Etomidate and RSI: How important is post-intubation hypotension?

To the editor: In a recent issue of *CJEM*, Zed and colleagues[1] and also Sivilotti[2] outlined the controversy surrounding the use of etomidate for emergent endotracheal intubations. At the root of this matter is the relevance of post-intubation hypotension (PIH). It is thought that etomidate use results in less PIH than other sedatives used in rapid sequence intubations (RSIs), such as propofol and thiopental. Unfortunately, although there may be short-term gain by avoiding PIH with the use of etomidate, there may also be delayed effects by causing relative adrenal suppression.

I congratulate the authors, as this is likely an important issue. However, I disagree with the suggestion by Sivilotti[2] that inducing PIH may be "beneficial, signalling marginal hemodynamic reserve (cryptic shock) well before a central line is inserted...". Few would agree that inducing hypotension in critically ill patients would have any benefit; rather, it is likely the opposite.[3] Although PIH has received relatively little attention in the literature, preliminary results from Halifax indicate that it is common and may have a significant effect on patient outcomes.[4,5] When 218 consecutive emergency department (ED) intubations were reviewed, the incidence of PIH was found to be 60.9%. In addition, patients with PIH required significantly more invasive procedures and an additional 8 days in hospital (9.0 v. 17.4 d). Although these data require further examination and may only describe an epiphenomenon, they highlight the potential importance of PIH.

I would also caution against the insertion of central lines and the measurement of central venous pressure (CVP) for the diagnosis of hemodynamic reserve. Central venous access is appropriate for the infusion of vasopressor or inotropic medications, and in some instances, volume resuscitation. Unfortunately, a single measurement of CVP is unlikely to provide reliable, treatment-modifying information in an acutely ill patent. I would advise against using CVP measurement as the sole determinant in the decision for or against volume expansion, and would suggest that this decision be guided by combining other variables, such as patient presentation, comorbidities, vital signs, investigations and clinical course.

Sedative medications used in RSI should be tailored to a patient's condition and physiologic needs. Do we need etomidate? I have not been convinced, as illustrated in the fact that I have only personally used it twice in the last 4 years, which includes approximately 5–15 intubations per month between the ED and the intensive care unit. But, avoiding post-intubation hemodynamic instability makes sense on many levels, and should be a priority. Other medications are available, and their use at doses that minimize PIH should be at least considered until the relevance of etomidate-associated adrenal insufficiency is better clarified.

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References

1. Zed PJ, Mabasa VH, Slavik RS, et al. Etomidate for rapid sequence intubation in the emergency department: Is adrenal suppression a concern? [editorial]. Can J Emerg Med 2006;8(5):347-50.
2. Sivilotti MLA. You need tube, me give one amp of etomidate and SUX [editorial]. Can J Emerg Med 2006;8(5):351-3.
3. Jones AE, Yiannibas V, Johnson C, et al. Emergency department hypotension predicts sudden unexpected in-hospital mortality: a prospective cohort study. Chest 2006;130:941-6.
4. Edwards JP, Green RS. Post intubation hypotension: incidence, risk factors and outcomes [abstract]. Can J Emerg Med 2006;8(3):210.
5. Edwards JP, Green RS. Severe hypotension following endotracheal intubation [abstract]. Can J Emerg Med 2006;8(3):210-1.

[Dr. Sivilotti responds:]

I thank Dr. Green for weighing in on the etomidate commentary.[1,2] His skepticism regarding the need for etomidate and his reluctance to use this drug are eloquent testimonials. I also congratulate his group on their valuable observations on the association between PIH and in-hospital outcomes.

I do wish to clarify one apparent misunderstanding however. The quotation attributed to me has been modified slightly, and the casual reader might be misled into believing that I recommend inducing hypotension in critically ill patients. This is simply not true. Instead, I believe that a fall in blood pressure shortly after endotracheal intubation represents not only an adverse event, but also an opportunity to recognize and correct circulatory instability that might otherwise be missed. This contrarian view was intended to illustrate how brief hypotension might not be entirely bad. Indeed, the argument that, since PIH is associated with and precedes negative events, any medication that reduces its incidence must improve outcomes is a typical example of the *post hoc, ergo propter hoc* ("after this, therefore because of this") fallacy. Green concedes that PIH "may only describe an epiphenomenon." Indeed, I think that the balance of probability favours this view, namely that it is an early marker of disease severity.

The causes of PIH are multifactorial,

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and include direct drug effects, decreases in sympathetic outflow, changes in oxygen supply/demand, and a rapid reversal of net intrathoracic pressures from negative to positive. Much like a single CVP measurement, or like a single mixed venous oxygen saturation (another indication for central access), a low mean arterial pressure 3 minutes after endotracheal intubation should be seen as an input datum into the complex decision making required during resuscitative care, rather than being seen as an outcome per se. Of course, physicians should aim to reduce the likelihood and degree of PIH, but not necessarily at all costs.

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References

1. Sivilotti MLA. You need tube, me give one amp of etomidate and SUX [editorial]. Can J Emerg Med 2006;8(5):351-3
2. Zed PJ, Mabasa VH, Slavik RS, et al. Etomidate for rapid sequence intubation in the emergency department: Is adrenal suppression a concern? [editorial]. Can J Emerg Med 2006;8(5):347-50.

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