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# Under (Not enough) pressure: The use of Etomidate or Ketamine for RSI

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There you are working your glorious shift with your program director. You've already successfully resuscitated every uncomplicated back pain and 1-year-of-body-aches you have seen for the day. You've addressed every learning opportunity correctly provided to by your PD. You're essentially a god, if not THE god, of emergency medicine . . . or at least that's how you're feeling.

Next thing you know, you see an EMS stretcher drift around the corner and wheeled directly into the room in front of your



The patient doesn't look good. There was no overhead alert. There was no forewarning. All you see is a group of professional, although mildly frazzled, paramedics urgently transferring the patient from the stretcher to the bed, all the while desperately squeezing a BVM over an obtunded-appearing patient's face. You step out to face this challenge of the day equipped with all the knowledge that a few podcasts and quickdraw attitude have given you so far. You overhear the medic giving report: "He was hypoxic . . . not responding to verbal commands . . . his first blood pressure was 120/80". As your techs and nurses assist with setting the patient up on the monitor, you start your primary survey and address some reversible causes of altered mental status. Sugar, good. Naloxone, no response. Airway? Breathing? Circulation? Airway seems patent, but patient does not appear to have strong, spontaneous respirations nor is he responding the various pokes and prods, so you decide it's time to intubate.

You call for your airway equipment, RT, and pharmacist. Next thing you see is the patient's first blood pressure: 80/40, heart rate is 110. You remember a thing about shock index and, although you were never a big math person, you know that 110/80 is greater than 1. You know this person needs airway protection, so you start doing your thing as a young "resuscitator." Your nurses establish large bore IV access and you provide a small bolus of IV fluids, you ask your pharmacist for push-dose pressors, and the next blood pressure you see is 98/60 with a heart rate of 105. Not great, but you'll take it, especially since the patient isn't any more responsive.

You're about to call out for your RSI medications when you look up, you make eye contact with your PD, and they ask: "what are you going to use for induction?"

Midazolam? No, who uses midazolam? Propofol? I'd prefer not run a code after all this. So, now you're down to two options: etomidate and ketamine. You know that both of these are good in more hemodynamically tenuous patients. However, based on all your "reading", you know that etomidate does something to the kidneys. Or is it the top of the kidneys? Does it really matter? Then you think of ketamine and about your previous procedural sedations, and it seems like that would be a good option, but you're having a hard time remembering the IV vs. IM dosing.

As a small dribble of drool drips out of the side of your mouth and your eyes glaze over from the mental effort it's taking to make this decision, your PD looks at your pharmacist and says, "Can you draw up some ketamine at 1 mg/kg?"

Next things next, your patient is intubated, your analgesia and sedation are up, and the CXR confirms your tube placement. You're placing orders and your PD looks you straight in the face and asks what you've been thinking, "is there really a difference between ketamine and etomidate for induction?"

You don't know how to respond, so you decide to do some real reading after shift, so you can be better prepared to address this question and provide optimal patient care. Also, you have a blog post to write.

RSI is a common, yet high-risk, procedure performed in the emergency department, especially in patients with more tenuous hemodynamics<sup>1,2</sup>. Commonly recommended induction medications include propofol, etomidate, and ketamine, with etomidate and ketamine being preferable in patients with unfavorable hemodynamics<sup>2</sup>. However, a common concern when selecting etomidate is the risk of adrenal suppression and the subsequent negative clinical impact<sup>3,4</sup>. Although, this known risk of etomidate use has often been downplayed, and possibly underestimated.<sup>4</sup>



As such, there have been multiple investigations to interrogate the possible negative effects of etomidate when compared to the other hemodynamically neutral induction agent, ketamine. Namely, there have been two meta-analyses comparing the use of etomidate versus ketamine for RSI, both of which offer the conclusion that etomidate confers higher mortality compared to ketamine<sup>5,6</sup>. In the older study, the analysis demonstrated a statistically significant reduction in mortality with a number needed to harm (NNH) of 31<sup>6</sup>. In the more recent analysis, the mortality benefit was not statistically significant with a NNH of 50; however, it should be noted that the authors explain the lack of statistical significance may be due to the Bayesian analysis performed<sup>5</sup>.

Regardless, both articles argue for the use of alternative induction agents in place of etomidate in critically ill patients<sup>5,6</sup>.

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