an insensitive indicator of the degree of the intravascular volume deficit following hemorrhage and that it responds slowly to volume resuscitation. Esophageal and sublingual capnometry, however, appear to provide near instantaneous information regarding the degree of the volume deficit and the adequacy of volume resuscitation [22–25]. This technology is simple and noninvasive, and is ideally suited for use in the emergency room and the trauma bay. The esophageal or sublingual pCO_2 gap may prove to be a useful endpoint for the resuscitation of trauma victims.

Conflict of interest

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